EFFECTS OF WORK-RELATED POSITIVE AFFECT ON STRESS APPRAISALS AND CARDIOVASCULAR STRESS RESPONSE

by

Farah W. Elsiss

A thesis submitted in the partial fulfillment
of the requirements for the degree of
Master of Science
(Psychology)
in the University of Michigan- Dearborn
2016

Master's Thesis Committee:

Associate Professor Susana Peciña, Co-chair
Associate Professor David Chatkoff, Co-chair

Acknowledgements

This thesis was written with the assistance of my great advisor, Dr. Peciña. Her wealth of knowledge and dedication has been very beneficial in helping me succeed with my research. I would also like to thank my amazing co-advisor, Dr. Chatkoff, for his excellent contribution, ongoing dedication, and commitment to ensure that I am always on the right track. I would also like to thank my research team at the University of Michigan-Dearborn for their devotion and enthusiasm throughout the data collection process. Finally, I would like to thank my husband and family for their endless support and encouragement to stay committed in advancing my academic career.

Table of Contents

Chapter

List of Tables

Table

List of Figures

Figure

List of Appendices

Abstract

Introduction: Work-based affects have been implicated in employees' health and well-being and have been identified as predictors of occupational stress and coping mechanisms. Occupational stress has been implicated in the genesis of cardiovascular disease, the number one killer in the US and other industrialized countries. Furthermore, arousal levels within affective experiences lead to differential activation of the central nervous system. Given the lack of research on the different levels of arousal of work-related positive affect (PA) within the context of stress and health correlates, the purpose of this study was to examine the associations between work-related high-arousal and low-arousal PA and cognitive appraisals and cardiovascular reactivity to induced psychological stress. It was hypothesized that: 1) Work-related PA (high and low arousal) will differentially but negatively predict threat appraisals during stress tasks, while challenge appraisals will be differentially but positively predicted; 2) Work-related high-arousal PA would be positively correlated with BP and CRV measures at baseline, while work-related low- arousal PA would be negatively correlated; 3) Work-related high-arousal PA will positively predict cardiovascular reactivity variables and work-related low arousal PA will negatively predict cardiovascular reactivity variables.

Methods: The sample consisted of 70 (M= 19.74, SD=3.674) university undergraduate students. Baseline cardiovascular measures were collected including blood pressure and heart rate variability measures. Participants completed the Stress Appraisal Measure (SAM) during both segments of the Trier Social Stress Task (TSST). At the completion of the task, all participants completed the Job Related Affective Well Being Scale.

Results: No associations were found between work-related PA (high and low arousal) and appraisals of threat and challenge during the speech and mental arithmetic (MA) tasks. No significant correlations were found between work-related PA (high and low arousal) with any of the cardiovascular variables during the baseline period. No significant associations were present between work-related PA and BP and all cardiovascular reactivity variables during the speech task. However, during the MA task, results showed that work-related low-arousal PA was associated with a decrease in DBP and the interaction term between high and low arousal PA and DBP was significant. The results also indicated that high-arousal PA was associated with a significant decrease in low frequency, whereas low-arousal PA was associated with a significant increase in low frequency. Low-arousal PA was also associated with an increase in LF /HF ratio, whereas high arousal PA was marginally associated with a decrease in LF/HF ratio.

Discussion: In general, work-related high-arousal and low-arousal positive affect did not predict cognitive appraisals of stress. Low-arousal PA did predict decrease in DBP in response to stress. These results demonstrate that low-arousal work-related PA is important to investigate in relation to occupational stress and cardiovascular health. Unexpectedly, high-arousal work related PA negatively predicted LF (ms^2), a measure often used as an indicator of sympathetic nervous system domination. [Keywords: Occupational stress, work-related positive affect, cardiovascular reactivity, heart rate variability reactivity, cognitive stress appraisals]

Chapter 1: Introduction

Many factors in the workplace have been found to impact health and job performance. For example, job burnout and job stressors have been implicated in employees' physical and psychological health (Auerbach & Gramling, 1998). Stressful working environments and consequent health conditions have been found to negatively affect employees' productivity and performance through job inefficiency and lost workdays. The emergence of work-stress related maladies have also led to major increases in healthcare expenditures for large US companies (Bloom et al., 2011). Given the magnitude of the problem, it is not surprising that occupational stress has received a significant amount of attention. In particular, predictors of stress, including negative affective states have been extensively studied, showing that work-related negative affect is associated with many negative health consequences including cardiovascular disease, the number one killer in the United States (CDC, 2015). Despite the significant research on stress and negative affective states, very little research has been done on the role of work-related positive affective states and potential health implications.

When considering occupational stress and job environments, the work-induced affective states and the relation of these states to physical health and functioning have been extensively studied. Work-related affective states have been conceptualized along two primary dimensions, arousal (high and low) and valence (pleasure and displeasure)

(Posner, Russell, & Peterson, 2005). The activation aspect used in models of affect is described as arousal, and the arousal is further associated with physiological activation that can have a negative impact on health (Ahn & Shi, 2015). The vast majority of studies on work-related affect and health have examined the consequences of negative affect (displeasure valence), both in association with high and low arousal, on a variety of health conditions including cardiovascular functioning (Fredrickson, 2001). These studies generally support the existing literature on the adverse impact of general negative affect on health. Despite these findings, very few studies have examined work-related positive affect (pleasure valence) on health functioning. Furthermore, researchers argue that although affects that fall under positive affect share the same qualities on valence, they argue that specific positive affects that vary on the arousal dimension (high arousal versus low arousal) differ on multiple motivational and behavioral functions (Ahn &Shi, 2015). It is therefore noteworthy to explore if work-related positive affect poses a protective factor on health as well as possible differences across the different levels of arousal.

While there are many mechanisms by which work-related affect could impact health, in particular cardiovascular health (e.g. altered cardiovascular activity, increased smoking) (Countryman, Saab, Schneiderman, McCalla, & Llabre, 2014). Of particular relevance to this study are the potential associations of work- related affect, stress related cognitions, and cardiovascular functioning. Significant literature points to the association between stress and cardiovascular health (Redmond et al, 2013). While no single definition of stress has been agreed upon, many of the stress-cardiovascular studies have examined the role of cognitive stress appraisals (e.g. threat, challenge) as described by the Transactional Model of Stress. Indeed, the association of threat appraisals to cardiovascular health includes risks of hypertension and heart attacks (Wright & Kirby, 2003). Negative affect has been extensively studied within the context of the workplace and health, and is associated with modified cardiovascular reactivity and negative appraisals of stress (Watson & Pennebaker, 1989), however there is lack of research on work-related positive affect (either in terms of high or low arousal) and both cognitive stress appraisal and concomitant cardiovascular functioning and health.

Research on the effects of positive affect on physiological responses has not received the wide scope that research on negative affect has reached. Even though a substantial amount of research has pointed to a protective characteristic of positive affect, the literature has reported inconsistent results; some research points to a significant association, while others found no associations between positive affect and negative physiological variables. However, the majority of the findings suggest an inverse association between adverse psychophysiological markers and positive affect (Fredrickson, 2001). Of those few articles on positive affect and physiological markers, fewer tap into the different dimensions of work-related positive affect; high-arousal versus low-arousal, for the examination of possible differential effects on employees' health. Thus, the purpose of this study is to explore whether work-related positive affect, like general positive affect, buffers against both negative cognitive stress appraisals and adverse cardiovascular functioning, as well as to examine if the different activation levels within positive affect are associated with differential effects on cardiovascular reactivity and cognitive appraisals to psychological stress.

The sections that follow provide further exploration of the various variables involved in the current study. An overview of the different forms of occupational stress and their impact on health is presented along with an examination of general and work-related affective experiences and their associations with stress. The introduction also provides an outline of the general stress models within psychosomatic medicine and specifies pathways by which stress can lead to health consequences, specifically cardiovascular disease. A review on cardiovascular health, including an examination of cardiovascular reactivity and related variables and their role in cardiovascular disease is also examined. The introduction concludes with an overview of the current study.

Chapter 2:The Work Environment and Occupational Stress

For most adults in the United States and other industrialized countries, being employed goes beyond just earning an income; it is considered a source of self-esteem, self-worth, and social support (Auerbach &Gramling, 1998; Quick et al., 2013). However, when employees' capabilities are overtaxed and work conditions become intolerable, work can turn into a source of dissatisfaction, stress, and alienation for the individual (Peter & Siegrist, 1999; Levi, 1990). As a result, employees' well-being is jeopardized and job performance suffers. Auerbach and Gramling (1998) disclose that a national survey of American workers reported that 72% of the employees suffer from chronic stress-related health conditions, physical and psychological, that consequently increase spikes in healthcare costs. Furthermore, according to the National Institute for Occupational Safety and Health (NIOSH), some of the leading occupation-related diseases include lung diseases, musculoskeletal injuries, cardiovascular diseases, and psychological disorders (Levi, 1990). Levi (1990) further indicates that, all of these stress-related diseases can be preventable since the etiology, prevention, and other aspects are dependent on individuals' reactions to the inflicted stressors.

Occupational stress is defined as a reaction to prolonged stress associated with the workplace and is usually portrayed by physiological and emotional fatigue, as well as subsequent stress-related disorders (Orly, Rivka, Rivka, & Dorit, 2012). Numerous empirical studies have been conducted to pinpoint the sources of occupational stress (job strain). Routine, low control, long hours, insufficient rewards and insecurity at work have been linked to adverse health outcomes (Cheng, Park, Kim, & Kawakami, 2012). Furthermore, work overload is characterized by over commitment and working for long hours on multiple tasks (Straub, 2012), and is considered a risk factor for adverse health conditions. Role ambiguity has also been linked to adverse health outcomes. Positions in which requirements and expectations are not clarified lead to frustration and subsequent stress and decrease in work performance (Quick et al., 2013).

Role overload (a condition of endorsing multiple role at the same time) and role conflict (a condition of competing demands between personal roles and job roles) have also been associated with negative impact on health. Gender differences have been apparent in the studies of role overload and conflict. Role overload and conflict, especially among mothers (role of caregiver is mainly contributed to women), have drawn substantial amount of attention in the past few years. More women today hold jobs and are committed to longer hours and workdays typical of the labor system; more households feature both parents working or are headed by a single mother (Coverman, 1989). Therefore, mothers are forced to balance between two extremely demanding roles, children caregiver and an employee, and in turn overtax their coping resources.

According to Marshall and Barnet (1993), the “scarcity hypothesis” postulates that role overload emanates from lack of time and energy to appropriately manage the roles. A study by Lundberg, Mardberg, & Frankenhaeuser (1994) used a “total workload scale” to measure the amount of conflicting demands in full-time employed women and men. They looked at stress related to autonomy at work, paid work, unpaid work and caregiving, and found that employed women face more role conflicts than men and that this disparity increases as the number of children in the house increases. They found that in houses with three or more children, women, on average, work twenty hours more than men per week. That also constitutes them doing most of the unpaid work of the household and child caregiving. Therefore, women have to put in a much larger effort than men, face chronic stress, and role overload (Lundberg et al., 1994).

Another main source of occupational chronic stress is job burnout. Job burnout is considered a response to chronic stressors at work and is characterized by fatigue, exhaustion, withdrawal, and loss of interest in one’s job and can be assessed using the Maslach Burnout Inventory-MBI (Maslach, Schaufelli, & Leiter, 2001). The inventory taps onto three broad burnout dimensions of emotional exhaustion, depersonalization, and decreased personal accomplishments (Auerbach & Gramling, 1998). In the United States, incidence rates for burnout range from 15 to 30% and costs about 150 to 200 billion dollars each year (Browning et al., 2005). Burnout is common in occupations in which highly driven individuals do not meet the high expectations of their work position. High burnout levels are extremely common in human services occupations in which the individual is responsible for taking care of other individuals, as well as in jobs that entail high personal commitment and involvement (Auerbach & Gramling, 1998). Such occupations include medical doctors, nurses, paramedics, and firefighters. A lot of studies have pinpointed to unrealistic high job expectations as the cause, but individual characteristics have been deemed to play a much bigger role in developing job burnout. It is not necessarily the job itself that causes burnout, but rather the combination of occupational-induced stress and personal characteristics that does so. For example, in a study by Browning, Ryan, Greenberg, and Rolniak (2005), individuals with strong cognitive adaptation disposition have a bias for information that maintains their sense of control, optimism, and self-esteem, and therefore serves as a protective factor or a buffer against complications from job-induced stress. On the other hand, lower sense of control, optimism, and self-esteem was positively correlated with emotional exhaustion, depersonalization, and low accomplishments (Browning et al., 2005).

Few models have been introduced to elucidate the role of job stress in the workplace. One such model is the job strain model (also called demand-control model), which postulates that when individuals have little control or power over aspects of their jobs along with “high psychosocial demands”, they exhibit heightened levels of stress (job strain) and increases in risks of health complications (Lee et al., 2002). In support of the model, depression has been associated with high demand work conditions and low control (Marshall, Barnett, & Sayer, 1997).

Furthermore, a cross-sectional study of young black and white workers found that low levels of control and social support predicted more frequent and intense bouts of anger towards co-workers, with those with the least levels of control and social support exhibiting the most anger in the workplace. Anger is also considered one of the main factors behind violence in the workplace (Auerbach & Gramling, 1998). Alternatively, high social support acted as a buffer against the adverse effects of low control in workers (Fitzgerald et al., 2003). Therefore, low occupational control is considered an “anger-causing stressor” (Ferroli, 1996). Anger, which is a negative emotion of hostility, has been linked to strong indicators of coronary heart disease such as heightened heart rate, blood pressure, high levels of circulating catecholamines, platelet activation (inflammation), and dysregulations of the autonomic system. Furthermore, low social support was linked to a 2-3-fold increase in cardiovascular disease, with hypercortisolism, high resting heart rate, and high arterial blood pressure (pressure of blood can damage blood vessels and cause atherosclerosis) being the mediators (Rozanski et al. 1999). According to Ferroli (1996), long-term anger predicted “seven-times-higher rates” of death due to cardiovascular disease in lawyers who exhibit anger when compared to lawyers who do not.

Another model that has been implicated in the association between chronic work stress and health is the effort-reward imbalance model. This model aims at pinpointing specific work-induced stressors that cause job strain and, in turn, mediate those factors’ effects on health. This model associates chronic occupational stress with the imbalance between high efforts of the individual such as over commitment, demands, and high standards with low rewards, such as money and social status in return (Peter & Siegrist 1999). This lack of “reciprocity” between efforts and gains constitutes a chronic occupational stressor that is exhaustive and detrimental to the inflicted individual’s health. A review on chronic stress literature found that individuals who are exposed to effort-reward imbalance in their workplaces experience 2.7 to 6.1 fold increase in health complications, such as cardiovascular disease after controlling for behavioral and pre-existing confounding factors (Peter & Siegrist, 1999). Risk factors such as high levels of hypertension, blood lipids (fatty acids and cholesterol- heightened risk for hyperlipidemia), fibrinogen (blood clotting protein) have been detected in individuals with heightened occupational effort-reward imbalance and are implicated in the relation between chronic occupational stress and cardiovascular health. Furthermore, one study found that high demand, high effort, and low coping resources predicted a significant increase of cardiovascular mortality (Peter & Siegrist, 1999).

The effort- reward imbalance model also points to more traditional work hazards. “Shift work” has been implicated as a chronic occupational stressor that is a risk factor for health complications. It is characterized by unstable or changing work shifts or shifts that are during unusual hours of the day (for example, midnight shifts) (Puttonen et al., 2010). According to Puttonen et al. (2010) individuals working night or unstable shifts exhibited less control and more conflicts when compared with individuals who worked day shifts (job strain model). Furthermore, shift employees are more likely than day workers to be insufficiently rewarded for their efforts (effort-reward imbalance model). Circadian stress, which refers to the unstable or irregular sleep-wakefulness cycle, manifests itself in shift work and poses serious adverse psychological, behavioral and physiological implications. Shifts that are inflexible and to which an individual has no control (job strain model) or power over, can induce significant occupational stress. Furthermore, unusual working hours that limit an individual’s relaxation or social time can induce work-life balance dysfunction (Puttonen et al., 2010). Sleep deprivation, which is induced by this dysfunction, can result in sympathetic nervous system activation and increases in blood pressure and heart rate.

A study by Meier- Ewert et al. (2004) found that complete and incomplete sleep deprivation in healthy participants (no infections) resulted in significant increase in basal C-reactive proteins (mainly produced by Interleukin 6, a pro inflammatory cytokine) which are responsible for producing plasma proteins; first responders to inflammation and are markers for cardiovascular risk, specifically for strokes and myocardial infarction. More indirectly, accumulating occupational stress can lead an individual to engage in risky behaviors such as smoking, alcohol and drug consumption, low physical activity, and unhealthy eating habits. All of these physiological, behavioral, and psychological factors interplay to increase the risk of adverse health outcomes in the context of occupational stress (Puttonen et al. 2010).

Over the past few years there has been a rapid increase in problems associated with occupational stress. This trend has been closely tied to the shift in the US economy to include more service-based occupations (Marshall et al., 1997). Such occupations are characterized by high use of interpersonal skills and patient contact; skills that are associated with high burnout rates due to insufficient training (Auerbach & Gramling, 1998). Furthermore, in the business domain, the ever-changing structure of corporate companies elicits a sense of uncertainty and loss of control among low and mid-level employees.

Across different occupations there are general underlying set of factors that contribute to occupational stress. Unpleasant physical surroundings, including loud noises, social isolation, and inadequate lighting, lead to increase in stress levels and dissatisfaction in the workplace. Shift work, work overload, role ambiguity, and perception of physical danger (e.g. while working with HIV/AIDS patients) are all common factors underlying occupational stress across wide variety of positions (Auerbach & Gramling, 1998). Individual differences also play a key role in occupational stress. Gender, ethnicity, age, and personality traits have been associated with stress in the workplace. For instance, the relationship between age and occupational well-being tends to form a U-shape, in which middle-aged workers report lower well-being than their younger and older counterparts (Warr, 1992). Furthermore, high trait anxiety, emotional states, external locus of control, and type A personality traits have been identified as predictors of occupational stress and coping mechanisms (Auerbach & Gramling, 1998). In particular, affective experiences towards work or work-based affects such as job satisfaction have been implicated in employee's health and well-being. Work-based affects have also been identified as predicators of occupational stress as well as everyday stressors, posing significant impact on perception, cognition, and responses.

Chapter 3: Affect: Valence and Arousal

Affect is a psychological state that describes the relationship between an individual and the present environment or event (Duncan &Barrett, 2007). It is a subjective experience that is fundamental to moods and emotions and plays a major role in individuals' perceptions of the world around them with its influence extending beyond mood to impact cognitive factors, social behaviors, and overall quality of life (Kuppens, Russell, Tuerlinckx, & Barrett, 2013; Kuperman, Brysbaert, Estes, & Warriner, 2014). Affective experiences are portrayed along two primary dimensions, arousal and valence (Russell, 2003). Theorists and researchers converge on the importance of the two properties as central to understanding the affective experience. Arousal refers to the range at which an event elicits excitement or relaxation, while valence refers to the range at which an event is considered pleasant or unpleasant (Kuperman et al., 2014). The relation between valence and arousal is considered a prerequisite to understanding the implications and role of affect. It helps delineate the role affect plays in cognitive functioning, executive functioning, psychopathology, and health (Kuppens et al., 2013). Therefore, individual variations in affect structure are often addressed when examining the effects of affect on other domains, specifically on health factors. Evidence linking individual variations in the affective experience to different personality traits (e.g. extraversion) and cultural differences has also been found (Kuppens et al., 2013).

Furthermore, a number of models have been addressed in the literature to describe the relation between valence and arousal. One of the most widely used and theoretically supported models, the circumplex model, presents valence and arousal as orthogonal dimensions (Kuppens et al., 2013; Posner et al., 2005; Russell, 1980). According to this model, all affective experiences are produced from two central neurophysiological systems, one is associated with valence and the other is associated with arousal (Posner et al., 2005). Emotions are derived from varying degrees of these two dimensions, and therefore individuals can experience emotions characterized by pleasant activation, pleasant deactivation, unpleasant activation, or unpleasant deactivation (Russell, 1980). Furthermore, the different patterns of activation of these two neurophysiological systems produce distinctive emotions, cognitive explanations, and labels to describe the related physiological experiences (Posner et al. 2005). Therefore, emotions that endorse similar linear combinations of the two dimensions (e.g. unpleasant activation) are characterized by distinct underlying physiological, behavioral, and cognitive indications.

Differential physiological responses have also been determined for emotions within the valence and arousal activation dimensions. Numerous studies have demonstrated differential activation patterns of the mesolimbic system and prefrontal cortex associated with emotions of different valences, positive and negative (Posner et al., 2005). Moreover, different arousal levels have been shown to lead to differential activation of the reticular formation, the main regulator of arousal in the central nervous system (Posner et al., 2005). According to the circumplex model, these distinct neurophysiological changes are represented through different cognitive representations. The prefrontal cortex receives the signals associated with specific valences and arousal levels and integrates that information with context, memories, and expectations to form a full affective experience that is characterized by distinct cognitive interpretations (Posner et al., 2005).

Emotional States

In the literature, positive affect and negative affect are terms used to indicate the different emotional states (Watson & Clark, 1984). Extensive literature has linked emotional states to physiological and psychological health (Hu & Gruber, 2008). Negative affect is associated with unfavorable health outcomes (e.g. depression) and poor quality of life, whereas positive affect is associated with protective health factors such as lower systolic blood pressure and reduced negative physiological and psychological impact of stress (Hu & Gruber, 2008). General emotional traits as well as dispositional states of both negative and positive affect have been examined in relation to health outcomes. General emotional traits have been determined as better predictors of individuals' functional status. While not being directly examined in this study, the majority of research in the literature has explored the aversive effects of negative affective traits and states.

Negative Affect and Arousal

Negative affect is defined as a universal dimension for unpleasurable experiences and engagement, as well as subjective distress (Hu & Gruber, 2008). Negative emotions classified as high-arousal include frustration, anger, and tension, whereas negative emotions classified as low-arousal include boredom, sadness, and gloom. Studies have shown that individuals with higher levels of negative affect endorse greater negative perceptions towards their health and lower health–related quality of life (Hu & Gruber, 2008). Furthermore, Watson and Pennebaker (1989) found a positive association between state and trait negative affect and somatic complaints across multiple studies. Negative affect is also associated with illness symptoms of chronic diseases such as diabetes, arthritis, and cardiovascular disease, as well as psychological disorders including anxiety and depression. It is also classified as a likely risk factor for a number of precursors of cardiovascular disease such as hypertension.

A number of health models and theories postulate that negative affect constitutes physiological arousal through increases in blood pressure, heart rate, and other indicators of increased cardiovascular activity, which in the long term can lead to the development of cardiovascular disease (Hilmert, Teoh, & Roy, 2013). Furthermore, psychological states that are characterized by high negative affect, such as stress, anxiety, and anger, have also been implicated in cardiovascular disease. In a study by Hilmert, Teoh, & Roy (2013), researchers found that highly activated, negatively charged affect in response to laboratory-induced stress task was positively associated with rates of cardiovascular disease, whereas low-arousal negative affect was unrelated to CVD. Moreover, individuals who experienced strong high-arousal negative emotions during the stress tasks also demonstrated larger increases in blood pressure and heart rate. The study also concluded that when activation in negative affect is high, it moderates the effects of the emotion on cardiovascular functioning, explaining a high percentage of variance in systolic blood pressure, diastolic blood pressure, and heart rate reactivity (20.70%, 16%, and 8.47%, respectively) (Hilmert, Teoh, & Roy, 2013).

Positive Affect

Positive affect refers to a range of pleasurable feelings that individuals experience. Joy, excitement, enthusiasm, and contentment are different forms of positive affect. According to the literature, positive affect is classified into two distinct forms: short-term positive feelings, also labeled as state positive affect (PA), and more stable, trait-like positive feelings, or trait positive affect (PA) (Pressman & Cohen, 2005). Extensive research points to the importance of sustained PA on health with direct benefits including lower stroke incidents, improved pregnancy outcomes, reduced pain perception, advanced skin barrier recovery, and overall lower morbidity (Robles, Brooks, & Pressman, 2009). The findings remained to be true even after controlling for effects of negative affect.

Two general frameworks have been established to explain how trait PA leads to positive health consequences. The direct effects model stipulates that PA affects overall health regardless of its impact on stress responses; alternatively, the stress-buffering model focuses the light on stressors. In the latter model, it is proposed that PA impacts health states through its protective effects against the negative outcomes of prolonged stressors. In a study by Bostock, Hamer, Wawrzyniak, Mitchell, & Steptoe (2011), higher positive affect or positive emotional style (PES) corresponded with complete diastolic blood pressure recovery as well as lower cortisol response to stress. Furthermore, participants in the study reported lower perceived tension and higher locus of control during the stress tasks. It has also been proposed that positive affect has a profound effect on cognitive appraisals of stressful events, specifically inducing lower threat perceptions and reduced physiological activation of the autonomic nervous system and hypothalamic-pituitary axis (HPA) (Bostock et al., 2011). According to Papousek and colleagues (2010) state positive affect or short-term PA is not associated with cardiovascular recovery or subjective recovery following a stressor, and therefore, is not often studied within the context of CVD.

Positive Affect and Arousal

Valence-based approach to evaluating affect has been the predominant form of examining methods by which affective experiences, positive and negative (pleasure and displeasure), can influence various domains including decision-making, judgment, and health (Hu & Gruber, 2008; Ahn &Shin, 2015). However, this approach has put constraints on the differential effects of specific emotions, especially those within PA. Alternatively, by utilizing the circumplex model of emotion, effects of PA can be related to the corresponding combinations of valence and activation-arousal. Excitement is considered a high positive affect and is characterized by positive valence and high level of activation, whereas contentment is a low positive affect with low levels of activation. This model allows us to differentiate between high-arousal and low-arousal positive emotions, as well as their respective differentiating effects on health outcomes, particularly within cardiovascular health.

Furthermore, Ahn and Shin (2015) argue that although affects that fall under positive affect share the same qualities on valence, they argue that different arousal levels can to a variety of functioning, including cognitive and motivational functions. For instance, differential effects within positive affects have been demonstrated in the adoption of new technology, with those exhibiting contentment and relaxation more likely to not adopt new technology when compared to those high in interest and excitement (Ahn &Shin, 2015). Excitement has also been shown to intensify certain behaviors such as increasing the level of subsequent aggression, as well as increasing the level engagement in pro-social actions (Ahn &Shin, 2015). Furthermore, studies have shown that the positive affects of pride and contentment are distinct from one another and pose different effects on behaviors and cognitions (Ahn & Shin, 2015). Other studies also presented distinct functions for different positive emotions in regards to processing of persuasive messages (Griskevicius, Shiota, & Neufeld, 2010). Understanding the level of activation or arousal within different positive affects is essential to gain a full assessment of their different impacts on health.

Affective activation is often described as physiological arousal in the literature, the biological way through which affective experiences can impact health outcomes (Pressman & Cohen, 2005). General positive affect has been extensively linked to favorable health markers including decreased cardiovascular risks and decreased mortality (Armon, Melamed, Berliner, & Shapira, 2014). However, recent studies have shown that affective activation can modulate those effects, especially with respect to cardiovascular activity (Armon et al., 2014). Several studies have found increases in cardiovascular activity, including heart rate and blood pressure, in individuals with high-arousal positive affect. Alternatively, moderately high-arousal positive affects have been consistently correlated with lower cardiovascular reactivity to stress tasks (Armon et al., 2014).

Chapter 4: Stress

While stress has not yet been defined in the paper, it is a complex construct that was examined in the current study. Stress is a normal process of our everyday lives and prevails across cultures and societies. There is no single consensus on the definition of psychological stress. However, within psychosomatic medicine, three main models have been introduced to elucidate the role of stress on health. One of the models is Walter Cannon's acute stress response model. Walter Cannon mainly focused on the immediate bodily responses to acute events or stress. He introduced the term "fight- or-flight" to describe the sympathetic nervous system activation in response to perceived threat to the body's "homeostasis" or balance. Short-term fight-or-flight response is essential for survival as it relocates biological resources, including endocrine and cardiovascular, to initiate a reaction or retraction from the threat. Cannon also coined the term "homeostasis" to describe the body's maintenance of stability; any threats to this stability initiate the physiological stress response (Vaessen, Hernaus, Germeys, & Amerlosvoort, 2015).

Another model that conceptualizes the role of stress in health comes from Hans Selye. Endocrinologist Hans Selye was one of the first to explore the physiological impact of long-term, or chronic, stress on health (Cannon, 1994). According to his fundamental stress theory, stressful events are implicated in the genesis of disorders (Matthieu & Ivanoff, 2006). Selye further identified stress as a "nonspecific" bodily response to outside influences, and presented the notion of a “stressor” defined as “an agent which elicits stress” (Filaretova, 2012, p.195). A stressor can be a real or an imagined object, situation, or stimulus that initiates a stress response in individuals (Matthieu & Ivanoff, 2006). Furthermore, Selye coined the general adaptation syndrome; a three-stage adaptation process that individuals engage in when encountered with a major event or a stressor (Thoits, 2010). Each stage is characterized by distinct physiological reactions; with the alarm stage instating a fight-or-flight response, the adaptation stage initiating resistance to the stressor, and finally, an exhaustion stage, characterized by the depletion of bodily resistance. The latter stage is highly associated with consequent adverse health outcomes (Thoits, 2010).

According to Everly and Lating (2002), stressors are classified into two types, psychosocial and biogenic. Biogenic stressors, including caffeine and extreme temperatures, can elicit a physiological stress response without the need for a cognitive appraisal (Everly & Lating, 2002). Psychosocial stressors, however, involve a reaction based on how an individual perceives the stimuli. Richard Lazarus and Susan Folkman were among the first to identify that psychosocial stressors involve a cognitive interpretation continuum that ranges from harmless to highly harmful, and consequently, this interpretation influences an individual’s reactions (Everly & Lating, 2002). Furthermore, according to Matthieu and Ivanoff, the perception of a situation as psychologically stressful is a key prerequisite to defining an event as a psychosocial stressor requiring a stress response (Matthieu & Ivanoff, 2006).

Lazarus and Folkman classify mental stress as a “transaction” between the individual and the environment (Matthieu & Ivanoff, 2006). This transaction involves a cognitively based interaction between the person and events to which subjective thoughts, appraisals, and perceptions determine subsequent physiological and cognitive stress responses. This type of "transaction" has been described as the cognitive primacy perspective. Theorists and researchers widely use this term to describe the process by which subjective interpretations precedes and formulates the stress response (Matthieu & Ivanoff, 2006). One of the main concepts within the transactional framework is the appraisal theory (Lazarus and Folkman, 1984).

Lazarus' cognitive appraisal theory defines the process by which differential cognitive evaluations and appraisals of events elicit different stress responses. According to this model, the objective nature of the event is not the forefront determinant of the subsequent responses, but rather it is the appraisal process. Lazarus and Folkman (1984) differentiated between the two aspects of the appraisal process, primary and secondary appraisals. Primary appraisals constitute an evaluation of the stressor as it relates to the individual's well being; when the attention of an individual is focused on the extent or potential of an event to elicit harm (Matthieu & Ivanoff, 2006). Appraisals vary across different individuals and events can be interpreted as positive (benign), stressful, or irrelevant. Positive appraisals constitute projections of positive results and the absence of any harmful or negative consequences to the well-being of the individual. However, stressful appraisals constitute projections of negative and harmful impact on the well-being of the individual. Irrelevant appraisals constitute a lack of interest in the event and the subsequent results (Matthieu & Ivanoff, 2006).

Secondary appraisals constitute an evaluation of one's own coping resources to undertake a harmful stressor. The evaluation of available coping skills is always done within the context of the primary appraisal and both can occur simultaneously (Matthieu & Ivanoff, 2006). Furthermore, three perceptions resulting from secondary appraisals have been identified: Challenge, harm (loss), or threat (Lewis, 2001). Challenge signals a risk of possible negative consequences but is usually characterized by positive events that are accompanied with mastery. It also involves personal growth resulting from the application of coping skills. Harm or loss is a belief that relates to past physical or emotional loss, and threat is a belief that relates to an anticipation of future physical or emotional harm or loss (Matthieu & Ivanoff, 2006). Both, primary and secondary, appraisals are strictly cognitive in nature and are often precedents to the formulation of an event's significance and subsequent action.

Following the cognitive appraisal, an individual decides on and engages in the appropriate coping mechanism to decrease the discrepancy present between the situation's demands and available resources. According to Quick, Wright, Adkins, Nelson, and Quick "coping is a cognitive and behavioral process of mastering, tolerating, or reducing internal and external demands" (2013, p.314). Coping mechanisms can be classified into two broad categories; problem focused coping and emotion-focused coping (Lazarus & Folkman, 1984). Problem focused coping involves active involvement to change or avoid the stressful situation, whereas, emotion focused coping involves reframing ones' own emotions and cognitions to better deal with the threatening situation. Denial and wishful thinking are common practices among those who engage in emotion-focused coping. It is essential to consider all aspects of the stress experience within the transactional framework; stress appraisal, coping mechanisms, and stress response, in order to understand the impact of stress on well-being and health.

More recently, new models have been introduced to elucidate the process of stress. The concept of allostatic load has received increased attention by a number of researchers in the field (Ganzel, Morris, & Wethington, 2010). The theory of allostasis, a replacement of the theory of homeostasis, is identified as a working model that classifies the emotional regions of the brain as the main moderators for constant overall physiological adjustments in response to ongoing outside challenges (Ganzel et al., 2010). The model stresses the importance of examining variations of stress responses as well as the associations between history, the current stressor, context, the brain, and physiological responses to assess consequent health outcomes (Ganzel et al., 2010). It identifies ongoing changes in stress responses across a person's lifetime as a means to establish "stability through change" (Ganzel et al., 2010).

Acute and Chronic Stress/Stressors

Non-threatening, short-term stress (acute stress) can sometimes be considered an adaptive survival mechanism; however, when stress is prolonged, intensified, chronic, or recurrent, it can lead to major dysregulations of the stress response system and lead to a predisposition to a variety of psychopathology (Vaessan et al., 2015). Moreover, compared with acute stressors, long-term, persistent chronic stressors are more difficult to study due to ethical issues and their ongoing nature. Slow but adverse effects of chronic stressors are, therefore, not always self-reported by patients. According to Baum (1990), chronic stressors' duration can vary across different studies, ranging from one week to several years, adding to the problem of operationalizing chronic stressors. Chronic stressors are defined as situations or events that pose threats or challenges for an extended period of time and require an emotional response (Baum, 1990). Both acute and chronic stressors are associated with adverse medical outcomes such as diabetes, cardiovascular disease, and autoimmune disease. Chronic stressors, however, are associated with worse prognosis and treatment outcomes.

Stress and Cardiovascular Disease

Chronic stressors, including occupational stress, increase risks of adverse health events through multiple “universal” mechanisms. According to the American Heart Association (AHA), chronic exposure to psychological stress/ stressors can lead to pathophysiology of cardiovascular disease by activating bodily responses involving the hypothalamic-pituitary-adrenal axis (HPA), sympathetic-parasympathetic systems, and inflammatory processes (Redmond et al, 2013). When exposed to stress, the endocrine system communicates with the body through the activation of the HPA axis in which adrenal medulla (ordered by the sympathetic nervous system- hypothalamus and pituitary gland) secretes epinephrine and norepinephrine. Those hormones stimulate the body’s fight-or-flight reaction characterized by increases in heart rate, blood pressure, and cortisol secretion (stress hormones). Elevated levels of catecholamines and cortisol (hypercortisolism) have been implicated in individuals facing chronic occupational stressors (Orth-Gomer et al, 2000; Rozanski et al., 1999), signaling that prolonged sympathetic nervous system activation and a fight-or-flight process are present.

This dysfunction in autonomic nervous system can also lead to decreases in heart rate variability, which is a major contributor to cardiovascular mortality. Furthermore, individuals who suffer from chronic job stress and exhibit low control over job-related decisions have “blunted baroreflex sensitivity” or the inability to readjust blood pressure after its increase (Dimsdale, 2008). Therefore, the exposure to chronic stress due to work hinders the body's ability to achieve homeostasis, or baseline levels, since production of cortisol is not being ceased and the body's fight or flight reaction is not shutting down. Moreover, individuals endorsing perceptions of greater work overload due to role conflict (e.g. women as caretaker and employee) have higher levels of norepinephrine levels even after leaving work (Straub, 2012). This indicates autonomic nervous system dysregulations in which high activity of sympathetic nervous system is maintained and is overpowering the parasympathetic nervous system.

Indirectly, through psychosocial mechanism, chronic stressors increase the chance for an individual to engage in maladaptive behaviors such as smoking, alcohol and drug consumption, poor sleep hygiene (complete or partial sleep deprivation), low physical activity, and lack of good nutrition. According to the American Heart Association (2014), obesity functions as a risk factor for diabetes mellitus and serious cardiovascular diseases such as strokes and coronary heart disease. Furthermore, sleep deprivation has been associated with increased blood pressure and higher levels of leucocytes and Interleukin-6 (pro-inflammatory cytokine) circulation indicating the presence of inflammation, which, if prolonged, can lead to cardiovascular disease (Meier-Ewert et al., 2004). Excessive levels of C-reactive protein, an inflammatory marker for cardiovascular disease, were found in individuals with partial and complete sleep deprivation (Meier-Ewert, et al., 2004). C-reactive proteins have also been implicated in the production of blood-clotting agents (restrict blood flow) and aid in the development of atherosclerosis (Mussleman et al., 1998). Furthermore, stress has shown to have strong negative implications on therapy adherence and patient-provider relationship, which can exacerbate preexistent health conditions.

Chapter 5: Cardiovascular Disease

Cardiovascular disease, including diseases of the heart and circulatory system, has earned the label as the number one killer in the United States for both men and women and across different racial and ethnic groups (CDC, 2015). According to the Centers for Disease Control and Prevention (CDC), 610,000 Americans die from heart disease each year (CDC, 2015) with men maintaining a higher risk for developing cardiovascular disease than women (Novak, Sandberg, & Harper, 2014). Coronary heart disease is considered to be the most common form of heart disease and the deadliest, claiming the lives of more than 370,000 people each year (CDC, 2015). And as of 2010, 15 million individuals have a history of CHD and 7.6 million had suffered a myocardial infarction (Redmond et al., 2013).

Two common forms of cardiovascular disease include, arteriosclerosis, in which the arteries lose their elasticity, or more commonly, atherosclerosis; in which cholesterol or fats build up (plaques) on the walls of the arteries restricting blood flow. Both disease processes and their interaction contribute to restrictive blood flow in the arteries and potential occlusions of arteries, particularly in the brain (stroke) and heart (myocardial infraction and Angina Pectoris) (Straub, 2012). A number of risk factors have been identified for these disease processes, including hypertension, high LDL cholesterol, diabetes, obesity, inflammatory processes, and smoking (CDC, 2015). In addition, negative emotions and psychosocial environment have also been found to have significant impact on cardiovascular health (AHA, 2014; Watson & Pennebaker, 1989).

Psychological factors including anger, anxiety, depression, stress as well as personality traits (e.g. dispositional hostility) have been implicated as risk factors for cardiovascular disease. For example, Mussleman, Evans, and Nemeroff (1998) found that the diagnosis of depression in patients suffering from cardiovascular disease ranges from 16% to 23% irrelevant of the severity of the disease. Furthermore, people who suffer from cardiovascular disease and depression have higher rates of cardiovascular morbidity and mortality even after controlling for other causal factors; implicating depression as a direct risk factor for developing CVD (Musselman et al., 1998). Hopelessness, a main trait of depression, has been linked to the development of CVD, nearly doubling its risk and mortality levels post CVD events (Rozanski, Blumenthal, & Kaplan, 1999). Physiological differences that predispose individuals, particularly those with depression, to increased risks of cardiac events include reduced heart rate variability and increased activity of the sympathetic nervous system (Musselman et al., 1998).

A positive relationship has been found between high levels of anxiety and sudden cardiac death, more so than other cardiac events (e.g. myocardial infarction). Panic disorders and worry have also been studied within the context of cardiovascular disease with many studies pointing to possible positive associations (Rozanski et al., 1999). In addition, personality traits have been heavily linked to cardiovascular disease, in particular with hostility as a dispositional trait. Hostile individuals have higher blood pressure and heart rate than non-hostile individuals, putting them at a greater risk for developing cardiovascular disease such as coronary artery disease (Rozanski et al., 1999). Overall, disposition hostility can directly impact cardiovascular health through a number of mechanisms, including over activation of sympathetic nervous system (maintenance of the flight or flight reaction of the body), platelet aggregation (blood clotting), reduced heart rate variability, and autonomic regulation of cardiac rhythm (kubazansky & Kawachi, 2000) or indirectly by inducing maladaptive behaviors such as smoking and maintaining an unhealthy diet.

Even though negative emotions can be considered as by-products of stress, stress can be considered as a separate risk factor for developing CVD. Stress can be viewed as a response or perception of a negative event (response) or as an event in itself (stressor) that poses a challenge or threat to a person. The responses can be psychological in nature (depression, anxiety) or physiological in nature (sympathetic nervous system activation) (Peter & Siegrist, 1999). More recently, newer risk factors for cardiovascular disease have been identified. Alterations to cardiovascular reactivity and recovery have been associated with increases in adverse cardiovascular-related health conditions. While many physiological and psychological processes have been found to link psychological stress to cardiovascular disease (e.g. inflammation), for the purpose of this study, cardiovascular reactivity to stressors is of particular interest.

Cardiovascular Reactivity

Cardiovascular reactivity constitutes changes in cardiovascular activity measurements in response to challenges or stressors. Heart rate, systolic blood pressure, diastolic blood pressure, arterial mean pressure, and impedance cardiography (stroke volume, cardiac output, total peripheral resistance) are among the measures used to detect cardiovascular reactivity. Exaggerated cardiovascular reactivity has been established as a risk factor contributing to the development of cardiovascular diseases, especially when co-occurring with other risk factors such as obesity and hypertension (Countryman, Saab, Schneiderman, McCalla, & Llabre, 2014). Furthermore, according to the Reactivity Activation model, individuals who respond to laboratory stressors with elevated cardiovascular responses are more likely to display similar trends in everyday situations (Countryman et al., 2014).

There is mixed literature on the ideal way of measuring cardiovascular changes occurring between baseline and the challenge period, especially due to the variation in baselines across different individuals. However, utilizing multiple cardiovascular measures to assess reactivity allows for the examination of a composite pattern of cardiovascular responses as it pertains to the specific individual (Countryman et al., 2014). This yields a more useful depiction of stress-related cardiovascular reactivity.

Blood Pressure

Blood pressure (BP) is a biological variable used to identify the force by which blood pumped by the heart is exerting pressure against the arteries in the circulatory system (Poulter, Prabhakaran, & Caulfield, 2015). It is partly influenced by heredity, as studies estimate that 30% of BP variance is impacted by genetic factors (Poulter et al., 2015). It is mainly measured by systolic blood pressure and diastolic blood pressure. However, mean arterial pressure and pulse pressure readings are also used for the assessment of BP. Systolic blood pressure measures the pressure in the arteries when the heart pumps blood (beats), whereas diastolic blood pressure measures the pressure in the arteries during the heart's resting period between beats.

Internationally dictated cutoff point for a diagnosis of high blood pressure or hypertension is a systolic blood pressure of at least 140 mg Hg and a diastolic blood pressure of at least 90 mm Hg (Poulter et al., 2015). Environmental causes for high blood pressure include high sodium, calorie, and alcohol intake (Poulter et al., 2015). Psychological stress is also a widely implicated environmental influence in the development of hypertension (Fauvel et al., 2003).

Hypertension has been identified as the main contributor to disease load and mortality globally, leading to around 9.4 million deaths each year (Poulter et al., 2015). These deaths are often mediated through consequent kidney complications, central nervous system damage, and cardiovascular disorders; with the risks for development increasing as the blood pressure values increase (Ortiz, 2012). According to the International Society of Hypertension, blood pressure-related diseases account for half of the global cardiovascular-disease burden (Zmuda & Kammerer, 2008). Furthermore, women who displayed daily increase in cardiovascular reactivity in response to emotional conflicts and novel situations tended to be at a higher risk of developing hypertension (Lavoie, Miller, Conway, & Fleet, 2001).

Data from the Oxford Vascular Study point to the large role of episodic hypertension, detected through blood-pressure variability, in stroke and transient ischemic attack incidents (Rothwell et al., 2004). The study also found that episodic hypertension that is characterized by a very low mean systolic BP posed a greater risk for developing adverse cardiovascular outcomes than constant high mean blood pressure. The results suggest that individuals with highly variable BP might be at a higher risk and measures should be instated for the proper and thorough assessment of BP (Rothwell et al., 2004). Few studies have claimed that BP variability is reflected upon by heart rate variability, however, a large number of studies have shown distinct variability patterns (James, Bovbjerg, & Hill, 2015).

Heart Rate Variability

Heart rate variability (HRV) reflects the fluctuations of heart rate occurring between consecutive heartbeats (Acharya, Joseph, Kannathal, Lim, & Suri, 2006). It displays the interaction of multiple physiological factors that produce the heart rhythm, particularly the interaction and balance between the sympathetic and parasympathetic nervous system. Parasympathetic nervous system alters heart rate through the release of acetylcholine and consequent inhibition of hyperpolarization, whereas the sympathetic nervous system controls heart rate through the release of epinephrine and norepinephrine and faster depolarization (Task Force, 1996). Due to its noninvasive nature and useful diagnostics, HRV analysis is increasingly being employed in the assessment of the autonomic nervous system and overall cardiovascular health. Extensive literature has established a link between autonomic nervous system functioning and mortality due to cardiovascular complications (Acharya et al., 2006). Studies have found HRV to be heavily receptive to environmental changes including stress and physical activity (Goedhart, Der Sluis, Houtveen, Willemsen, & De Geus, 2007).

HRV can be measured using multiple methods. The most common methods are mainly divided into time domain and frequency domain measures. Nonlinear domain measures are less commonly used and will not be examined in this paper. Both measures, time and frequency domain measures, can be derived through ambulatory monitoring which makes the analysis of cardiovascular reactivity in controlled laboratory settings possible (Goedhart et al., 2007). Complete electrocardiogram recordings can be analyzed, however calculating variables across different preselected portions of the recording (5 minute speech task) allows for comparisons between different activities (Task Force, 1996).

Time Domain Measures:

Time domain measures are considered the simplest methods to assess HRV. The measures are further divided into short-term variability (STV) and long-term variability (LTV) indices. Both indices are calculated based on a preselected time slot (e.g. 5 minutes) and utilize RR intervals (time between heart beats; heart rate). STV indices represent the instantaneous beat-to- beat fluctuations in heart rate, whereas LTV indices represent slower heart rate fluctuations. Parameters retrieved from time domain analysis are computed by simple mathematical calculations using RR-interval data (Kim, Kim, Lim, & Park, 2009). They include mean heart rate (NN interval), the standard deviation of the normal-to normal (NN) intervals (SDNN), standard error of the mean (SENN), standard deviation of differences between consecutive NN intervals (SDSD), and the square root of the mean squared differences of successive intervals of NN (RMSSD) and are highly inter-correlated.

SDNN, overall HRV estimate, is also referred to as the square root of variance between NN intervals in a recording. When assessing a 24-hour recording, it reflects short-term high and low frequency variations within that timespan. However, SDNN is affected by the total length of the recording and consequently, as the recording increases so does the variance of HRV (Task Force, 1996). Therefore, short, standardized recordings (e.g. 5 minutes) are ideal to calculate SDNN. RMSSD is another commonly used short-term parameter for high frequency HRV (Task Force, 1996).

Time domain parameters are widely used for the assessment of HRV due to their simplicity, however, they don’t allow for the distinction between sympathetic and parasympathetic contribution to heart rate variability (Acharya et al., 2005).

Frequency Domain Measures:

Examining fluctuations in frequency domains, or spectral parameters, allows for better insight and differential contributions of sympathetic and parasympathetic nervous systems on HRV. Power Spectral Density (PSD) assesses the distribution of heart rate variance based on frequency level. PSD can be calculated using non-parametric and parametric methods. Non-parametric methods are more commonly used due to the simpler mathematical algorithm involved (Fast Fourier Transform algorithm) and faster processing. Short-term spectral parameters constitute power in the very-low frequency range (VLF), power in the low-frequency range (LF), power in the high-frequency range (HF), and low frequency to high frequency power ratio (LF/HF) and are often measured in absolute values of power (ms2) (Task Force, 1996).

There is no consensus among researchers on the physiological mechanisms responsible for heart rate changes of the VLF, and is, therefore, not often utilized in assessing PSD. Extensive literature points to the major influence of parasympathetic nervous system or vagal activity on HF (Task Force, 1996). HF increases has been produced through tasks that are noted to display high involvement of the parasympathetic nervous system such as coordinated breathing, cold stimulation of the face, and rotational stimuli (Task Force, 1996). The agreement on the physiological contributor for LF has not found absolute consensus. A lot of studies point to LF as a quantitative indicator for sympathetic system variations, while others consider it as indicative of both sympathetic and parasympathetic activity (Task Force, 1996). Nonetheless, studies of mental stress and physical exercise have produced increases in LF (Task Force, 1996).

Extensive literature has identified HRV as an independent predictor of all-cause mortality (Task Force, 1996). Low HRV has been associated with various adverse health consequences including diabetes, arthritis, Alzheimer's disease, and cancer (Thayer, Yamamoto, & Brosschot, 2010). It is also associated with a host of cardiovascular-related outcomes such as mortality due to myocardial infarction, coronary heart disease, and hypertension (Scroeder, Whitsel, Evans, Prineas, Chambless, & Heiss, 2004). Studies point to the association between decreased HRV and the predominance of the sympathetic nervous system through the diminishing activity of parasympathetic nervous system after a myocardial infarction (MI) incident (Task Force, 1996). Several hypotheses have been suggested to explain the cardiac changes following MI. One hypothesis suggests that non-functional cardiac tissue can signal an increase in the activity of the sympathetic nerve fibers, and, consequently diminish the activity of vagal fibers that lead to the sinus node (Task Force, 1996). This is further supported by studies that found a decrease in HF and increase in LF power in resting survivors of MI. The resulting autonomic imbalance is highly associated with negative health outcomes.

The Autonomic Imbalance model has been widely used to disseminate the role of HRV in health and well-being. Autonomic imbalance is often characterized by the domination of the sympathetic nervous system paired with the attenuation of the parasympathetic nervous system. When prolonged, this maladaptive pattern can overtax the autonomic nervous system, resulting in numerous physiological pathologies (Thayer et al., 2010). HRV is often used for the assessment of autonomic imbalances. Time and frequency domain measures have been utilized as indices of vagal activity. Furthermore, it was detected that one standard deviation difference in the LF power “was associated with 1.7 times greater relative risk of all cause mortality” using a Framingham Heart Study sample (Thayer et al., 2010, p.123). Low HRV has been associated with cardiovascular disease risk factors such as obesity, smoking, decreased exercise, and diabetes. It has also been associated with psychosocial risk factors for CVD such as negative affect, hostility, and stress, particularly work stress (Thayer et al., 2010). A study by Hintsanen and colleagues (2007) found that young women working in jobs characterized by high effort demands and low rewards endorsed lower levels of RMSSD. Another study found that prolonged work-related stress exposure correlated with HRV measures (Thayer et al., 2010). Furthermore, studies that found associations between decreased HRV measures and work-related stress also found increased rates of cardiovascular-related disorders such as metabolic syndrome and mild hypertension in the same samples.

A large portion of studies identify autonomic imbalance as the common culprit behind all risk factors for CVD. Therefore, most methods aimed at reversing the effects of modifiable risk factors on cardiovascular health involve increasing HRV (Thayer et al., 2010). Meditation, exercise, smoking cessation, and healthy diets have shown favorable results in reducing or eliminating risk factors for CVD and all are associated with increased HRV.

Chapter 6: The Present Study

The role of work related affects in relation to physical health has garnered increased attention in recent years. Emotional reactions to one's environment, especially in the context of the workplace, have been linked to individuals' responses to everyday stressors. Negative affect (NA), including sadness, anger, and fatigue, has been implicated in the adverse impact of stress on health and well-being. Extensive literature points to its associations with modified cardiovascular reactivity and prolonged recovery, negative appraisals of stress, and as a predictor of general increase in adverse health outcomes and mortality (Papousek et al., 2010; Cohen & Pressman, 2006). Positive affect (PA), often neglected in past literature, has also shown strong relations to health consequences through buffering the effects of stress on health and its abilities to "undo" the adverse effects of NA (Fredrickson, 2001). Happiness, excitement, and relaxation are all considered emotions that are positive in nature and share the broad, global favorable implications of PA. However, according to Griskevicius et al. (2010), this overgeneralization of the positive implications of PA across its dimensions, activation and deactivation, may be misleading in both clinical work and research.

In the context of the workplace, Warr's (1990) model of work-related affective well-being ascertains the associations between a wide variety of different emotions experienced at the workplace and outcomes related to one's well being. The model taps onto the two dimensions of the well–validated circumplex model of emotion (Katwyk, 2000). In the circumplex model of emotion (Russell, 1980), affect can be displayed upon two dimensions, valence (positive and negative) and activation arousal (Posner et al., 2005). This model allows us to differentiate between high-arousal and low-arousal PA as well as their respective differentiating effects on health. Activation, according to the literature, equates to physiological arousal, paving the way through which affect can impact physical health (Pressman & Cohen, 2005). And according to Steptoe et al. (2005), psychophysiological pathways further mediate the associations between affect and physical health. Psychosocial factors trigger those biological pathways by initiating central nervous system activation of autonomic, immune, and inflammatory response (Steptoe et al., 2005). Furthermore, Ahn and Shin (2015) argue that although affects that fall under positive valence share the same qualities on valence, they argue that specific positive affects differ on multiple cognitive, behavioral, and motivational functions.

In the context of stressful situations, an association between challenge appraisal and high-arousal PA has been found, but not with low-arousal PA. Given that different appraisals are associated with different patterns of physiological arousal (McGowan et al., 2006), it is feasible that low-arousal PA may have different patterns of cardiovascular activity than highly high-arousal PA. Furthermore, according to the spillover model, PA that is exhibited towards one domain in an individual’s life prolongs into other domains (Armon et al., 2014). It is, therefore, noteworthy to examine if effects of work-related PA are monolithic, if all emotions within PA are identical in terms of their effects on employees’ health, specifically, on stress appraisals and cardiovascular reactivity, for an analysis of sympathetic nervous system differential activation.

Hypotheses of the Present Study

Hypothesis 1: Psychological threat for speech and mental arithmetic stress tasks *will be differentially but negatively predicted by low- arousal and high-arousal work-related positive affect. We anticipate challenge appraisals to both, speech and mental arithmetic, stress tasks will be differentially but positively associated with low-arousal and high-arousal work-related positive affect.*

Hypothesis 2: A positive association will be found between high-arousal work-related PA exhibited over the past 30 days and systolic blood pressure, diastolic blood pressure, low frequency power, low frequency to high frequency ratio, and negative association with high frequency power at baseline. A negative association will be found between low-arousal work-related PA exhibited over the past 30 days and systolic blood pressure, diastolic blood pressure, low frequency power, low frequency to high frequency ratio, and a positive association with high frequency power at baseline.

Hypothesis 3: Cardiovascular (SBP, DBP,HR, LF, LF/HF, HF withdrawal) variability for each stress task (speech and mental arithmetic) will be positively predicted by high arousal work-related positive affect and negatively predicted with low-arousal work-related positive affect .

Chapter 7: Methodology

The final sample for this study included 70 (M= 19.74, SD=3.674) undergraduate students from the University of Michigan- Dearborn. Data was initially collected from 74 participants. From those 74 participants, 4 were excluded based on outlier data. The recruitment procedure followed the standard departmental protocol. Participants' identifying information were protected by the confidentially agreement and remained anonymous. Upon arrival, each participant read and signed a copy of the informed consent, and was provided with a copy for their records. The participant then completed the Demographics and Screening questionnaire as well as the Positive and Negative Affect Schedule (PANAS) as forms for screening. The Demographics and Screening questionnaire was then reviewed for the exclusion criteria which included a status of unemployment, cardiovascular –related health conditions (e.g. hypertension, diabetes), and medications that can alter cardiovascular functioning (e.g. steroids, SSRIs), and then reviewed PANAS to ensure that the participants fall on high positive affect range. Those who did not meet the criteria were thanked for their time and dismissed. The final sample included participants who provided informed consent, did not meet the exclusionary criteria, and did not obtain a high score on the negative affect dimension of PANAS.

Upon determination of eligibility, measurements of height and weight were taken. The researcher then briefly informed the participant of the process of the ECG electrodes application and then, after prepping the sites, applied one electrode to each wrist and one to the left in a 2-lead configuration. Researchers were trained on the proper methods of preparation, application, and removal of the physiological instrumentations (electrocardiogram electrodes and blood pressure cuff). Using a Critikon 8100 System, the blood pressure cuff was applied on the participant's non-dominant arm by the researcher. The participant was then instructed to stay seated without moving for the next 10 minutes to collect baseline physiological data. A total of 4 blood pressure/heart rate readings were collected in the baseline period.

Upon the completion of the 10-minute baseline, the researcher introduced the Trier Social Stress Test (TSST) in segments. In the first segment, the researcher informed the participant that they have 5 minutes to prepare a 5-minute speech describing why he/she would be a good candidate for their ideal job. The researcher then appeared to switch on a video camera (although it was not used) in order to amplify the stress response and told them they now have to start the speech, but before they started it, the Stress Appraisal Measure (speech task) was presented. The speech was presented in front the two researchers and the presentation was "mock" video-recorded (no video recording actually took place). The researcher informed the participant that a performance analysis would be conducted on the video recorded presentation at a later time. In the second segment, the researcher informed the participant of the mental arithmetic task in which the participant has to sequentially subtract the number 7 from a predetermined large number. The Stress Appraisal Measure (mental arithmetic task) was presented prior to the start of the task. A total of 3 blood pressure and heart rate readings were collected in every task.

The TSST is widely used in studies investigating biological pathways through which stress can impact health. It has been designated as the most standardized protocol for inducing psychological stress in controlled lab settings, effectively inducing elevations in several physiological stress markers, including cardiovascular markers. The research assistants were trained in providing nonverbal neutral and negative feedback essential for the success of the stress inducement tasks compromising the TSST. The protocol was be terminated if, at any point during the tasks, participants became overly upset or tearful, defined as tearfulness beyond what would normally occur during an emotionally sad movie, and were provided with the debriefing form, which included a list of sources.

Upon completion of the mental arithmetic task, physiological data collection was suspended. The researcher removed the blood pressure cuff and electrodes and then provided the participant with the JAWS. Upon the completion of the scale, the researcher provided the participant with the debriefing form. The researcher informed the participant of the deceptions in regards to the video recording and that he/she is free to withdraw data. The debriefing form also included information regarding the UM-counseling center if their services are needed as well as contact information of the advisor for any inquiries or questions.

Data Analysis Plan

Descriptive statistics and associations between all variables were examined using correlations. Initial manipulation checks (T-tests) were completed to ensure that the stress task did elicit corresponding changes in physiological activity and to examine any significant group differences in the stress responses between males and females. After controlling for covariates (gender, BMI), three separate hierarchical linear regressions were completed to test the three hypotheses. Separate analyses were conducted for the speech task and mental arithmetic task. For the first hypothesis, high-arousal PA, low-arousal PA, and the interaction term were included as predictor variables, whereas challenge and threat appraisals were included as outcome variables. In the second hypothesis, a correlation analysis was conducted to examine the association between high-arousal PA and low-arousal PA with basal cardiovascular markers (SBP, DBP, HR, LF, HF, LF/HF). For the third hypothesis, high-arousal PA, low-arousal PA, and their interaction term were included as predictor variables, and cardiovascular reactivity markers (SBP, DBP, HR, LF, HF, LF/HF) were included as outcome variables.

Measures

Demographics and Screening Questionnaire: The Demographics and Screening questionnaire was administered to all participants. Age, ethnicity, and employment status and type were collected for research purposes and to ensure that all participants are employees. The questionnaire also screened for health conditions and certain medications that are known to impact cardiovascular functioning. Participants' height and weight was collected and used to calculate Body mass index (BMI). Research points to the importance of controlling for BMI in studies involving cardiovascular functioning.

Cardiovascular Assessment: Cardiovascular reactivity is assessed by utilizing blood pressure readings (systolic and diastolic), heart rate readings, low frequency, high frequency, and low frequency to high frequency ratio that are obtained during baseline, speech task, and math task. Blood pressure readings were collected by a Critikon Dinamap Vital Signs Monitor 1846sxp automated blood pressure machine. Heart rate variability variables were collected using an electrocardiogram, a Biopac MP150 system. A Biopac system EL503 EKG/ECHO, Stress Gel Vinyl 1-3/8" Electrodes were attached on the participants' extremities (right wrist, left wrist, and left ankle). Acqknoweldge version 4.1 was used to record the ECG data. Data was then transported to Kubios for heart rate variability analysis.

Stress Appraisal Measure: The 12-item Stress Appraisal Measure taken from the original Stress Appraisal Measure (SAM; Peackock &Wong, 1990) will be administered on two different occasions during the study, prior to the speech and math tasks. The 12-item measure taps onto two subscales, challenge and threat, that assess cognitive appraisals of anticipatory stress (Peacock & Wong, 1990). Each subscale consists of 4 items with likert-type scale and responses range from "not at all" to "Extremely". Stressful appraisals can be classified as threatening (lack of coping resources) or challenging (chance for mastery and growth). Furthermore, challenge appraisals are related to problem-focused coping, whereas threat appraisals are connected to emotion-focused coping methods (Peacock &Wong, 1990). Internal reliability is adequate (α: ranging from .74-.90) (Peacock & Wong, 1990). In the current study, Cronbach's alpha for overall SAM 1 (speech task) was .74; Cronbach's alpha was .73, .71, and .62 for challenge, stressfulness, and threat respectively. Cronbach's alpha for overall SAM 2 was .79; Cronbach's alpha was .82, .84, and .76 for challenge, stressfulness, and threat respectively.

Positive and Negative Affective Schedule: The Positive and Negative Affective Schedule (PANAS; Watson et al, 1988) is utilized as a screening tool to exclude participants who fall on the negative affect dimension. The measure consists of 20 items with a 5-point likert-type scale and responses range from “very slightly” to “not at all”. The PANAS consists of two scales, positive affect and negative affect. Research reports adequate reliability and validity with Cronbach’s alpha (α=.86 -.90) for the positive affect scale and (α=.84-.87) for the negative affect scale (Schmukle et al., 2002). In the current study, Cronbach’s alpha for overall PANAS was .82. Cronbach’s alpha for positive affect was .88 and Cronbach’s alpha for negative affect was .78.

Work-Related Positive Affect: The Job-Related Affective Well-Being Scale (JAWS; Katwyk, Spector, Fox, & Kelloway, 2000) is used to assess emotional dispositions towards work. The measure is based off of Russell’s circumplex model (Russell, 1980), the two-dimensional model of emotions (valence and arousal) that has been empirically validated. Unlike the Positive and Negative Affective Schedule (PANAS) which provides a general two-domain structure, JAWS taps into four main subscales with four items each: high-arousal NA, high-arousal PA, low-arousal NA, low-arousal PA. It also assesses context-specific affect (Warr, 1990). Internal consistency reliability is adequate (α: total JAWS:.95; high-arousal negative affect:.80 ;high-arousal positive affect:.90; low-arousal negative affect:.80; low-arousal positive affect:.81). In the current study, Cronbach’s alpha for overall JAWS was .69. Cronbach’s alpha for high-arousal work-related positive affect was .86 and Cronbach’s alpha for low-arousal work-related positive affect was .80.

Chapter 8: Results

Data from 70 participants were included in the final statistical analyses (M=19.74, SD= 3.674) for the current study. Descriptive statistics for Age, BMI, JAWS- High Positive- High Arousal, JAWS- High Positive- Low Arousal, SAM-Threat, SAM-Challenge, SAM- Stressfulness, Baseline Systolic Blood Pressure, Baseline Diastolic Blood Pressure, Baseline Heart Rate Variability variables, Speech Systolic Blood Pressure, Speech Diastolic Blood Pressure, Speech Heart Rate Variability variables, Mental Arithmetic Systolic Blood Pressure, Mental Arithmetic Diastolic Blood Pressure, and Mental Arithmetic Heart Rate Variability variables are presented in Table 1 below. An assessment of skewness and kurtosis has been conducted in the sample. A total of four variables presented extreme skewness and were transformed using a natural logarithm; Baseline HF (ms^2), Speech HF (ms^2), Mental Arithmetic HF (ms^2), and Speech LF/HF Ratio. Both transformed and non-transformed variables were used in the regressions and no differences in results were found. All results will be interpreted using non-transformed variables for better understanding.

Hypothesis 1: Psychological threat for speech and mental arithmetic stress tasks will be differentially but negatively predicted by low- arousal and high-arousal work-related positive affect. We anticipate challenge appraisals to both, speech and mental arithmetic, stress tasks will be differentially but positively associated with low-arousal and high-arousal work-related positive affect.

A series of hierarchical multiple regression analyses as well as correlation analyses were conducted to examine the effect of work related positive affect on predicting appraisals of threat and challenge. Hierarchical multiple regressions between high arousal positive affect, low arousal positive affect, the interaction between high and low arousal positive affect and appraisals of threat and challenge were conducted for speech and mental arithmetic tasks and results are displayed in Table 5 below. Furthermore, correlation analysis for speech task is displayed in Table 6 and correlation analysis for mental arithmetic task are displayed in Table 7. We hypothesized that those reporting high-arousal work-related positive affect would be more likely to appraise the stress tasks as challenging, however, this was not supported in the current study. No associations were found between work-related positive affect (both high and low arousal) and appraisals of threat and challenge during the speech task. Likewise, no associations were found between work-related positive affect (high and low arousal) and appraisals of threat and challenge for mental arithmetic task.

Hypothesis 2: A positive association will be found between high-arousal work-related PA exhibited over the past 30 days and systolic blood pressure, diastolic blood pressure, low frequency power, low frequency to high frequency ratio, and negative association with high frequency power at baseline. A negative association will be found between low-arousal work-related PA exhibited over the past 30 days and systolic blood pressure, diastolic blood pressure, low frequency power, low frequency to high frequency ratio, and a positive association with high frequency power at baseline.

A correlational analysis was conducted to assess the association between high-arousal work-related positive affect and SBP, DBP, and heart rate variability variables at baseline. Results are displayed in Table 8 below. We hypothesized that increases in high-arousal work-related positive affect reports would yield to an increase in basal cardiovascular activity; however, this was not supported in the current study. No significant correlations were found between high-arousal work-related positive affect and any of the variables during the baseline period. Moreover, a correlational analysis was conducted to assess the association between low -arousal work-related PA and SBP, DBP, and heart rate variability variables at baseline. No significant correlations were found between low-arousal work-related PA and any of the variables during the baseline period.

Hypothesis 3: Cardiovascular (SBP, DBP,HR, LF, LF/HF, HF withdrawal) variability for each stress task (speech and mental arithmetic) will be positively predicted by high arousal work-related positive affect and negatively predicted with low-arousal work-related positive affect .

Hierarchical linear regressions between high-arousal work-related PA, low-arousal work-related PA, the interaction between high-arousal and low –arousal work-related PA, blood pressure, and cardiovascular reactivity variables are shown below in Table 9 and Table 10. We predicted that a positive association would be present between high-arousal work-related positive affect and blood pressure and cardiovascular reactivity variables during the stress tasks, while a negative association would be present between low-arousal work-related positive affect and the cardiovascular reactivity variables. This hypothesis was partially supported in the current study. There were no significant associations between work-related PA, high-arousal and low-arousal, and systolic blood pressure, diastolic blood pressure, and most cardiovascular reactivity variables during the speech task.

There were several statistically significant and marginally significant associations between work-related positive affect and blood pressure and cardiovascular reactivity variables during the mental arithmetic task that provided partial support to our hypothesis. Low-arousal work-related positive affect was significantly associated with a .67 mmHg-point decrease in diastolic blood pressure during the mental arithmetic task (R^2 =.455, F (5,64)=10.682, $p<.05$). There was also a significant association between the interaction between low-arousal PA and high-arousal PA and diastolic blood pressure (F (R^2 = .491,F (6,63)=10.126, $p<.05$).

The current study also presented some puzzling findings that opposed our predictions. Multiple regression analyses were used to test if high-arousal and low-arousal work-related positive affects predict participants' low frequency as measured in millisecond squared (LFms^2). The results of the regression indicated that the two predictors explain 7% of the variance (R^2=. 401, F (5,64)=8.571p<. 05). However, the directions of the associations do not support our hypothesis. High-arousal positive affect was significantly associated with a 146.655-millisecond squared decrease in LF (ms^2) (β=-146.655, $p<.01$), whereas low-arousal positive affect was significantly associated with a 161.449-millisecond squared increase in LF (ms^2) (β=161.449, $p<.01$). Furthermore, low-arousal and high-arousal work-related positive affect predicted participants' low frequency to high frequency ratio. The results of the regression indicated that the two predictors explain 7% of the variance (R^2=.262, F(5,64)=4.539, $p<.05$). Low-arousal work-related positive affect was significantly associated with a .15

point increase in LF/HF ratio during the mental arithmetic task (β=.152, p<.01), whereas high-arousal PA was marginally significantly associated with a .11 point decrease in LF/HF ratio during the mental arithmetic task (β=-.110, p<.01).

Multiple paired samples T-tests were used to assess blood pressure and heart rate variability data for baseline, speech task, and mental arithmetic task. This manipulation check was conducted to ensure the effectiveness of both stress tasks (speech and mental arithmetic) in eliciting corresponding physiological responses. Results are displayed in Table 2 below. As expected, there was a significant change across all variables (except for speech task-HF (ms^2); marginally significant in mental arithmetic- SDNN) and therefore the stress tasks (speech and mental arithmetic) were effective in eliciting the corresponding physiological responses. The stress tasks remained effective in eliciting corresponding physiological responses when compared across different genders, males and females.

A demographics check was also conducted to assess effects of gender on the stress response. Multiple T-Tests were conducted to compare means between males and females. There was no significant difference found between females and males on SAM-challenge and threat appraisals. Results are displayed in Table 3 below. There was also no significant difference found between females and males on age, diastolic blood pressure (DBP), and most of the heart rate variability variables. Results are displayed in Table 4 below. There was a statistically significant difference between females and males in Systolic Blood Pressure (SBP). Females display lower SBP than males at baseline, speech task, and mental arithmetic task (t(68)=2.544; t(68)=2.728; t(68)=2.948, all p <.01; respectively). There was also a statistically significant difference between females and males in Low Frequency/High Frequency ratio at baseline and mental arithmetic task (LF/HF ratio; $t(65.189)= 2.304$; $t(67.164)=2.286$, all $p<.05$; respectively). Females displayed lower LF/HF ratio than males at baseline and mental arithmetic task. Females displayed statistically significant higher Body Mass Index (BMI) and heart rate (HR) than males at baseline ($t\ (34.658)=-2.120$, $t\ (68)=-2.352$, all $p <.05$; respectively). There was also a statistically significant difference between females and males in Low Frequency in milliseconds squared (LF ms^2), in which females displayed lower LF (ms^2) scores than males at baseline ($t\ (67.728)=2.363$, $p<. 05$). All gender differences were statistically controlled for in the hierarchical regressions.

Chapter 9: Discussion

The purpose of this study was to investigate if the different dimensions of arousal within positive affect, high arousal and low arousal, towards the workplace predict cardiovascular reactivity and cognitive appraisals of threat and challenge in response to laboratory induced psychological stress tasks, a speech task and a mental arithmetic task. Findings presented some significant and partially significant associations between work-related positive affect and the cognitive and physiological variables. Low-arousal work related positive affect was significantly associated with a decrease in diastolic blood pressure during mental arithmetic task. Results also presented some significant findings that opposed our predictions. For instance, low-arousal positive affect predicted an increase in LF (ms^2), an indicator of sympathetic system dominance, a direction that is usually associated with high levels of arousal. These results and others are explored in detail in the section below.

An initial manipulation check was completed to ensure that the stress tasks elicited the anticipated physiological responses in participants. The Trier Social Stress Test (TSST) is one of the most reliable stress protocols for inducing and examining stress response in a lab setting (Allen, Kennedy, Cryan, Dinan, &Clarke, 2014). It consists of elements such as anticipation, lack of control, and social evaluation that can elicit changes in Hypothalamic- Pituitary-Adrenal axis response, immune system response, sympathetic-adrenal –medullary response, cardiovascular response, as well as cognitive assessments of situations (Allen et al., 2014). Results indicated significant changes in blood pressure and heart rate variability data between baseline and the stress tasks. Changes in HF (ms^2) and SDNN were inconsistent across speech task and mental arithmetic task. For instance, changes in HF (ms^2) were significant in the mental arithmetic task but not in the speech task. This is reflected in the mixed literature on the effects of TSST and cardiovascular effects. TSST does not always elicit consistent changes across both tasks in reference to blood pressure and heart rate variability measures (Allen et al., 2014).

A demographics check was also conducted to examine gender differences in the physiological and psychological responses to the stress tasks. Literature cites inconsistent evidence on distinctive physiological reactivity to stressors across different genders (Kelly, Tyrka, Anderson, Price, &Carpenter, 2008). However, the majority of findings on stress report little difference between males and females in physiological responses to stress including cortisol and autonomic reactivity (Kelly et al., 2008). This is consistent with the lack of gender differences in the stress appraisal measures, diastolic blood pressure, and most of the heart rate variability variables shown in the results.

The one distinction that has been consistently supported in the literature is that females experience a higher level of distress in response to a stressor when compared to males. This finding suggests that females may report higher rates of negative affect at a greater intensity than males following a stressor (Kelly et al., 2008). However, according to the literature, different types of stressors pose varying effects on different genders. Interpersonal stressors, stressors with elements of rejection and conflict, are more salient to females and elicit higher elevations in distress and negative affect. Studies utilizing the “social stress paradigm”, as is the current study, tend to include elements of achievement and are more salient to males (Kelly et al., 2008). Furthermore, studies utilizing achievement-based stress tasks have produced higher physiological reactivity in males when compared to females (Kelly et al., 2008). This is reflected in the current results since males displayed significantly higher systolic blood pressure than females during the stress tasks. Males also displayed a higher low frequency to high frequency ratio score, an indication of sympathetic dominance, than females during the mental arithmetic task.

Hypothesis 1

Although studies have found a positive association between challenge appraisals and high arousal PA, this has not been universally supported. There were no associations between high-arousal work-related positive affect and appraisals of challenge and threat in the current study. Likewise, there were no associations between low-arousal work-related positive affect and appraisals of challenge and threat. These results do not support the hypothesis that challenge appraisals will be differentially but positively predicted by high and low arousal positive affect. These results also indicate that the varying levels of arousal in work-related positive affect do not produce distinct differences in appraisals of challenge and threat. An individual with high-arousal work-related positive affect does not significantly differ from an individual with low-arousal work-related positive affect on cognitive appraisals of threat and challenge. An explanation of our lack of findings may be explained by a post hoc analysis that showed that general positive affect, as measured by PANAS, does positively predict challenge appraisals, an adaptive and favorable form of appraisals in stressful situations. Thus general PA may pose a buffering factor against associations between work related levels of affective arousal and cognitive stress appraisals.

Due to lack of significant associations, a post hoc analysis was completed to check for possible associations with the stressfulness scale. Stressfulness is an additional scale included in the Stress Appraisal Measure (SAM) and is considered a form of secondary appraisals with main focus on overall perceived stressfulness and coping. Results showed a significant negative association between low- arousal positive affect and appraisals of perceived overall stressfulness during the mental arithmetic task (F (2,67)=3.065, p<.05), with an R^2 of .084. An increase in low- arousal positive affect predicts a .340 decrease in stressfulness appraisals. There is no significant association between high-arousal work-related PA and the perceived overall stressfulness subscale in both speech and mental arithmetic tasks. Employees exhibiting low-arousal positive affect towards their workplace exhibited less appraisals of stressfulness. There were no association between high-arousal work-related positive affect and overall perceived stressfulness. Unlike challenge and threat appraisals, stressfulness taps into the concept of coping with the stress. It is possible that this finding was only significant in the mental arithmetic task and not the speech task because the participants did not believe they endorsed appropriate coping skills to overcome the mental arithmetic task.

Hypothesis 2

We propose several explanations for the lack of significant findings at baseline, in that no associations between high-arousal and low-arousal work-related positive affect and blood pressure and heart rate variability variables were present. Just like with hypothesis 1, it is possible that general positive affect is posing buffering effects against the associations between log and high work-related positive affect and ambulatory cardiovascular functioning. The lack of findings could also be attributed to the use of linear associations in the current analyses and perhaps a curvilinear association between the different levels of arousal of positive affect and baseline cardiovascular activity may have been a better fit. This is further supported by new research evidence that presented the possibility of a nonlinear relationship between work-related PA and various physiological measures (Armon, Melamed, Berliner, & Shapira, 2013). Findings from a single study revealed a U-shaped association between high-arousal work-related positive affect and basal blood pressure and heart rate. No linear associations were found between high arousal work-related positive affect and blood pressure and heart rate. However, associations were found when using quadratic terms, such that those with moderate levels of high arousal positive affect displayed lower systolic blood pressure and heart rate than those with high and low levels of high-arousal positive affect (Armon et al., 2013).

The curvilinear association between high-arousal positive affect and cardiovascular measures has been explained in the literature by the Vitamin Model (Warr, 2007). The benefits of high arousal positive affect pose most beneficial at moderate levels; extremely low or high levels of high arousal positive affect can have negative long-term effects on health that often accompany prolonged sympathetic nervous system activation (Armon et al., 2013). Non-linear analyses were not utilized in this study and might have been the reason behind the lack of support for the hypothesis that high arousal work-related positive affect would be associated with high systolic blood pressure, high diastolic blood pressure, high low frequency power, high low frequency to high frequency ratio, and lower high frequency power at baseline a negative association would be found between low arousal work-related positive affect and systolic blood pressure, diastolic blood pressure, low frequency power, low frequency to high frequency ratio, and a positive correlation with high frequency power at baseline.

Hypothesis 3

It was also hypothesized that high-arousal work-related positive affect would be associated with increases in blood pressure and heart rate reactivity variables in response to a lab induced psychological stressor whereas, low-arousal positive affect would be associated with decreases in the corresponding variables. This was not reflected during the speech task of the current study. Differential effects on cardiovascular activation across different stress tasks have been outlined in the literature (Lipman, Grossman, Bridges, Hamner, &Taylor, 2002). Several factors, such as difficulty, salience, and physical demands required by the stress tasks have been postulated as the culprits behind different activation profiles across the mental arithmetic and speech stress tasks (Lipman et al., 2002). Another potential explanation for the differences in stress tasks may be because the speech task utilized in this study required students to assume the role of a job applicant applying for their ideal job. This might have not been as salient to first year college students who constituted the majority of participants in this study and who might have not declared future careers yet. Significant associations found during the mental arithmetic could have been due to increased difficulty level (increased cognitive demand as compared to a free speech) as well as performance evaluation.

Significant and marginally significant associations between positive affect and blood pressure and cardiovascular reactivity variables were found during the mental arithmetic task. As we anticipated, there was a significant negative association between low- arousal positive affect and diastolic blood pressure, in such that greater endorsement of low-arousal positive affect in relation to the workplace indicated lower diastolic blood pressure reactivity during a stress task. This has been widely supported in the literature on blood pressure and stress. A post hoc analysis was done to examine the significant association between the interaction of low and high work-related positive affect and diastolic blood pressure. A plot of the interaction is displayed in figure 1 below. The results of the analysis showed that diastolic blood pressure tended to be lower at higher levels of low-arousal work-related positive affect when high-arousal work-related positive affect is low.

Findings of frequency domain measures included some puzzling and contradicting results. There was a statistically significant negative association between high-arousal positive affect and low frequency power as measured in millisecond squared (LF-ms^2), whereas a positive association was present between low-arousal positive and LF (ms^2). Even though, studies of mental stress have seen increases in LF, a main physiological contributor is yet to be identified. A lot of studies point to LF as a quantitative indicator for sympathetic system variations, while others consider it as indicative of both sympathetic and parasympathetic activity (Task Force, 1996). The present results lend support to the possibility that LF spectral component might not be solely controlled by the sympathetic nervous system. It is also possible, given the wide variation present in spectral components within the frequency domain (e.g. LF, HF, LF/HF) and the small sample present in the current study, an issue of inconclusive assessment for the associations between positive affect and the spectral components might be present. It is also possible that participants who endorsed low-arousal work-related positive affect might just be disengaged from tasks at work. So when they encountered a novel task (e.g. mental arithmetic task), they displayed greater engagement and physiological activation. Further analysis would be required to explore this possibility.

Furthermore, we anticipated a positive relationship between high-arousal work related positive affect and low frequency to high frequency ratio (LF/HF ratio). This is not supported in the current study mainly due to the previous finding (negative association between LF and high-arousal work-related positive affect) since LF is used in calculating LF/HF ratio score. High-arousal positive affect is actually marginally associated with a decrease in LF/HF ratio, whereas low-arousal positive affect is significantly associated with an increase in LF/HF ratio.

Strengths and Limitations

Limitations. The current study has a number of notable limitations. The first limitation is that only undergraduate students from the University of Michigan- Dearborn participated in the study. Most of the students were employed on a part-time basis, therefore limiting the generalizability of the findings to full-time employees. Another limitation is that the sample consisted of 70 participants only. Due to the limited time allotted for a master’s thesis, a larger sample was not possible. Furthermore, given the great variation inherent to some spectral components, the current sample might have been too small in order to efficiently conduct the spectral component analyses. Another limitation to the study is that the measure of work-related affect (JAWS) was given outside of the work context. Providing this measure within the place of employment would have yielded more reliable responses.

Strengths. To our knowledge, this is the first study to look at differential effects of high-arousal and low-arousal work-related positive affect on cardiovascular stress reactivity. While few studies have looked at similar effects of positive affect, no study has looked at it with respect to the workplace. Moreover, very few studies have looked at low-arousal positive affect within the context of stress. Current results show a favorable association between low-arousal positive affect and cognitive appraisals of stress and coping, therefore they can have future implications for possible cognitive and physiological targets in formulating stress reduction and management interventions in the workplace.

Another strength of the current study is that, unlike most studies involving physiological measurements, it employed constant monitoring of heart rate and blood pressure rather than typical single readings. Research points to the increased reliability and predictability of cardiovascular outcomes with repeated measures of heart rate and blood pressure (Armon et al., 2014). Furthermore, the current study utilized a validated self-report measure of job-related positive affect (JAWS) rather than the less effective laboratory method of inducing positive emotion. Another strength of the study lies in the strict exclusion criteria. Substance intake, caffeine intake, cardiovascular and psychiatric conditions, and certain medications were controlled for due to their impact on cardiovascular functioning. Therefore, any possible confounding factors were eliminated to ensure that the results reflect a true association between cardiovascular reactivity and the affective experience.

Future Research

Future research is necessary to determine if these results can be generalized to the general public within the workforce. Including data from non-college student, full-time employees can add strength and applicability to the results in the general workforce. Furthermore, future research should include a larger sample with a variety of occupations. Most of the participants in the current study held positions in customer service or retail; it would be interesting to explore if different occupations could elicit different stress responses in individuals through affective experiences at work. Furthermore, given that research evidence points to a possible non-linear associations between high-arousal and low-arousal positive affect and basal cardiovascular variables, future research should attempt to utilize non-linear analyses to explore the differences in responses to stressors. Furthermore, given that this study found that participants who endorsed low-arousal work-related positive affect experienced increases in Low Frequency (ms^2), an indicator often used to detect sympathetic activity dominance, the concept of task engagement may provide an explanation. Previous research have utilized the “interested” term on the Positive and Negative Affective Schedule (PANAS) as an index for task engagement (Chatkoff, Maier, Javaid, Hammoud, & Munkrishna, 2009). Future analyses controlling for task engagement’s effects on the associations between affective experience and physiological activation could potentially help explain the puzzling findings found in this study.

Future research may also want to utilize different stress tasks in assessing the relationship between affective experiences and cardiovascular response. According to the literature, different stressors can elicit different changes in individuals. Using a more salient, targeted stress task can be more effective at eliciting a stronger cardiovascular response. Furthermore, given that high-arousal work-related positive affect did not have significant associations with challenge appraisals, while general positive affect (PANAS) did, it is recommended that future research explore the differences between the two variables especially since the literature highly associates the two with each other. Finally, future research may want to focus on the differential long-term effects of work-related low-arousal and high-arousal on job performance and stability as well as long-term health markers.

References

Acharya, U., Joseph, P., Kannathal, N, Lim, C., & Suri, J. (2006). Heart Rate Variability: A Review. *Medical & Biological Engineering and Computing, 44* (12), 1031-1051.

Ahn, D. & Shin,D. (2015). Differential effect of Excitement versus Contentment, and Excitement versus Relaxation: Examining the influence of Positive Affects on adoption of new technology with a Korean Sample. *Computers in Human Behavior, 50,* 283-290.

Allen, A., Kennedy, P., Cryan, J., Dinan, T., &Clarke, G. (2014). Biological and Psychological Markers of Stress in Humans: Focus on the Trier Social Stress Test. *Neuroscience and Biobehavioral Reviews, 38,*94-124.

Armon, G., Melamed, S., Berliner, S., &Shapira, I. (2014). High Arousal and Low Arousal Work-Related Positive Affects and Basal Cardiovascular Activity. *The Journal of Positive Psychology, 9* (2), 146-154.

Auerbach, S., Gramling, S. (1998). *Stress Management :Psychological Foundations.* Upper Saddle River, New Jersey: Prentice-Hall, Inc.

Baum, A. (1990). Stress, Intrusive Imagery, and Chronic Distress. *Journal of Health Psychology, 9*(6), 653-675.

Bloom,E., Cafiero, T., Jané-Llopis, E., Abrahams-Gessel, S., Bloom, R., Fathima, S., Feigl, B., Gaziano, T., Mowafi, M., Pandya, A., Prettner, K., Rosenberg, L., Seligman, B., Stein, Z., & Weinstein, C. (2011). *The Global Economic Burden of Noncommunicable Diseases*. Geneva: World Economic Forum.

Bosma,H.,Marmont,M.G.,Hemingway,H., Nicholson, A.C. et al. (1997). Low Job control and Risk of Coronary heart disease in Whitehall II: Prospective Cohort Study. *British Medical Journal, 314* (7080), 558-564.

Bostock, S., Hamer, M., Wawrzyniak, A.J., Michell, E.S., & Steptoe, A. (2011). Positive Emotional Style and subjective, cardiovascular and cortisol responses to acute laboratory stress. Psychoneuroendocrinology, 36,1175-1183.

Brewer, L.C. & Cooper, L.A. (2014). Race, Discrimination, and Cardiovascular Disease. The *American Medical Association Journal of Ethics, 16* (6), 455-460.

Brown, D.L. (1990). Disparate Effects of 1989 Loma Prieta and 1994 Northridge Earthquakes on Hospital Admissions for Acute Myocardial Infarction: Importance of Superimposition of Triggers. *The American Heart Journal, 137* (5), 830-836.

Browning, L., Ryan,C.S., Greenberg, M.S., & Rolniak,S. (2005). Effects of Cognitive Adaptation on the Expectation-Burnout Relationship Among Nurses. *Journal of Behavioral Medicine, 29* (2), 139-150.

Bond, F., Bruce, D. (2000). Mediators of Change in Emotion-Focused and Problem-Focused Worksite Stress-Management Interventions. *Journal of Occupational Health Psychology, 5*, 156-163.

Brinkborg, H., Michanek, J., Hesser, H., Bergulnd, G. (2011). Acceptance and Commitment Therapy for the Treatment of Stress Among Social Workers: A Randomized Controlled Trial. *Behavior Research and Therapy, 49,* 389-398.

Byczek, L., Walton, S.M., Conrad, K.M.,Reichelt, P.A.,Samo,D.G. (2004). Cardiovascular Risks in Firefighters. *AAOHN Journal, 52* (2), 66-76.

Cannon, B. (1994). Walter Bradford Cannon: Reflections on the Man and His Contributions. *International Journal of Stress Management, 1,* 145-158.

Centers for Disease Control and Prevention. *Strategies to Prevent Obesity and Other Chronic Diseases: The CDC Guide to Strategies to Increase Physical Activity in the Community.* Atlanta: U.S. Department of Health and Human Services; 2011.

Chatkoff, D., Maier, K., Javaid, J., Hammoud, M., Munkrishna, P. (2009). Dispositional Hostility and Gender Differentially Relate to Cognitive Appraisal, Engagement, and Cardiovascular Reactivity Across Cognitive and Emotional Laboratory Tasks. *Personality and Individual Differences, 47*, 122-126.

Cheng, Y., Park, J., Kim, Y., & Kawakami, N. (2012). The Recognition of Occupational Diseases Attributed to Heavy Workloads: Experiences in Japan, Kora, and Taiwan. *International Archives of occupational and Environmental Health*, *85*, 791-799.

Cohen, S., & Williamson, G. (1988). Perceived stress in a probability sample of the United States. In S. Spacapan, & S. Oskamp (Eds.), *The social psychology of health: Claremont symposium on applied social psychology*. Newbury Park, CA: Sage.

Cohen,S. & Pressman, S.D. (2006). Positive Affect and Health. *Current Direction in Psychological Science, 15*(3), 122-125.

Countryman, A., Saab, P., Schneiderman, N., McCalla, J., & Llabre, M. (2014). Cardiovascular Reactivity and Cardiometabolic Risk in Adolescents. *International Journal of Behavioral Medicine, 21* (1), 122-130.

Coverman, S. (1989). Role Overload, Role conflict, and Stress: Addressing consequences of Multiple Role Demands. *Social forces, 67*(4), 965-982.

Dimsdale, J.E. (2008). Psychological Stress and Cardiovascular Disease. *Journal of the American College of Cardiology, 51*(13), 1237-1246.

Duncan, S. & Barret, L. (2007). Affect is a Form of Cognition: A Neurobiological Analysis. *Cognition and Emotion, 21* (6), 1184-1211.

Eschleman, K., Alacron, G., Lyons, J., Stokes, C., & Schnieder, T. (2011). The Dynamic Nature of the Stress Appraial Process and the Infusion of Affect. *Anxiety, Stress, & Coping: An International Journal, 25* (3), 309-327.

Everly, G. & Lating, J. (2002). *A clinical guide to the treatment of the human stress response* (2nd ed.). New York: Kluwer Academic.

Ewart, C.K., Taylor, C.B., Kraemer, H.C., & Agras, W.S. (1991). High Blood Pressure and Marital Discord: Not being Nasty Matters more than Being Nice. *Health Psychology, 10* (3), 155-163.

Fauvel, J., M'Pio, I., Quelin, P., Rigaud, J., Laville, M., & Ducher, M. (2003). Neither Perceived Job Stress nor Individual Cardiovascular Reactivity Predict High Blood Pressure. *Hypertension, 42* (6), 1112-1116.

Ferroli, C. (1996). Anger could be a Fatal Fault. *The Saturday Evening Post, 268* (1), 18.

Filaretova, L. (2012). Stress in Physiological Studies. *Neuroscience and Behavioral Physiology, 42*, 193-199.

Fitzgerald, S.T., Haythornthwaite, J. A., Suchday, S., & Ewart, C. (2002). Anger in Young Black and White workers: Effects of Job Control, Dissatisfaction, and Support. *Journal of Behavioral Medicine, 26* (4), 283-296.

Folkman, S., Moskowitz, J. (2000). Positive Affect and the Other Side of Coping. *American Psychologist, 55* (6), 647-654.

Fredrickson, B. (2004). The Broaden-and-Build Theory of Positive Emotions. *Philosophical Transactions of the Royal Society B: Biological Sciences, 359* (1449), 1367-1378.

Fredrickson, B.L. (2001). The Role of Positive Emotions in Positive Psychology: The Broaden-and- Build Theory of Positive Emotions. American Psychologist, 56(3), 218-226.

Ganzel, B., Morris, P., & Wethington, E. (2010). Allostasis and the Human Brain: Intergrating Models of Stress from the Social and Life Sciences. *Psychological Review, 117* (1), 134-174.

Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, Dai S, Ford ES, Fox CS, Franco S, Fullerton HJ, Gillespie C, Hailpern SM, Heit JA, Howard VJ, Huffman MD, Judd SE, Kissela BM, Kittner SJ, Lackland DT, Lichtman JH, Lisabeth LD, Mackey RH, Magid DJ, Marcus GM, Marelli A, Matchar DB, McGuire DK, Mohler ER 3rd, Moy CS, Mussolino ME, Neumar RW, Nichol G, Pandey DK, Paynter NP, Reeves MJ, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Wong ND, Woo D, Turner MB; on behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2014 update: a report from the American Heart Association. *Circulation*. 2014;129:e28–e292.

Goedhart, A., Van Der Sluis, S., Houtveen, J., Willemsen, G., De Geus, E. (2007). Comparison of Time and Frequency Domain Measures of RSA in Ambulatory Recordings. *Psychophysiology, 44* (2), 203-215.

Greer, T.M. (2010). Perceived Racial Discrimination in Clinical Encounters among African American Hypertensive Patients. *Journal of Health Care for the Poor and Underserved, 21,* 251-263.

Griskveicius,V., Shiota, M.N., & Nowlis, S.M. (2010). The Many Shades of Roes-Colored Glasses: An Evolutionary Approach to the influence of Different Positive Emotions. *Journal of Consumer Research,* Inc., 37(2), 238-250.

Heffner, K. L., Loving, T.J., Kieclot-Glaser, J.K., Himawan, L.K., Glaser, R., & Malarkey, W.B. (2006). Older Spouses’ Cortisol Responses to Marital Conflict: Association with Demand/Withdraw Communication Patterns. *Journal of Behavioral Medicine, 29* (4), 317-325.

Hickens, M.T., Lee, H., Morenoff, J., House, J.S., & Williams, D.R. (2014). Racial/Ethnic Disparities in Hypertension prevalence: Reconsidering the role of Chronic Stress. *American Journal of Public Health, 104* (1), 117-123.

Hilmert, C., Teoh, A., & Roy, M. (2013). Effort and Negative Affect Interact to Predict Cardiovascular Responses to Stress. *Psychology and Health, 29* (1), 64-80.

Hintsanen, M., Elovainio, M., Puttonen, S., Kivimaki, M., Koskinen, T., Raitakari, O., Keltikangas-Jarvinen, L. (2007). Effort-Reward Imbalance, Heart Rate, and Heart Rate Variability: The Cardiovascular Risk in Young Finns Study. *International Journal of Behavioral Medicine, 14* (4), 202-212.

Hu, J. & Gruber, K. (2008). Positive and Negative Affect and Health Functioning Indicators Among Older Adults with Chronic Illnesses. *Issues in Mental Health Nursing, 29* (8), 895-911.

Ivancevich, J. M., Matteson, M. T., Freedman, S. M., & Phillips, J. S. (1990). Worksite stress management interventions. *American Psychologist, 45,* 252-261.

James, G., Bovbjerg, D., & Hill, L., (2015). Daily Environmental Differences in Blood

Pressure and Heart Rate Variability in Healthy Premenopausal Women. *American Journal of Human Biology, 27* (1), 136-138.

Jiang W, Babyak M, Krantz D, et al. (1996) Mental stress-induced myocardial ischemia and cardiac events. *The Journal of the American Medical Association, 275,* 1651-1656.

Katwyk, P.V., Spector, P.E., Fox, S., Kelloway, E.K. (2000). Using the Job-Related Affective Well- Being Scale (JAWS) to investigate Affective Responses to Work Stressors. *Journal of Occupational Health Psychology, 5*(2), 219-230.

Kelly, M., Tyrka, A., Anderson, G., Price, L., & Carpenter, L. (2008). Sex Differences in Emotional and Physiological Responses to the Trier Social Stress Test. *Journal of Behavior Therapy and Experimental Psychiatry, 39* (1), 87-98.

Kiecolt-Glaser, J.K., Loving, T.J., Stowell, J.R., Malarkey, W.B., Lemeshow, S., Dickinson, S.L., & Glaser, R. (2005). Hostile Marital Interactions, Pro inflammatory, Cytokine Production, and Wound Healing. *Archives of General Psychiatry, 62,* 1377-1384.

Kiecolt-Glaser, J.K. & Newton, T.L. (2001). Marriage and Health: His and Hers. *Psychological Bulletin, 127* (4), 472-503.

Kim, K., Kim,J., Lim, Y., & Park, K. (2009). The Effect of Missing RR-Interval Data on Heart Rate Variability Analysis in the Frequency Domain. *Physiological Measurement, 30* (10), 1039-1050.

Kubzansky, L. D. & Kawachi, I. (2000). Going to the Heart of the Matter: Do Negative Emotions Cause Coronary Heart Disease? *Journal of Psychosomatic Research, 48,* 323-337.

Kuperman, V., Estes, Z., Brysbaert, M., & Warriner, A. (2014). Emotion and Language: Valence and Arousal Affect Word Recognition. *Journal of Experimental Psychology: General, 143* (3), 1065-1081.

Kuppens, P., Tuerlinckx, F., Russell, J., & Barrett L. (2013). The Relation Between Valence and Arousal in Subjective Experience. *Psychological Bulletin, 139* (4), 917-940.

Lavoie, K., Miller, S., Conway, M., Fleet, R. (2001). Anger, Negative Emotions, and Cardiovascular Reactivity During Interpersonal Conflict in Women. *Journal of Psychosomatic Research, 51* (3), 503-512.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping.* New York, NY: Springer Publishing Company.

Lee, S., Colditz, G., Berkman, L., & Kawachi. I. (2002). A Prospective study of Job Strain and coronary heart disease in US women. *International Journal of*

Epidemiology, 31,1147-1153.

Lee, E. (2012). Review of the Psychometric Evidence of the Perceived Stress Scale. *Asian Nursing Research, 6* (4), 121-127.

Levi,L. (1990). Occupational Stress: Spice of Life or Kiss of Death. *American Psychologist, 45,* 1142-1145.

Lewis, S. J., & Roberts, A. R. (2001). Crisis assessment tools: The good, the bad, the available. *Brief Treatment and Crisis Intervention, 1*, 17–28.

Lipman, R., Grossman, P., Bridges, S., Hamner, J., & Taylor, J. (2002). Mental Stress Response, Arterial Stiffness, and Baroreflex sensitivity in Healthy Aging. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 57* (7), B279-B284.

Lloyd, J., Bond, F., Flaxman, P. (2013). The Value of Psychological Flexibility: Examining Psychological Mechanisms Underpinning a Cognitive Behavioral Therapy Intervention for Burnout. *Work and Stress, 27,* 181-199.

Lowe, B., Decker, O., Muller, S., Brahler, E., Schellberg, D., Herzog, W., & Herzberg, P. (2008). Validation and Standardization of the Generalized Anxiety Disorder Screener (GAD-7) in the General Population. *Medical Care, 46* (3), 266-274.

Lundberg, U., Mardberg, B., & Frankenhaeuser, M. (1994). The Total Workload of male and female white collar workers as related to age, occupational level, and number of children. *Scandinavian Journal of Psychology, 35,* 315-327.

Luoma, J., Hayes, S., Walser, R. (2007). *Learning ACT: An Acceptance and Commitment Therapy Skill-Training Manual for Therapists*. Oakland,CA: New Harbinger Publications, Inc.

Maroufizadeh, S., Zareiyan, A., Sigari, N. (2014). Reliability and Validity of Persian Version of Perceived Stress Scale (PSS-10) in Adults with Asthma. *Archive of Iranian Medicine, 17* (5), 361-365.

Marshall, N.L. & Barnett, R.C. (1993). Work-Family Strains and Gains among Two-Earner Couples. *Journal of Community Psychology, 21,* 64-78.

Marshall, L, Barnett, C., Sayer, A. (1997). The Changing Workforce, Job Stress, and Psychological Distress. *Journal of Occupational Health Psychology, 2,* 99-107.

Maslach,C., Schaufeli,W.B., & Leiter,M.P. (2001). Job Burnout. *Annual Review of Pscyhology 52,397-422.*

Matthieu, M. & Ivanoff, A. (2006). Using Stress, Appraisal, and Coping Theories in Clinical Practice: Assessments of Coping Strategies After Disasters. *Brief Treatment and Crisis Intervention, 6* (4), 337-348.

McGowan, J., Gardner, D., & Fletcher, R. (2006). Positive and Negative Affective Outcomes of Occupational Stress. New Zealand Journal of Psychology, 35(2), 92-98.

McLachlan, C., Ocsan, R., Spence, I., Brett, H., Matthews, S., Wang, L., Herbert, J. (2010). Increased Total Heart Variability and Enhanced Cardiac Vagal Autonomic Activity in Healthy Humans with Sinus Bradycardia. *Baylor University Medical Center Proceedings, 23* (4), 368-370.

Meier-Ewert, H.K., Ridker, P. M. Rifai, N., Regan, M.M., Price, N.J., Dinges, D.F., Mullington, J.M. (2004). Effect of Sleep Loss on C-Reactive Protein, an Inflammatory Marker of Cardiovascular Risk. *Journal of American College of Cardiology, 43* (4), 678-683.

Musselman, D. L., Evans, D. L., & Nemeroff, C. B. (1998). The Relationship of Depression to Cardiovascular Disease. *Archives of General Psychiatry 55*, 580-592.

Nealey-Moore, J.B., Smith, T.W., Uchino, B.N., & Hawkins, M.W. (2007). Cardiovascular Reactivity during Positive and Negative Marital Interactions. *Journal of Behavioral Medicine, 30*, 505-519.

Novak, J.R., Sandberg, J.G., & Harper, J.M. (2014). Older Couples with and without Cardiovascular Disease: Testing Associations between and among Affective Communication, Marital Satisfaction, Physical and Mental Health. *American Psychological Association: Families, Systems, and Health, 32* (2), 186-197.

Oken, B.S., Fonareva, I., & Wahbeh, H. (2011). Stress-Related Cognitive Dysfunction in Dementia Caregivers. Journal of Geriatric Psychiatry and Neurology, 24, 191-198.

Orly, S., Rivka, B., Rivka, E., Dorit, S. (2012). Are Cognitive-behavioral Interventions Effective in Reducing Occupational Stress Among Nurses? *Applied Nursing Research, 25*. 152-157.

Ory, M., Ahn, S., Jiang, L., Lorig, K., Ritter, P., Laurent, D., Whitelaw, N., Smith, M. (2013). National Study of Chronic Disease Self-Management: Six-Month Outcome Findings. *Journal of Aging and Health, 25* (7), 1258-1274.

Orth-Gomer, K., Wamala, S.P., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittleman, M.A. (2000). Marital Stress Worsens Prognosis in Women with Coronary Heart Disease: The Stockholm Female Coronary Risk Study. *The Journal of the American Medical Association, 284* (23), 3008-3014.

Ortiz, A. (2012). Blood Pressure. *Clinical Therapeutics, 34* (4S), e12-213.

Papousek, I., Naushnegg, K., Paechter, M., Lackner, H.K., Goswami, N., & Shulter, G.(2010). Trait and State Positive affect and cardiovascular recovery from experimental academic stress. *Biological Psychology, 83*,108-115.

Peacock, E., & Wong, P. (1990). The Stress Appraisal Measure (SAM): A Multidimensional Approach to Cognitive Appraisal. *Stress Medicine, 6,* 227-236.

Peter, R. & Siegrist, J. (1999). Chronic Psychosocial Stress at Work and Cardiovascular disease: The Role of Effort-Reward Imbalance. *International Journal of Law and Psychiatry, 22* (5-6), 441-449.

Posner, J., Russell, J., & Peterson, B. (2005). The Circumplex Model of Affect: An Integrative Approach to Affective Neuroscience, Cognitive Development, and Psychopathology. *Development and Psychopathology, 17* (3), 715-734.

Poulter, N., Prabhakaran, D., & Caulfield, M. (2015). Hypertension. *Lancet, 386* (9995), 801-812.

Pressman, S.D. & Cohen, S. (2005). Does Positive Affect Influence Health? Psychological Bulletin, 131(6), 925-971.

Puttonen, S., Harma, M., & Hublin, C. (2010). Shift Work and Cardiovascular disease-pathways from circadian stress to morbidity. *Scandinavian Journal of Work, Environment & Health, 36* (2), 96-108.

Quick, C.J.., Wright, T., Adkins, J., Nelson, D., Quick, D. J. (2013). *Preventive Stress Management in Organizations*. Washington, DC: American Psychological Association.

Redmond, N., Richman, J., Gamboa, C.M., Albert, M.A., Sims, M., Durant, R.W., Glasser, S. P., & Safford, M.M. (2013). Perceived Stress is associated with Incident Coronary Heart Disease and All-Cause Mortality in Low- but not High-Income participants in the Reasons for Geographic and Racial Differences in Stroke Study. *Journal of the American Heart Association,2*(6):e000447. doi: 10.1161/JAHA.113.000447.

Robles, T., Brooks, K., & Pressman, S. (2009). Trait Postiive Affect Buffers the Effects of Acute Stress on Skin Barrier Recovery. *Health Psychology, 28* (3), 373-378.

Rothwell, P., Coull, A., Giles, M., Howard, S., Silver, L., Bull, L., Gutnikov, S., Edwards, P., Mant, D., Sackley C., Farmer, A., Sandercock, P., Dennis, M., Warlow, C., Bamford, J., Anslow, P., Oxford Vascular Study. (2004). Change in Stroke Incidence, mortality, case- fatality, severity, and risk factors in Oxfordshire, UK from 1981 to 2004 (Oxford Vascular Study). *Lancet, 363* (9425), 1925-1933.

Rozanski, A., Blumenthal, J., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*, 2192-2217.

Russell, J. A. (2003). Core Affect and the Psychological Construction of Emotion. *Psychological Review, 110* (1), 145-172.

Russell, J.A. (1980). A Circumplex model of Affect. *Journal of Personality and Social Psychology, 39*, 1161-1178.

Schmuckle, S.C., Elgoff, B., & Burns, L.R. (2002). The Relationship Between Positive and Negative Affect in the Positive and Negative Affect Schedule. *Journal of Research in Personality, 36* (5), 463-475.

Schroeder, E., Whitsel, E., Evans, G., Prineas, R., Chambless, L., & Heiss, G. (2004). Repeatability of Heart Rate Variability Measures. *Journal of Electrocardiology, 37* (3), 163-172. Relationship

Smith, T.W , Baron, C.E., & Grove, J.L. (2013). Personality, Emotional Adjustment, and Cardiovascular Risk: Marriage as a Mechanism. *Journal of Personality, 82* (6), 502-514.

Sparen, P., Vagero, D., Shestov, D., et al. (2004). Long Term Mortality After Severe Starvation During the Siege of Leningrad: a Prospective Cohort sSudy. *British Medical Journal, 328* (7430) 11–14.

Stein, G.L., Kiang, L., Supple, A.L., & Gonzalez, L.M. (2014). Ethnic Identity as a Protective Factor in the Lives of Asian American Adolescents. *Asian American Journal of Psychology, 5* (3), 206-213.

Steptoe, A., Wardle, J., & Marmot, M. (2005). Positive Affect and Health-Related Neuroendocrine, Cardiovascular, and Inflammatory Processes. Proceedings of the National Academy of Sciences of the United States of America,102 (18),6508-6512.

Straub,R.O. (2012) *Health Psychology: A Biopsychosocial Approach.* New York, NY: Worth Publishers.

Task Force of the European Society of Cardiology & the North American Society of Pacing and Electrophysiology (1996). Heart rate variability. *European Heart Journal, 17,* 354-381.

Thayer, J., Yamamoto, S., & Brosschot, J. (2010). The Relationship of Autonomic imbalance, Heart Rate Variability and Cardiovascular Disease Risk Factors. *International Journal of Cardiology, 141* (2), 122-131.

Thoits, P. (2010). Stress and Health: Major Findings and Policy Implications. *Journal of Health and Social Behavior, 51,* S41- S53.

Vaessen, T., Hernaus, D., Myin-Germeys, I., & Amelsvoort, T. (2015). The Dopamingergic Response to Acute Stress in Health and Psychopathology: A Systematic Review. *Neuroscience and Behavioral Review, 56*, 241-251.

Warr,P. (1990). The Measurement of Well-being and other aspects of Mental Health. *Journal of Occupational Psychology, 63,*193-210.

Warr, P. (2007). *Work, Happiness, and Unhappiness.* Mahwah, NJ: Erlbaum.

Watson, D. & Clark, L. (1984). Negative Affectivity: The Disposition to Experience Aversive Emotional States. *Psychological Bulletin, 96* (3), 465-490.

Watson, D., Clark, L., & Tellegen, A. (1988). Development and Validation of Brief Measures of Postive and Negative Affect: The PANAS Scales. *Journal of Personality and Social Psychology, 54 (*6), 1063-1070.

Watson, D., & Pennebaker, J. (1989). Health Complaints, Stress, and Distress: Exploring the Central Role of Negative Affectivity. *Psychological Review, 96* (2), 234-254.

Wright, R., Kirby, L. (2003). Cardiovascular Correlates of Challenge and Threat Appraisals : A Critical Examination of the Biopsychosocial Analysis. *Personality and Social Psychology Review, 7* (3), 216-233.

Zmuda, M. , Kammerer, M. (2008). Snipping Away at Osteoporosis Susceptibility. *Lancet, 371,* 1479-1480.

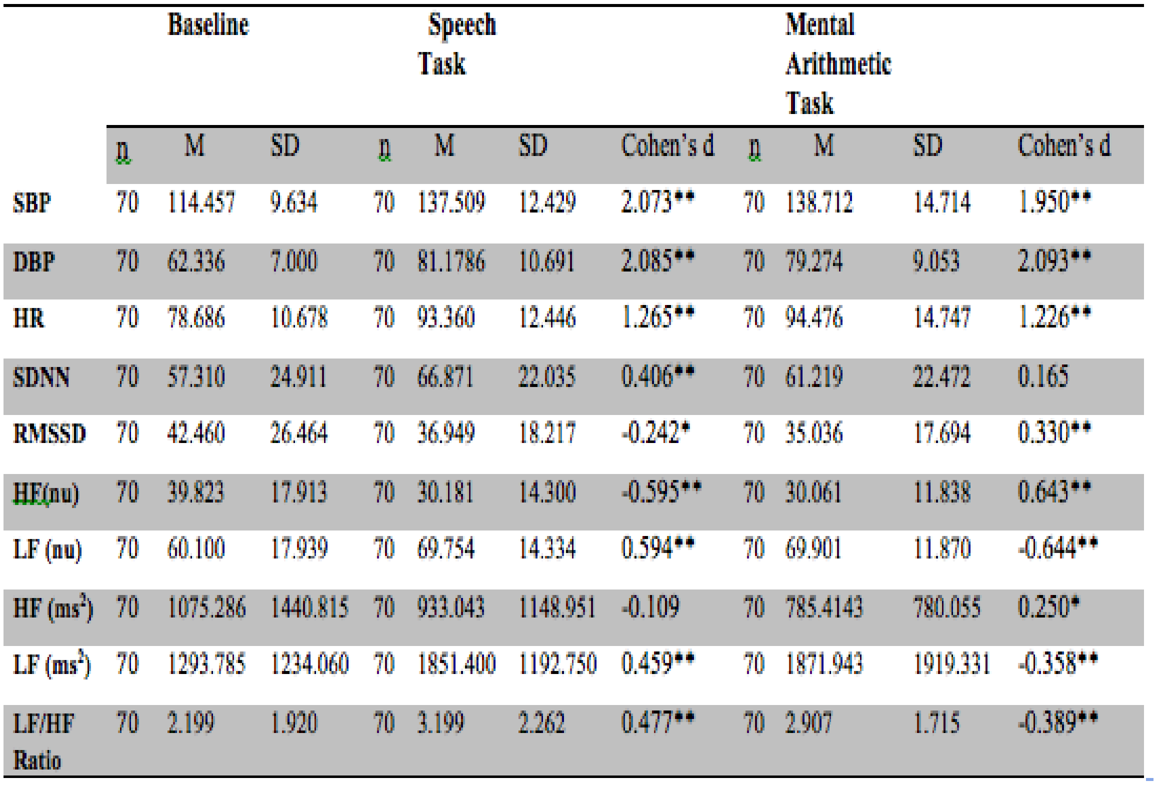
TABLES

Table 1: Descriptive Statistics for Sample Data (non- transformed)

Variable	N	M	SD	SEM
Age	70	19.74	3.674	.439
BMI	70	25.450	5.230	.625
SAM1-threat	70	8.143	2.515	.301
SAM1-challenge	70	11.300	3.033	.362
SAM1-stressfulness	70	10.729	2.859	.342
SAM2-threat	70	7.558	3.086	.369
SAM2-challenge	70	8.857	3.645	.436
SAM2-stressfulness	70	10.127	3.55	.424
JAWS-HPHA	70	13.971	4.011	.479
JAWS-HPLA	70	15.071	3.743	.447
Baseline SBP	70	114.457	9.635	1.151
Baseline DBP	70	62.336	7.000	.837
Baseline HR	70	78.686	10.678	1.276
Baseline SDNN	70	57.310	24.911	2.977
Baseline RMSSD	70	42.460	26.464	3.163
Baseline HF (nu)	70	39.823	17.913	2.141
Baseline LF (nu)	70	60.100	17.939	2.144
Baseline HF (ms^2)	70	1075.286	1440.815	172.210
Baseline LF(ms^2)	70	1293.786	1234.060	147.498
Baseline LF/HF ratio	70	2.199	1.920	.229

Note: BMI = Body Mass Index, SAM = Stress Appraisal Measure, JAWS= Job Affective Well-being Scale, HPHA: High Positive High arousal, HPLA= High positive Low arousal, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HR= Heart Rate, RMSSD = Root Mean Square of Successive Difference, SDNN=Standard Deviation of normal-to-normal intervals, LF = Low frequency spectral power, HF = High frequency spectral power, ms2= milliseconds squared, nu = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers.

Table 2: Manipulation Check for Psychological and Physiological Data (non-transformed)



	Baseline			Speech Task				Mental Arithmetic Task			
	n	M	SD	n	M	SD	Cohen's d	n	M	SD	Cohen's d
SBP	70	114.457	9.634	70	137.509	12.429	2.073**	70	138.712	14.714	1.950**
DBP	70	62.336	7.000	70	81.1786	10.691	2.085**	70	79.274	9.053	2.093**
HR	70	78.686	10.678	70	93.360	12.446	1.265**	70	94.476	14.747	1.226**
SDNN	70	57.310	24.911	70	66.871	22.035	0.406**	70	61.219	22.472	0.165
RMSSD	70	42.460	26.464	70	36.949	18.217	-0.242*	70	35.036	17.694	0.330**
HF(nu)	70	39.823	17.913	70	30.181	14.300	-0.595**	70	30.061	11.838	0.643**
LF (nu)	70	60.100	17.939	70	69.754	14.334	0.594**	70	69.901	11.870	-0.644**
HF (ms^2)	70	1075.286	1440.815	70	933.043	1148.951	-0.109	70	785.4143	780.055	0.250*
LF (ms^2)	70	1293.785	1234.060	70	1851.400	1192.750	0.459**	70	1871.943	1919.331	-0.358**
LF/HF Ratio	70	2.199	1.920	70	3.199	2.262	0.477**	70	2.907	1.715	-0.389**

Note: SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HR= Heart Rate, RMSSD = Root Mean Square of Successive Difference, SDNN=Standard Deviation of normal-to-normal intervals, LF = Low frequency spectral power, HF = High frequency spectral power, ms2 = milliseconds squared, nu = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers.

Table 3: Independent T-Tests Comparing Means between Females and Males on the Stress Appraisal Measure (SAM)

	Males n=44		**Females n=26**		
	M	SD	M	SD	Cohen's d
SAM1- Threat	8.341	2.420	7.808	2.683	0.209
SAM1-Challenge	11.636	3.035	10.731	3.001	0.210
SAM1-Stressfulness	10.500	2.758	11.115	3.037	-0.212
SAM2-Threat	7.528	3.288	7.615	2.772	-0.029
SAM2-Challenge	9.182	3.847	8.308	3.271	0.245
SAM2-Stressfulness	9.704	3.625	10.846	3.367	-0.326

Note: SAM= Stress appraisal measure, SAM1=During speech task, SAM2=during mental arithmetic task. *=p<.05, **=p<.01

Table 4: Independent T-Tests Comparing Means Between Females and Males on Physiological Variables

	Males n=44		**Females n=26**		
	M	SD	M	SD	Cohen's d
Age	19.14	1.153	20.77	5.764	-0.392
BMI	24.323	3.791	27.357	6.691	-0.558*
BL-SBP	116.625	9.525	110.788	8.824	0.636**
BL-DBP	61.659	6.301	63.481	8.049	-0.252
BL-HR	76.451	10.357	82.469	10.320	-0.582*
BL-SDNN	60.848	25.602	51.323	22.935	0.392
BL-RMSSD	43.691	27.783	40.377	24.457	0.127
BL-HF (nu)	37.766	19.113	43.304	15.401	-0.319
BL-LF (nu)	62.198	19.131	56.550	15.422	0.325
BL-HF (ms^2)	1059.114	1333.411	1102.654	1634.087	-0.029
BL-LF (ms^2)	1521.204	1399.648	908.923	767.226	0.542*
BL-LF/HF ratio	2.536	2.231	1.630	1.037	0.521*
S-SBP	140.489	11.679	132.468	12.236	0.671**
S-DBP	81.273	10.746	81.019	10.806	0.023
S-HR	91.774	12.489	96.043	12.139	-0.347
S-SDNN	69.332	23.334	62.708	19.363	0.309
S-RMSSD	38.582	20.009	34.185	14.653	0.251
S-HF (nu)	30.007	15.441	30.477	12.416	-0.033
S-LF (nu)	69.941	15.469	69.438	12.462	0.036
S-HF (ms^2)	1064.636	1368.961	710.346	581.389	0.337
S-LF (ms^2)	1979.909	1193.047	1633.923	1183.251	0.291
S-LF/HF ratio	3.341	2.430	2.958	1.966	0.173
MA-SBP	142.492	15.198	132.314	11.5114	0.755**
MA-DBP	79.186	9.218	79.423	8.944	-0.026
MA-HR	94.493	14.696	94.448	15.124	0.003
MA-SDNN	62.643	24.709	58.808	18.278	0.176
MA-RMSSD	35.398	17.826	34.423	17.802	0.055
MA-HF (nu)	28.784	12.757	32.223	9.957	-0.300
MA-LF (nu)	71.182	12.794	67.735	9.979	0.300
MA-HF (ms^2)	790.364	779.578	777.038	796.234	0.017
MA-LF (ms^2)	2008.045	2192.619	1641.615	1345.574	0.201
MA-LF/HF ratio	3.210	1.964	2.393	1.021	0.522*

Note: BMI=Body Mass Index, BL= Baseline, S= Speech task, MA= Mental Arithmetic task, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HR= Heart Rate, RMSSD = Root Mean Square of Successive Difference, SDNN=Standard Deviation of normal-to-normal intervals, LF = Low frequency spectral power, HF = High frequency spectral power, (ms^{2}2)=milliseconds squared, (nu)=normalized unites, LF/HF=Ratio of Low frequency to High Frequency spectral powers. *=p <.05, **=p<.01.

Table 5: Hierarchical Linear Regressions of JAWS-HPHA, JAWS – HPLA and SAM

Psychological Variable	**SAM Variable**	**R**	R^2	ΔR^2	***B***	***t***	β
JAWS-PA	SAM1-Threat						
	Step1: HPLA, HPHA	.104	.011	--			
	HPLA				.040	.398	.060
	HPHA				-.080	-.843	-.127
	Step2: InterHALA	.125	.016	.005	.013	.569	.534
	SAM1-Challenge						
	Step1: HPLA, HPHA	.141	.020				
	HPLA				-.036	-.295	-.044
	HPHA				.123	1.086	.163
	Step2: InterHALA	.183	.033	.013	-.026	-.960	-.893
	SAM1-Stressfulness						
	Step1: HPLA, HPHA	.154	.024				
	HPLA				-.127	-1.110	-.166
	HPHA				.016	.151	.023
	Step2: InterHALA	.246	.060	.037			
	SAM2-Threat						
	Step1: HPLA, HPHA	.147	.022				
	HPLA				-.149	-1.211	-.181
	HPHA				.070	.606	.091
	Step2: InterHALA	.147	.022	.000	.001	.044	.041
	SAM2-Challenge						
	Step1: HPLA, HPHA	.006	.000				
	HPLA				.008	.052	.008
	HPHA				-.005	-.033	-.005
	Step2: InterHALA	.038	.001	.001	-.010	-.303	-.286
	SAM2-Stressfulness						
	Step1: HPLA, HPHA	.290	.084*				
	HPLA				-.340	-2.474**	-.358
	HPHA				.177	1.377	.199
	Step2: InterHALA	.332	.110	.027	-.043	-1.403	-1.252

Note: JAWS: Job related Affective Well Being Scale, HPHA= High positive High arousal, HPLA= High positive Low arousal, InterHALA= Interaction term for HPHA and HPLA, SAM= Stress appraisal measure, SAM1=during speech task, SAM2=during mental arithmetic task. *=p<.05, **=p<.01.

Table 6: Correlations between JAWS-HPHA, JAWS-HPLA, and SAM during Speech Task

	JAWS-HPHA	**JAWS-HPLA**	**SAM1-Threat**	**SAM1-Challenge**	**SAM1-Stressfulness**
JAWS-HPHA	--	.590**	-.092	.137	-.075
JAWS-HPLA		--	-.015	.052	-.153
SAM1-Threat			--	-.030	.737**
SAM1-Challenge				--	.061
SAM1-Stressfulness					--

Note: JAWS: Job related Affective Well Being Scale, HPHA= High positive High arousal, HPLA= High positive Low arousal, InterHALA= Interaction term for HPHA and HPLA, SAM= Stress appraisal measure, SAM1=During speech task.*=p<.05, **=p<.01

Table 7: Correlations between JAWS-HPHA, JAWS-HPLA, and SAM during Mental Arithmetic Task

	JAWS-HPHA	**JAWS-HPLA**	**SAM2-Threat**	**SAM2-Challenge**	**SAM2-Stressfulness**
JAWS-HPHA	--	.590**	-.016	.000	-.012
JAWS-HPLA		--	-.128	.005	-.241*
SAM2-Threat			--	-.064	.791**.
SAM2-Challenge				--	-.019
SAM2-Stressfulness					--

Note: JAWS: Job related Affective Well Being Scale, HPHA= High positive High arousal, HPLA= High positive Low arousal, InterHALA= Interaction term for HPHA and HPLA, SAM= Stress appraisal measure, SAM2=During mental arithmetic task.*=p<.05, **=p<.01

Table 8: Correlations between JAWS-HPHA, JAWS-HPLA, and Baseline Cardiovascular activity

	HPHA	**HPLA**	**BLSBP**	**BLDBP**	**BLHR**	**BLSDNN**	**BL RMSSD**	**BLHF (nu)**	**BLLF (nu)**	**BLHF (ms^2)**	**BLLF (ms^2)**	**BL RATIO**
HPHA	--	.590**	.043	.114	.163	-.070	.011	.168	-.166	.088	-.101	-.134
HPLA	--	--	.013	.097	-.010	.110	.134	.134	-.133	.137	.117	-.108
BLSBP	--	--	--	.407**	-.084	.015	.083	.158	-.157	.068	-.146	-.141
BLDBP	--	--		--	.334**	-.145	-.044	.138	-.138	.048	-.182	-.096
BLHR	--	--	--	--	--	-.648**	.645**	-.327**	.324**	-.474**	-.354**	.178
BL SDNN	--	--	--	--	--	--	.874**	.334**	-.331**	.803**	.738**	-.096
BL RMSSD	--	--	--	--	--	--	--	.625**	-.622**	.917**	.466**	-.360**
BLHF (nu)	--	--	--	--	--	--	--	--	-1.00**	.670**	-.180	-.811**
BLLF (nu)	--	--	--	--	--	--	--	--	--	-.668**	.182	.811**
BLHF (ms^2)	--	--	--	--	--	--	--	--		--	.359**	-.393**
BLLF (ms^2)	--	--	--	--	--	--	--	--	--	--	--	.314**
BL LF/HF ratio	--	--	--	--	--	--	--	--	--	--	--	--

Note: BL = Baseline, JAWS: Job related Affective Well Being Scale, HPHA= High positive High arousal, HPLA= High positive Low arousal, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HR= Heart Rate, RMSSD = Root Mean Square of Successive Difference, SDNN=Standard Deviation of normal-to-normal intervals, LF=Low Frequency spectral power, HF=High Frequency spectral power, (ms2) = milliseconds squared, (nu) = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers, spectral power. *=p<.05, **=p<.01.

Table 9: Hierarchical Linear Regressions of JAWS-Positive Affect (HPHA and HPLA) and Cardiovascular Reactivity During Speech Task Controlling for Baseline Cardiovascular Activity, Gender, and BMI

Psychological Variable	**Physiological Variable**	**R**	R^2	ΔR^2	***B***	***t***	β
JAWS-PA	Systolic BP						
	Step1:BLSBP,BMI,Gender	.628	.395**	--	--	--	--
	Step2:HPHA, HPLA	.632	.399	.005	--	--	--
	HPHA	--	--	--	.188	.496	.061
	HPLA	--	--	--	-.277	-.685	-.084
	Step3:InterHALA	.632	.400	.000	-.021	-.220	-.173
	Diastolic BP						
	Step1:BLDBP,BMI,Gender	.678	.459**	--	--	--	--
	Step2:HPHA, HPLA	.698	.488	.028	--	--	--
	HPHA	--	--	--	.198	.658	.074
	HPLA	--	--	--	-.582	-1.804	-.204
	Step3:InterHALA	.699	.489	.001	-.029	-.401	-.285
	Heart Rate						
	Step1:BLHR,BMI,Gender	.700	.490**	--	--	--	--
	Step2:HPHA, HPLA	.720	.519	.028	--	--	--
	HPHA	--	--	--	.198	.317	.030
	HPLA	--	--	--	.409	1.119	.123
	Step3:InterHALA	.729	.531	.013	-.106	-1.299	-.882
	SDNN						
	Step1:BLSDNN,BMI,Gender	.633	.400**	--	--	--	--
	Step2:HPHA, HPLA	.648	.420	.020	--	--	--
	HPHA	--	--	--	.164	.245	.030
	HPLA	--	--	--	-.936	-1.309	-.159

Step3:InterHALA	.648	.421	.000	.019	.117	.089
RMSSD						
Step1:BLRMSSD,BMI,Gender	.657	.431**	--	--	--	--
Step2:HPHA, HPLA	.664	.441	.010	--	--	--
HPHA	--	--	--	-.048	-.090	-.011
HPLA	--	--	--	-.465	-.802	-.096
Step3:InterHALA	.666	.443	.002	.059	.452	.335
HF (ms^2)						
Step1:BLHFMS2,BMI,Gender	.460	.212**	--	--	--	--
Step2:HPHA, HPLA	.468	.219	.007	--	--	--
HPHA	--	--	--	-14.299	-.358	-.050
HPLA	--	--	--	-14.504	-.337	-.047
Step3:InterHALA	.468	.219	.000	-.660	-.068	-.059
HF (nu)						
Step1:BLHFNU,BMI,Gender	.439	.193**	--	--	--	--
Step2:HPHA, HPLA	.441	.194	.001	--	--	--
HPHA	--	--	--	-.051	-.101	-.014
HPLA	--	--	--	-.095	-.175	-.025
Step3:InterHALA	.445	.198	.004	-.069	-.559	-.497
LF (ms^2)						
Step1:BLLFMS2,BMI,Gender	.559	.313**	--	--	--	--
Step2:HPHA, HPLA	.579	.336	.023	--	--	--
HPHA	--	--	--	-13.602	-.350	-.046
HPLA	--	--	--	-38.127	-.917	-.120
Step3:InterHALA	.591	.349	.014	10.570	1.146	.917
LF (nu)						
Step1:BLLFNU,BMI,Gender	.441	.195**	--	--	--	--
Step2:HPHA, HPLA	.443	.196	.001	--	--	--
HPHA	--	--	--	.046	.090	.013
HPLA	--	--	--	.096	.176	.025
Step3:InterHALA	.447	.200	.004	.070	.568	.504

LF/HF Ratio						
Step1:BLRATIO,BMI,Gender	.373	.139**	--	--	--	--
Step2:HPHA, HPLA	.390	.152	.014	--	--	--
HPHA	--	--	--	-.062	-.751	-.109
HPLA	--	--	--	.087	.995	.145
Step3:InterHALA	.391	.153	.000	-.003	-.126	-.115

Note: BL = Baseline, JAWS: Job related Affective Well Being Scale, HPHA= High positive High arousal, HPLA= High positive Low arousal, InterHALA= Interaction term for HPHA and HPLA, BMI=Body Mass Index, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HR= Heart Rate, RMSSD = Root Mean Square of Successive Difference, SDNN=Standard Deviation of normal-to-normal intervals, LF = Low frequency spectral power, HF = High frequency spectral power, (ms2) = milliseconds squared, (nu) = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers, spectral power. *=p<.05, **=p<.01.

Table 10: Hierarchical Linear Regressions of JAWS-Positive Affect (HPHA and HPLA) and Cardiovascular Reactivity During Mental Arithmetic Task Controlling for Baseline Cardiovascular Activity, Gender, and BMI

Psychological Variable	**Physiological Variable**	**R**	**R^2**	**ΔR^2**	***B***	***t***	**β**
PA	Systolic BP						
	Step1:BLSBP,BMI,Gender	.623	.388**	--	--	--	--
	Step2:HPHA, HPLA	.629	.395	.007	--	--	--
	HPHA				.263	.585	.072
	HPLA				-.410	-.853	-.104
	Step3:InterHALA	.629	.396	.000	-.020	-.175	-.138
	Diastolic BP						
	Step1:BLDBP,BMI,Gender	.628	.395**				
	Step2:HPHA, HPLA	.674	.455	.060*			
	HPHA				.144	.548	.064
	HPLA				-.679	-2.412	-.281
	Step3:InterHALA	.701	.491	.036*	.131	2.112	1.498
	Heart Rate						
	Step1:BLHR,BMI,Gender	.652	.425**				
	Step2:HPHA, HPLA	.677	.459	.034			
	HPHA				.823	1.895	.224
	HPLA				-.287	-.627	-.073
	Step3:InterHALA	.685	.469	.011	-.115	-1.120	-.809
	SDNN						
	Step1:BLSDNN,BMI,Gender	.739	.546**				
	Step2:HPHA, HPLA	.755	.570	.024			
	HPHA				-1.110	-1.893	-.198
	HPLA				.614	.978	.102
	Step3:InterHALA	.755	.570	.000	-.008	-.055	-.036
	RMSSD						
	Step1:BLRMSSD,BMI,Gender	.743	.552**				

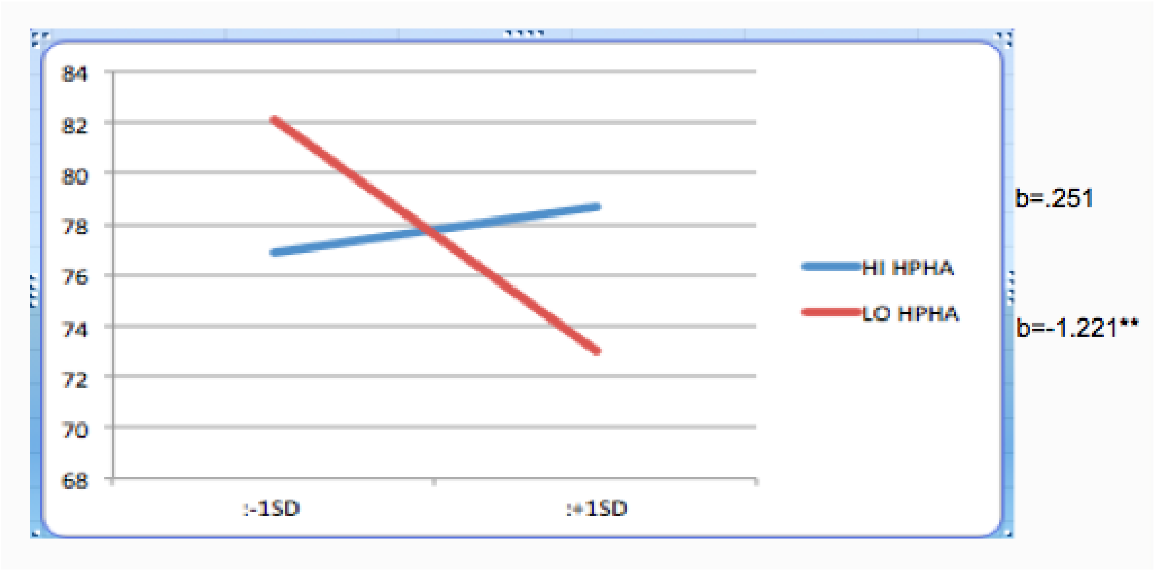
Step2:HPHA, HPLA	.757	.572	.020			
HPHA				-.662	-1.447	-.150
HPLA				.056	.115	.012
Step3:InterHALA	.757	.572	.000	.008	.071	.046
HF (ms^2)						
Step1:BLHFMS2,BMI,Gender	.558	.312**				
Step2:HPHA, HPLA	.587	.344	.033			
HPHA				-37.200	-1.497	-.191
HPLA				3.074	.115	.015
Step3:InterHALA	.591	.349	.004	3.951	.655	.524
HF (nu)						
Step1:BLHFNU,BMI,Gender	.490	.240**				
Step2:HPHA, HPLA	.525	.276	.036			
HPHA				.402	1.012	.136
HPLA				-.756	-1.780	-.239
Step3:InterHALA	.525	.276	.000	-.016	-.164	-.139
LF (ms^2)						
Step1:BLLFMS2,BMI,Gender	.573	.328**				
Step2:HPHA, HPLA	.633	.401	.073*			
HPHA				-146.655	-2.471	-.306
HPLA				161.449	2.541	.315
Step3:InterHALA	.634	.402	.001	-4.899	-.344	-.264
LF (nu)						
Step1:BLLFNU,BMI,Gender	.488	.238**				
Step2:HPHA, HPLA	.524	.274	.036			
HPHA				-.407	-1.020	-.137
HPLA				.758	1.776	.239
Step3:InterHALA	.524	.275	.000	.017	.174	.147
LF/HF Ratio						
Step1:BLRATIO,BMI,Gender	.435	.189**				
Step2:HPHA, HPLA	.512	.262	.073*			
HPHA				-.110	-1.904	-.258
HPLA				.152	2.449	.332

Step3:InterHALA	.523	.274	.012	-.014	-1.026	-.867

Note: BL = Baseline, JAWS: Job related Affective Well Being Scale, HPHA= High positive High arousal, HPLA= High positive Low arousal, InterHALA= Interaction term for HPHA and HPLA, BMI=Body Mass Index, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HR= Heart Rate, RMSSD = Root Mean Square of Successive Difference, SDNN=Standard Deviation of normal-to-normal intervals, LF = Low frequency spectral power, HF = High frequency spectral power, (ms2) = milliseconds squared, (nu) = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers, spectral power. *=p<.05, **=p<.01.

Figures

Figure 1: High arousal work-related positive affect, Low-arousal work-related positive affect, and diastolic blood pressure. This figure illustrates the significant interaction between high-arousal work-related positive affect and low –arousal work-related positive affect with diastolic blood pressure. HIHPHA= High high-arousal positive affect, LOHPHA= Low high-arousal positive affect.



.

APPENDIX A: DEMOGRAPHICS AND SCREENING QUESTIONNAIRE

Age: ___________ ID#: ___________

INITIAL QUESTIONS

1- Have you had any alcoholic or caffeinated products in the past 12 hours?
______YES ______NO
2- Have you had any tobacco products in the past 3 hours?
______YES ______NO

EMPLOYMENT STATUS

1-Do you currently have a paid employment? ______YES ______NO

______Full time (40hrs/week) _______ Part time (20hrs/week) ______Contingent

2-What category does your current job fall under? Check those that apply

_____Administrative/Clerical _____Customer Service

_____Health Care _____Research ______Other:_______

1- Gender
MALE _________ FEMALE____________ OTHER:___________
2- Religion
______Christianity
______Islam
______Buddhism
______Hinduism
______ Judaism
______Atheism
______Other: _____________

4- Are you currently taking any of the following types of medications? Put a check mark next to those that apply

______Oral contraceptives

______Hormone replacement therapy

______Anti-inflammatory medications (pain killers; NSAIDs, Motrin/ibuprofen)

______Steroids (topical eczema/psoriasis medications, prednisone)?

______Blood-pressure medications

______Mental health-related medications (i.e. any SSRIS, anti depressants, anti-anxiety medications, mood stabilizers, others etc..)

5-Have you ever been told that you have high blood pressure?

______YES ________ NO

6-To the best of your knowledge, do you have family history of stroke or heart attack prior to age 50?

________YES________ NO

7-Do you have an implanted medical device (e.g. pacemaker)?

________YES________ NO

8- To the best of your knowledge, do you have, have had, or show symptoms of the following medical conditions? Put a check mark next to those that apply

_____Heart attack

_____ Stroke

_____Cardiovascular problems

_____ Coronary heart disease

_____High blood pressure

_____ Heart arrhythmia

_____ Chest pain

_____Chronic kidney disease

_____Chronic liver disease

_____Diabetes

_____Current diagnosis of cancer

_____Autoimmune disease (i.e. arthritis)

_____Any current severe allergies

_____ Chronic infections

_____Hormone disorders (i.e. inflammation of the thyroid gland, Addison's disease, Conn's disease)

_____Current diagnosis of psychological disorder (i.e. clinical depression, Cushing's disease, PTDS)

9- Are you currently pregnant or breastfeeding?

______YES ______NO

10- Do you smoke?

______YES ______NO

11- If you smoke cigarettes, on average, how many cigarettes do you smoke per day? __________

12- Are you able to stay seated for 10 minutes without moving?

______YES ______NO

<u>For researchers</u>

HEIGHT: _________

WEIGHT:_________

APPENDIX B: THE POSITIVE AND NEGATIVE AFFECT SCHEDULE

PANAS

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you have felt this way during the past month. Use the following scale to record your answers.

1=	2=	3=	4=	5=
very slightly or not at all	a little	moderately	quite a bit	extremely

________ interested

________ distressed

________ excited

________ upset

________ strong

________ guilty

________ scared

________ hostile

________ enthusiastic

________ proud

________ irritable

________ alert

________ ashamed

________ inspired

________ nervous

________ determined

________ attentive

________ jittery

________ active

________ afraid

APPENDIX C: STRESS APPRAISAL MEASURE

SAM-1 (SPEECH TASK)

This questionnaire is concerned with your thoughts about the speech task that you are about to engage in. There are no right or wrong answers. Please respond according to how you view this situation right NOW. Please answer ALL questions.

Answer by CIRCLING the appropriate number corresponding to the following scale.

		Not At All	Slightly	Moderately	Considerably	Extremely
1.	Does this situation create tension in me?	1	2	3	4	5
2.	Does this situation make me feel anxious?	1	2	3	4	5
3.	Is this going to have a positive impact on me?	1	2	3	4	5
4.	How eager am I to tackle this problem?	1	2	3	4	5
5.	To what extent can I become a stronger person because of this problem?	1	2	3	4	5
6.	Will the outcome of this situation be negative?	1	2	3	4	5
7.	Does this situation tax or exceed my coping resources?	1	2	3	4	5
8.	To what extent am I excited thinking about the outcome of this situation?	1	2	3	4	5
9.	How threatening is this situation?	1	2	3	4	5
10.	To what extent do I perceive this situation as stressful?	1	2	3	4	5
11.	To what extent does this event require coping efforts on my part?	1	2	3	4	5
12.	Is this going to have a negative impact on me?	1	2	3	4	5

APPENDIX D: STRESS APPRAISAL MEASURE

SAM-2 (MENTAL ARITHMETIC TASK)

This questionnaire is concerned with your thoughts about the math task that you are about to engage in. There are no right or wrong answers. Please respond according to how you view this situation right NOW. Please answer ALL questions.

Answer by CIRCLING the appropriate number corresponding to the following scale.

		Not At All	Slightly	Moderately	Considerably	Extremely
1.	Does this situation create tension in me?	1	2	3	4	5
2.	Does this situation make me feel anxious?	1	2	3	4	5
3.	Is this going to have a positive impact on me?	1	2	3	4	5
4.	How eager am I to tackle this problem?	1	2	3	4	5
5.	To what extent can I become a stronger person because of this problem?	1	2	3	4	5
6.	Will the outcome of this situation be negative?	1	2	3	4	5
7.	Does this situation tax or exceed my coping resources?	1	2	3	4	5
8.	To what extent am I excited thinking about the outcome of this situation?	1	2	3	4	5
9.	How threatening is this situation?	1	2	3	4	5
10.	To what extent do I perceive this situation as stressful?	1	2	3	4	5
11.	To what extent does this event require coping efforts on my part?	1	2	3	4	5
12.	Is this going to have a negative impact on me?	1	2	3	4	5

APPENDIX E: JOB-RELATED AFFECTIVE WELL-BEING SCALE

JAWS

Below are a number of statements that describe different emotions that a job can make a person feel. Please indicate the amount to which <u>any part of your job (e.g., the work, coworkers, supervisor, clients, pay) has made you feel</u> that emotion in the past <u>30</u> days.

Please check **one** response for each item that best indicates how often you've experienced each emotion at work over the past 30 days.	Never	Rarely	Sometimes	Quite often	Extremely often
1. My job made me feel at ease					
2. My job made me feel angry					
3. My job made me feel annoyed					
4. My job made me feel anxious					
5. My job made me feel bored					
6. My job made me feel cheerful					
7. My job made me feel calm					
8. My job made me feel confused					
9. My job made me feel content					
10. My job made me feel depressed					
11. My job made me feel disgusted					
12. My job made me feel discouraged					
13. My job made me feel elated					
14. My job made me feel energetic					
15. My job made me feel excited					
16. My job made me feel ecstatic					
17. My job made me feel enthusiastic					
18. My job made me feel frightened					
19. My job made me feel frustrated					
20. My job made me feel furious					
21. My job made me feel gloomy					
22. My job made me feel fatigued					
23. My job made me feel happy					
24. My job made me feel intimidated					
25. My job made me feel inspired					
26. My job made me feel miserable					
27. My job made me feel pleased					
28. My job made me feel proud					
29. My job made me feel satisfied					
30. My job made me feel relaxed					

APPENDIX F: INFROMED CONSENT

(STUDENTS ENROLLED IN INTRODUCTORY PSYCHOLOGY COURSES)

EXPERIMENTAL SUBJECT POOL PARTICIPATION CONSENT FORM

Purpose of the Study:

The psychology faculty considers participation in experimental research by subjects to be an educational experience for the students as well as a most important service to the research of the University. This research project has been approved by the University of Michigan-Dearborn Institutional Review Board (IRB Dearborn). Participation is voluntary, if you choose **not** to participate as a research subject you may participate in another research related activity at no expense to your academic record or standing, or complete an alternate assignment instead of participating in a study. The purpose of today's experiment is to learn more about the relationship between work-related positive emotions and cardiovascular responses.

As a part of your participation in an Introductory Psychology course at the University of Michigan- Dearborn, you agree to serve as a research subject for this experiment. You have had the opportunity to read the "Subject Pool Participation" description information that was provided when you registered on the SONA System website as a research participant. You may withdraw at any time from today's study without penalty or loss of research participation credit.

Description of Subject Involvement:

If you agree to be part of the research study, you will be asked to complete the study's protocol, which will last approximately 70 minutes. The study's procedure involves completing several questionnaires assessing emotional and cognitive functioning. Height and weight measurements will be collected and blood pressure and heart functioning will be assessed. Cardiovascular functioning will be assessed using a non-invasive electrocardiogram via the placement of three electrodes on extremities (the electrodes will be placed on the inside wrists of both hands as well as the inside of the left ankle). A non-invasive blood pressure cuff will be placed on the upper non-dominant arm and will be used to monitor blood pressure. Finally, you will be asked to discuss with the researchers your career aspirations and solve some math problems.

We plan to publish or present the results of this study, but will not include any information that would identify you. There are some reasons why people other than the researchers may need to see information you provided as part of the study. This includes organizations responsible for making sure the research is done safely and properly, including the University of Michigan, government offices.

Benefits:

Although there are no direct benefits to this study, data from this study can be used for the further advancement of the role of emotions and cardiovascular functioning.

Risks/Discomforts:

The risks include slight discomfort due to the periodic inflation of the blood pressure cuff as well as upon the removal of the electrodes. Some participants may feel angry or nervous after talking about their career goals or completing math problems.

Compensation:

You will receive one and a half subject pool credits for your participation in today's study or one-half subject pool credit if you only complete the PANAS and demographics questionnaire.

Confidentiality:

The results of this study may be published. No information that would identify you will be included. Your privacy will be protected, and the research records will be confidential. It is possible that other people may need to see the information you provide as part of the study. Such organizations are responsible for making sure the research is done safely and properly, like the U-M Dearborn IRB.

Storage and future use of data:

The data you provide will be stored on a password-protected computer that only Farah Elsiss, the principal investigator, has access to. The data may be shared with other researchers but will never contain information that could identify you. If you are deemed ineligible, data obtained from the demographic and screening questionnaires will not be included in the study but will be retained for the length of the appropriate retention period and then safely destroyed. If deemed eligible, but choose to prematurely withdraw from the study, the data obtained will be saved for analysis in the same way that the other participants' data will be handled unless you explicitly state that you wish to withdraw your data from being included in the analysis. Retained data will be saved for the length of appropriate retention period which is three years after the study has ended and then safely destroyed.

Voluntary nature of the study:

Participating in this study is completely voluntary. Even if you decide to participate now, you may change your mind and stop at any time. If you decide to prematurely withdraw from the study, there is no penalty. . If you are eligible, your data will be retained and used in the analysis unless you explicitly choose to withdraw your data from being included in the analysis. If you are not eligible, your data will not be used in the analysis, but will be saved with the other data, and destroyed with the others at the designated time.

Contact Information:

If you have questions about the study you may contact Farah Elsiss (felsiss@umich.edu) or their faculty advisor Dr. Pecina (pesu@umich.edu).

If you have questions regarding your rights as a research participant, or wish to obtain information, ask questions, or discuss concerns with someone other than the researcher(s), you may contact the Dearborn IRB Administrator in the Office of Research and Sponsored Programs, 2066 IAVS, University of Michigan- Dearborn, Evergreen Rd., Dearborn, MI 48128-2406, (313) 593-5468; the Dearborn IRB Application specialist at (734) 763-5084, or email Dearborn-IRB@umich.edu.

Your participation will require approximately 70 minutes. The purpose and procedure as well as the benefits and risks of the study have been explained to you and the results will be made available to you upon your request. By signing this document, you are agreeing to be in the study. You will be given a copy of this document for your records and one copy will be kept with the study records. Be sure that questions you have about the study have been answered and that you understand what you are being asked to do. You may contact the researcher if you think of a question later.

I agree to participate in the study.

Signature____________________________
Name: ______________________________
Address: _____________________________
Enrolled in: Psychology _______________
Psychology Instructor__________________

To be filled by experimenter:

Experiment: ________________________

Date: _______________________________

Experimenter: ______________________

APPENDIX G: INFORMED CONSENT

(STUDENTS ENROLLED IN UPPER LEVEL PSYCHOLOGY COURSES)

EXPERIMENTAL SUBJECT POOL PARTICIPATION CONSENT FORM

The psychology faculty considers participation in experimental research by subjects to be an educational experience for the students as well as a most important service to the research of the University. This research project has been approved by the University of Michigan-Dearborn Institutional Review Board (IRB Dearborn). Participation is voluntary, if you choose **not** to participate as a research subject you may participate in another research related activity at no expense to your academic record or standing. The purpose of today's experiment is to learn more about emotions in relation to everyday stressors.

Upper Level Psychology Course Research Subjects

As part of your participation in an upper level psychology course at the University of Michigan- Dearborn you agree to serve as a research subject for this experiment. You have had the opportunity to read the "Subject Pool Participation" description information that was provided when you registered on the SONA System website as a research participant. Your professor will be notified of your participation so that you can earn the extra credit specified by the professor. You may withdraw at any time from today's study without penalty or loss of extra credit.

Description of Subject Involvement:

If you agree to be part of the research study, you will be asked to complete the study's protocol, which will last approximately 70 minutes. The study's procedure involves completing several questionnaires assessing emotional and cognitive functioning. Height and weight measurements will be collected and blood pressure and heart functioning will be assessed. Cardiovascular functioning will be assessed using a non-invasive electrocardiogram via the placement of three electrodes on extremities (the electrodes will be placed on the inside wrists of both hands as well as the inside of the left ankle). A non-invasive blood pressure cuff will be placed on the upper non-dominant arm and will be used to monitor blood pressure. Finally, you will be asked to discuss with the researchers your career aspirations and solve some math problems.

We plan to publish or present the results of this study, but will not include any information that would identify you. There are some reasons why people other than the researchers may need to see information you provided as part of the study. This includes organizations responsible for making sure the research is done safely and properly, including the University of Michigan, government offices.

Benefits:

Although there are no direct benefits to this study, data from this study can be used for the further advancement of the role of emotions and cardiovascular functioning.

Risks/Discomforts:

The risks include slight discomfort due to the periodic inflation of the blood pressure cuff as well as upon the removal of the electrodes but all measures have been taken to mitigate those risks. Some participants may feel angry or nervous after talking about their career goals or completing math problems.

Compensation:

Once the consent form is signed, your professor will be notified of your participation so that you earn the extra credit specified by them. You may stop at any time during the study without loss of extra credit.

Confidentiality:

The results of this study may be published. No information that would identify you will be included. Your privacy will be protected, and the research records will be confidential. It is possible that other people may need to see the information you provide as part of the study. Such organizations are responsible for making sure the research is done safely and properly, like the U-M Dearborn IRB.

Storage and future use of data:

The data you provide will be stored on a password-protected computer that only Farah Elsiss, the principal investigator, has access to. The data may be shared with other researchers but will never contain information that could identify you. If you are deemed ineligible, data obtained from the demographic and screening questionnaires will not be included in the study but will be retained for the length of the appropriate retention period and then safely destroyed. If deemed eligible, but choose to prematurely withdraw from the study, the data obtained will be saved for analysis in the same way that the other participants' data will be handled unless you explicitly state that you wish to withdraw your data from being included in the analysis. Retained data will be saved for the length of appropriate retention period which is three years after the study has ended and then safely destroyed.

Voluntary nature of the study:

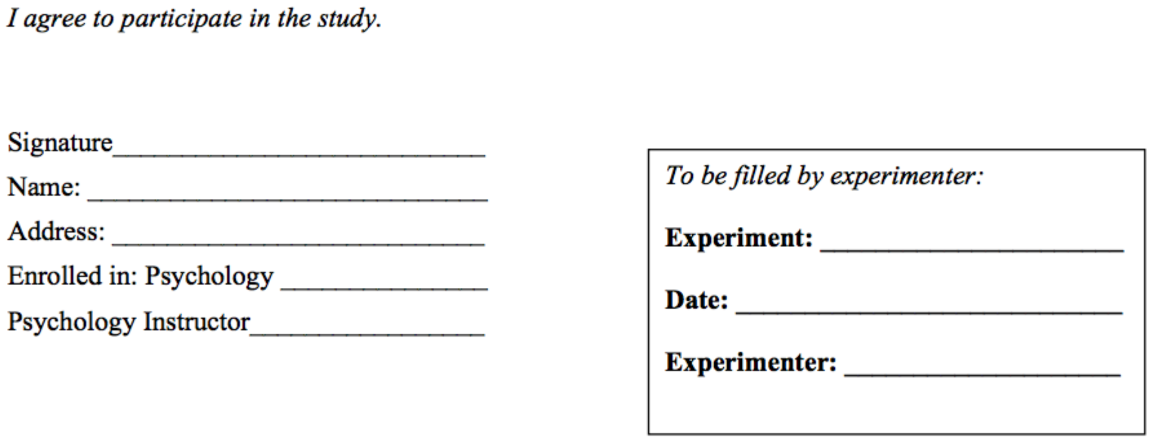
Participating in this study is completely voluntary. Even if you decide to participate now, you may change your mind and stop at any time. If you decide to prematurely withdraw from the study, there is no penalty. If you are eligible, your data will be retained and used in the analysis unless you explicitly choose to withdraw your data from being included in the analysis. If you are not eligible, your data will not be used in the analysis, but will be saved with the other data, and destroyed with the others at the designated time.

Contact Information:

If you have questions about the study you may contact Farah Elsiss (felsiss@umich.edu) or their faculty advisor Dr. Pecina (pesu@umich.edu).

If you have questions regarding your rights as a research participant, or wish to obtain information, ask questions, or discuss concerns with someone other than the researcher(s), you may contact the Dearborn IRB Administrator in the Office of Research and Sponsored Programs, 2066 IAVS, University of Michigan- Dearborn, Evergreen Rd., Dearborn, MI 48128-2406, (313) 593-5468; the Dearborn IRB Application specialist at (734) 763-5084, or email Dearborn-IRB@umich.edu.

Your participation will require approximately 70 minutes. The purpose and procedure as well as the benefits and risks of the study have been explained to you and the results will be made available to you upon your request. By signing this document, you are agreeing to be in the study. You will be given a copy of this document for your records and one copy will be kept with the study records. Be sure that questions you have about the study have been answered and that you understand what you are being asked to do. You may contact the researcher if you think of a question later.



I agree to participate in the study.

Signature___________________________
Name: ______________________________
Address: ____________________________
Enrolled in: Psychology _______________
Psychology Instructor_________________

To be filled by experimenter:

Experiment: ______________________

Date: ____________________________

Experimenter: ____________________

APPENDIX H: DEBRIEFING FORM

DEBRIEFING FORM

Thank you for your interest and participation in this study. We hope that through the better understanding the dynamics of different emotions we can make a distinctive link to subsequent health issues.

This sheet is provided to inform you that no video recording of your participation took place today and that no performance analysis will be conducted at a later time. The deception was important to the study protocol because research shows that this form of deception increases evaluative/performance stress and consequent cardiovascular responses. We ask that you do not share the protocol with anyone else to as to not jeopardize the effectiveness of the stress tasks. You may notify the researcher to have your data withdrawn from being included in the analysis if you prefer to do so.

If you have questions about the study or interested in findings once the study is completed you may contact Farah Elsiss (felsiss@umich.edu) or the faculty advisor Dr. Pecina (pesu@umich.edu).

If you have questions regarding your rights as a research participant, or wish to obtain information, ask questions, or discuss concerns with someone other than the researcher(s), you may contact the Dearborn IRB Administrator in the Office of Research and Sponsored Programs, 2066 IAVS, University of Michigan- Dearborn, Evergreen Rd., Dearborn, MI 48128-2406, (313) 593-5468; the Dearborn IRB Application specialist at (734) 763-5084, or email Dearborn-IRB@umich.edu.

A list of agencies listed below can be reached should you desire to seek additional help:

☐ UM-D Counseling and Support Services (UM-D students only)

o 313-593-5430

☐ Henry Ford Medical Center- Fairlane for Students, Faculty and Staff (UM-D students only)

o 313-982-8495

Thank you again for your participation.