A RANDOMIZED CONTROL TRIAL OF A HIGH INTENSITY INTERVAL TRAINING PROGRAM ON PSYCHOLOGICAL OUTCOMES

By

RICHARD LEWIS YOUNG

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To the Faculty of Washington State University:

The members of the Committee appointed to examine the dissertation or thesis of RICHARD LEWIS YOUNG find it satisfactory and recommend that it be accepted.

Janet Beary, Ph.D., Chair

Lindsey E. Miller, Ph.D.

Sterling McPherson, Ph.D.

Dennis Dyck, Ph.D.
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The aim of this study was to determine if contrasting forms of exercise intensities promote differing psychological outcomes that bolster tolerance and recovery from acute distressing events through cross-stressor adaptation. Repeated successions of physiologic stress that accompanies exercise may result in neurobiological adaptations that lead to a reduction in sensitivity to subsequent similar stimuli. These adaptations may also lead to the reduction in differing stimuli, such as a distressful event. Therefore, the intent of the study was to determine if high intensity interval training compared to moderate intensity steady-state training lead to contrasting psychological and physiological outcomes when exposed to acute distress. Twenty-five participants between 18-55 years of age were randomized into high intensity interval training or moderate intensity continuous training for 6 weeks with 3 exercise sessions per week. Before exercise intervention, participants were subjected to the Trier Social Stress Test (TSST) while Blood Pressure (BP) and Heart Rate (HR) were monitored and salivary cortisol (SA) samples taken before and after the TSST. Additionally, Perceived Stress Scale -10 (PSS-10), and Strait Trait Anxiety Inventory (STAI) were completed by each participant. The same
procedures were conducted post intervention to determine approximate change. There was no significant difference between group comparisons for PSS-10, STAI, BP, or SA. However when comparing measures across time for both groups together as an independent variable, there was significant difference in the aforementioned excluding only BP. Additionally there was significant difference in HIIT HR during TSST and a more robust SA response to stress compared to MICT across 4 measure collecting periods (29.3% ± .026) suggesting that exercise supports cross stressor adaptation to heterolytic stress and that HIIT is as efficacious as MICT showing trends towards significant differences. There can be many pre-existing provisions that contribute to an individual’s ability to tolerate distressing pressure that include personality, a challenge mindset, and a facilitative environment. Exercise may additionally help facilitate this recipe for stress resilience. Data from this study may directly impact the current methods prescribed for stress tolerance and recovery, and can lead to additional queries investigating the mechanisms that drive stress perception.
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Dedication

To my tenacity and courage
CHAPTER ONE: INTRODUCTION

The physiological and psychological health benefits associated with regular exercise have long been established and are well documented (Penedo and Dahn, 2005). However, there exists no current standard exercise prescription that health care providers can offer patients who present with stress related complaints leaving practitioners open to suggest a variety of exercise options based on a variety of sources. The Anxiety and Depression Association of America (ADAA), (2017) recommends complying with the 2008 Federal physical activity guidelines that call for 30-60 minutes of moderate intensity exercise (5 days/week) or 20-60 minutes of vigorous-intensity exercise (3 days /week), (ACSM, 2017); However, the American Psychological Association (APA), (2011) admits that there is limited data to suggest which type of exercise is most efficacious. Regardless of the ambiguity, aerobic-type Moderate-Intensity Continuous Training (MICT) has been the subject for the majority of research investigating correlations between exercise and stress and/or anxiety, and is also recommended by the aforementioned ADAA, APA, as well as the National Institutes of Health (NIH). In contrast, High Intensity Interval Training (HIIT) precipitates similar stress-buffering benefits, but with superior performance and health driven anthropometric outcomes when compared to an exercise model of MICT commonly recommended to help manage the undesirable effects of mental stress. In studies focused on athletic performance outcomes, HIIT has been shown to reduce the same physiological stress measures that are consistent with those also used in studies investigating the impact of psychological and psychosocial stress, to include blood pressure and heart rate variability (Munk et al, 2010; Di Blasio, 2014; Fisher et al, 2015; Shepard et al, 2015). On its own merit, exercise in any form seems to
solicit a stress-buffering capacity by creating neurobiological adaptations to the body’s response to stress, which increases tolerance to subsequent exposures to stress through a process known as cross-stressor adaptation (Michael, 1957). The key perspective as it relates to stress and anxiety within the adaptation model is that although the body’s physiological response to a perceived stress can be adapted by exercise to reduce sensitivity to subsequent similar stressors (i.e., exercise), it also manifests the capacity to reduce sensitivity to heterolytic stressors (i.e., psychosocial discomfort) (Klaperski et al, 2014). More importantly to note, the amount of exercise needed to solicit a reduction in the perception of stress is predicated on total energy expenditure and low amounts or low intensities of exercise are not effective in mitigating stress symptomology (Dunn et al., 2005). Since the magnitude of resilience is dependent on the dose of intensity of the exercise, HIIT offers a modality that may yield similar reduction in stress sensitivity and stress-buffering capacity relative to the commonly prescribed MICT exercise regimen, but with superior performance and anthropometric outcomes, and with significant less time investment. This makes HIIT an attractive alternative for individuals with busy schedules, restricted access to fitness facilities, and limited time, but who are encumbered by stress and/or commonly experience the symptoms of anxiety.

**Stress:**

In simple terms, stress is a constellation of events that begin with a stressor (stimulus), which precipitates a reaction in the brain (stress perception), that results in the activation of the fight or flight systems in the body (physiological stress response) (Dhabhar F, 2008). The source of stimuli falls in four main categories, which are classified as
circadian disruption, glycemic dysregulation, inflammatory signaling, and perception. In modern society, there are increasingly tremendous opportunities to experience stress in all four of these categories.

The mere nature of our 21st Century life-styles with employment types (Patterson L., 1983), social pressures (Festinger et al., 1950), and diets (Kamara et al., 1998) provides fertile soil and an opportune environment for the activation of the stress response, but without the life-threatening dangers the response was intended to help overcome. Worse, the levels of this stress amongst Americans are reported to be precariously higher than those compared to just 10 years ago with an un-arrested forecast for continued development (American Psychological Association, 2014). For most, this observed trend may not be a novel concept, however, the health implications that result from this phenomenon may not be as obvious.

Hans Selye (1976) was first to theorize that the body’s physiological stress response could be deleterious to physical health and pioneered the notion that a correlation existed between sustained stress and disease. Many studies have supported Selye’s theory; for example, research has implicated stress to be related to and or a contributor to weight gain via central and visceral adiposity (Shively et al, 2009), metabolic syndrome and diabetes (Siddiqui et al, 2015), immunosuppression (Randall, 2011), gastrointestinal disorders (Butorina, 2013), hypertension (Kulkarni et al, 1998), atherosclerosis and related cardiovascular diseases and events (Shively et al, 2009; Brudey et al, 2015), insomnia (Vgontzas et al, 1998), depression and anxiety (Mackin and Young, 2004), arthritis (Bradley et al, 1992), asthma (Bienenstock, 2002), short-term memory loss and dementia (Sheline et al, 1999; Vasterling et al, 2002; Conrad C, 2008) and infertility
Curiously, even though the concept of stress is moderately commonplace and well accepted by most, it is somewhat difficult to define for scientists and researchers in part because it is a highly subjective and personal experience that differs for each individual (Hackney, 2006). Not only can the manifestation of stress differ for a similar stimulus, but the subsequent response may vary greatly from person to person as well. For example, one individual might find the experience of flying a serene experience eliciting relaxation and pleasure while another might find the experience distressing evoking panic and fear; however, even though the perception of stress is highly individualized, most professionals and researchers agree that there are determinants of stress that would induce a physiological stress response in any person that is exposed to them.

These determinants make up the recipe for stress, but each ingredient is able to act independently in order to solicit a stress response within an individual. Typically, for an individual to perceive an event as being stress-inducing, the event must be interpreted as novel, contain unpredictability, be considered a threat to the ego, and/or elicit a feeling of powerlessness, helplessness, or uncontrollability (Lupien S. 2012; Centre for studies on human stress, 2015). Variants of stress are additionally considered to be either negative or positive (distress and eustress respectively) (Selye H, 1974), and peculiarly, the physiological responses that occur in the body as a consequence to distress are normally identical to the physiological responses in the body that occur due to eustress (Le Fevre et al, 2003). Interestingly however, even though the body may react to these differing variants with a similar physiological response, the health outcomes arising from the two
are dramatically different where negative health outcomes are observed from distress and positive health outcomes are observed from eustress (Dhabhar F, 2008).

The severity of the negative health outcomes that are correlated with distress are dependent on the frequency and duration of the distressing stimuli (McEwen, 2004; Bleier, 2011) therefore, in order to stratify exposure, the American Psychological Association categorizes stress as acute, episodically acute, and chronic. During periods of “acute stress” the body responds to the provocation with neuroendocrine and sympathetic stimulation, known as the fight or flight response, which produces the appropriate hormones needed to either challenge or retreat from the presented threat. Because of the short-term nature of acute stress, the physiological response returns to homeostasis quickly with little to no ill effects.

When acute stress is encountered frequently, individuals experience “episodic acute stress” and some often mold their personalities around this form of stress accordingly. These people are commonly referred to as Type-A personalities (ADA, 2017) and will tend to exhibit aggression, impatience, and an aggressive form of competitiveness (Friedman and Rosenman, 1959). Additionally, individuals who ruminate and worry will also experience episodically acute stress, but unlike the aforementioned aggressiveness of Type-As, these catastrophic-thinking individuals experience anxiety and depression from this form of stress. While under influences of episodic acute stress, the sympathetic nervous system attempts to restore physiological functions to normal levels by mobilizing resources to resolve the problem; however, if stress continues, the body enters a resistance phase where adaptations to the stress begin to occur (Selye, 1956). Beyond the persistent tension headaches and migraines, cardiovascular implications such as hypertension, chest pain, and heart disease can manifest during this juncture (McGonagle and Kessler, 1990).
Lifestyle and personality issues are habitual and are often ingrained in individuals; therefore it is important to note that some that suffer with episodic acute stress commonly believe that nothing is wrong with the way they conduct their lives and tend to believe situations and/or environments are at fault, (APA, 2017).

When stress becomes habitual and exists in perpetuity with unrelenting demands and pressures for seemingly interminable periods of time, individuals’ experience “chronic stress”. Chronic stress manifests a view of the world, or a belief system, that causes unending stress for the individual so that hope is lost or a situation is no longer navigable (Miller, 2007). When this occurs the ability to resist stress disappears, adaption fails, and the level of physiological function decreases, increasing risk of disease (Selye, 1956). A non-intuitive aspect of this form of stress is that individuals may forget that it is present by becoming accustomed to the chronicity of the exposure where it may take on a familiar, and sometimes, almost comfortable frame of reference and perspective to life (Dallman et al, 2003).

Though the categorization of stress is related to it’s chronicity, it is still a highly subjective phenomenon and is typically different for each individual. Certain individuals’ posses a resilience capacity that promotes mental fortitude against stressors that ultimately leads to sustained chronic exposure. Individual psychological factors, a challenge mindset, and a facilitative environment all support stress resiliency (Fletcher and Sarkar, 2016), but regardless of vigor and efficacy of mental fortitude, exposure to stressful stimuli in perpetuity will eventually elicit the body’s physiological response (Selye, 1946)
**Hypothalamus-pituitary-adrenal and Sympatho-Adreno-Medullary Axis:**

The physiological stress response is activated through the sympathetic branch of the Autonomic Nervous System (ANS) and is governed by the hypothalamic-pituitary-adrenal (HPA) and Sympatho-AdrenoMedullary (SAM) axis, which are controlled by the hypothalamus. In addition, the stress response also activates the norepinephrine system, which includes the aforementioned Sympathetic Nervous System (SNS) and the locus coeruleus-centered system in the brain (Wilmore and Cstill, 2004; Russell et al, 2008).

The stress response is assessed in terms of (1) stress reactivity – the peak of a stress response when exposed to stressful stimuli, and (2) stress recovery – the degree in which the elevation of physiological parameters persist after stressful stimuli cessation (Linden et al., 1997).

When an individual is introduced to a stressful stimulus, the hypothalamus responds within seconds by secreting corticotrophin-releasing hormone (CRH) in concert with vasopressin (AVP). The secretion of these hormones activates the secretion of adrenocorticotrophic hormone (ACTH) from the pituitary, which then travels in the blood to the adrenal glands and stimulates the secretion of glucocorticoids (cortisol) from the adrenal cortex. Simultaneously nerve impulses travel through the sympathetic branch to the adrenal medulla to secrete the catecholamines norepinephrine and epinephrine (Russell et al, 2008).

Cortisol’s primary role is to restore homeostasis from the HPA axis’ stress response following exposure to stressful stimuli. However, in response to this stimulus, cortisol also performs other important functions such as mobilizing and redistributing energy, and in concert with AVP, affects cardiovascular responsivity by raising blood pressure (BP)
through vasoconstriction. Cortisol also amplifies energy availability by increasing blood sugar via gluconeogenesis, and to insure glucose is available as an immediate energy source for major muscle groups, it inhibits the production of insulin to prevent free glucose from being stored. Cortisol not only serves a function of increasing energy availability for muscle utilization, but it acts as a steward of this energy, and in an attempt to conserve energy, cortisol redistributes energy by suppressing immune function and inflammation (Wilmore and Costill, 2004). It is important to note that these physiologic changes are not realized instantaneously. In fact, timing is an especially critical element pertaining to cortisol’s secretion. Plasma concentrations increase within 10 minutes of a perceived stress and reach peak concentrations approximately 20 minutes upon cessation of the stimulus (Foley and Kirschbaum, 2010). Cortisol concentrations then begin to reduce with the passage of time toward homeostasis.

Homeostasis is achieved by a cortisol mediated negative feedback loop. When cortisol levels become excessively elevated, several regions of the brain with high glucocorticoid and mineralocorticoid receptor density support the downregulating activity of the HPA axis. Cortisol participates in this negative feedback loop by blocking the secretion of CRH and ACTH, and thus, preventing further glucocorticoid secretion. De Kloet, (2004) noted that chronic elevations of cortisol could increase the duration of the physiologic stress response by decreasing the levels of central glucocorticoid receptors, effectively impairing the downregulation of the HPA axis. Therefore, it’s suspected that a continued release of cortisol by a hyper-activated HPA axis coupled with the inhibition of the glucocorticoid-mediated negative feedback loop is a logical mechanism that may
facilitate chronic hypercortisolemia and the medically related comorbidities as a result (Mackin and Young, 2004; Randall, 2011; Zschucke et al., 2015).

Other studies have looked at the impact that stress has on neuroendocrine reactivity and have reported polarizing results to the levels of plasma and urinary cortisol concentrations. Yehuda et al. (1995) observed blunted reactivity in a non-treatment-seeking PTSD population of holocaust survivors, which agreed with the author’s similar cortisol reactivity observation in previous work with combat veteran (Yehuda et al., 1990). In contrast, Pitman et al. (1990) showed no such correlation existing with combat veterans with PTSD, and moreover, a study looking at trauma induced PTSD in children suggested that low levels of baseline cortisol may compensate for periods of higher cortisol levels that accompany stress (Gotovac et al., 2003). Additionally, a recent meta-analysis of 58 studies investigating stress mediated adrenal fatigue and the ensuing hypoadreno-response elucidated these conflicting findings and concluded that there exists no substantial evidence that adrenal fatigue is a medical condition (Cadegiani and Kater, 2016). Clearly the research linking stress and HPA axis dysfunction is contradictory as to which variance occurs most frequently and under what conditions.

It is assumed and currently believed by the majority of investigators that variability in HPA response is attributed to both stressor and chronicity of those stressors where acute exposure promotes hypercortisolemia and chronic exposure may create hypocortisolemia (Fries et al., 2005); here we specifically look at the case of hypercortisolemia due to a hyper-activated neuroadreno response upon the onset of an acute stressful event and it’s subsequent rate of reduction during recovery.
Under normal conditions the secretion of cortisol follows a specific circadian rhythmicity. Concentrations reach a circadian peak in the morning, and then progressively decline from afternoon reaching a circadian trough in the early evening. Upon the first few hours of sleep, cortisol concentrations abruptly elevate and continue until awakening (Sack et al., 2007). When cortisol levels deviate from this rhythmicity throughout the wake cycle many serious and negative health outcomes can result. Previous studies have investigated these medical ramifications and have demonstrated that prolonged exposure to abnormally high concentrations of cortisol result in a variety of negative health outcomes. These comorbidities include: Type II diabetes and metabolic syndrome (Siddiqui et al., 2015), immune suppression (Randall, 2011; Strahler et al., 2015; Stenius et al., 2011), gastrointestinal disorders (Butorina et al., 2013), cardiovascular disease (Kulkarni et al., 1998; Carroll et al., 2003; Matthews et al., 2004; Kaplan et al., 1996), obesity (Vicennati et al., 2014; Epel et al., 2000), memory dysfunction (Sapolsky R, 1992; Smith M, 1996; Tatomir et al., 2014), and depression and anxiety (Mackin and Young, 2004; Mantella et al., 2008; Yonekura et al., 2014; Delaney E, 2015).

Like cortisol, catecholamines (norepinephrine and epinephrine) share similar hormonal as well as neurotransmitter roles. From the locus coeruleus located in the Pons, norepinephrine is produced by closely packed brain cell neurons that when secreted increases alertness, promotes vigilance, enhances and promotes the formation and retrieval of memory, and focuses attention; however, elevated levels will also lead to increases in restlessness and anxiety (Mezzacappa et al., 1999).
Epinephrine is also implicated in long-term memory, but is primarily produced in the adrenal glands (Cahill and Alkire, 2003). When epinephrine is released due to an emotionally stressful event, it can adjust memory consolidation of the event, which aids in ensuring that the strength of the memory is proportional to the importance of the memory (McGaugh, 2000). An example of this memory consolidation is the learning process that occurs when a child sticks their finger into an electrical outlet for the first time. The resulting painful experience releases epinephrine that assists in ensuring the strength of the memory formed is relative to the memory’s importance. Mezzacappa et al., (1999) demonstrated that there are learned associations between negative feelings and levels of epinephrine and that elevated levels of epinephrine have an impact on the intensity and recall of negative memories moderated by a fear stimulus.

When functioning as a hormone from sympathetic activation of the adrenal medullary, both epinephrine and norepinephrine gain further access to a wide variety of tissues through the vasculature system (Hamill et al., 1996). Both increases heart rate and blood pressure, triggers the release of glucose from energy stores, increases blood flow and glucose to skeletal muscles, and reduces blood flow to the gastrointestinal system (Bell Drl, 2009).

The sympathetic nervous system is the primary pathway that the immune system and the brain interact. The thymus, lymph nodes, and spleen receive these neural inputs, and though the effects from this process is complex, some immune processes are activated while others are mostly inhibited (Kenney MJ and Ganta CK, 2014). Unlike cortisol, both norepinephrine and epinephrine are rapidly degraded into their associated biologically inactive metabolites and excreted through the urine (Nussey et al., 2001).
Though its persistence to remain circulating in the blood is not matched to cortisol, chronic catecholamine production is positively correlated with insomnia (Devine JK and Wolf JM, 2016; Ji X et al, 2015), heart disease (O’Donnell et al., 1987), nervousness and lowered immunity toward illness (Sandrini et al., 2014; Kox et al., 2014). Additionally, both children and adults with over-production of epinephrine often exhibit traits of Attention Deficit Disorder (ADD) (Won et al., 2011; Kim et al., 2013; Chamberlain S and Robbins T, 2013).

**Cross stressor adaptation hypothesis and physical exercise:**
A very common choice of intervention prescribed today to address the symptomology associated with stress and its covariates (fear and anxiety) is the use of pharmacotherapy and psychosocial interventions. Mindfulness meditation (Koszycki et al., 2016), self-efficacy (Wright et al., 2016), Cognitive Behavior Therapy (CBT) (Stiles-Shields et al., 2015; NIH, 2015), breathing exercises, and auto-regulation exercises (Zeidan et al, 2015) are very popular and often sought after approaches to attenuate the pathology of fear based internal dialogue that promotes anxiety and stress. In addition to traditional cognitive approaches, there exists evidence that aerobic endurance exercise is efficacious at mitigating the deleterious effects that coincide with distress and can be a robust multiplier to promote stress resilience (Smits et al., 2008; Ströhle A., 2009; Gaudlitz et al., 2014).
Exercise elevates mood within minutes of commencement and sustains the effect for hours upon cessation. In a study comparing antidepressants for patients with major depressive disorder, Blumenthal et al. (2007) concluded that 4 months of exercise was as equally effective as antidepressants. Additionally, Smits et al. (2011) discovered that individuals with high anxiety sensitivity who also reported high activity levels were not as likely to experience anxiety or panic episodes as individuals who exercised less frequently suggesting that exercise can ward off anxiety episodes. Though the exercise-mental health connection is becoming difficult to ignore, just how much exercise is needed, what mechanisms are responsible for the stress-buffering benefits, and why it occurs are questions that researchers are currently investigating, but still remain unanswered.

Studies have indicated that the secretion of cortisol and epinephrine from the endocrine and catecholaminergic systems during stress responses to psychological stimuli is similar to the endocrine and catecholaminergic responses induced by exercise and physical activity (Dunn et al., 2005; Klaperski et al., 2014; Zschuke et al., 2015). The hypothalamus does not seem to differentiate between a legitimate threat of wellbeing and/or safety, and a deliberate controlled exercise state, therefore it responds to purposeful and premeditated excise as an acute stress stimuli (Hackney, 2006; Gerber, 2013). Since the physiology of an exercise condition interestingly mimics that of a stressful condition, an assumption can be constructed that exercise may lead to mal-adapted perceptions of emotional discomfort and malaise. However, this is not true. Voluntary exercise elicits mood enhancing, anxiolytic, and rewarding characteristics and traits, and therefore, is seldom depicted or interpreted as a disagreeable stimuli/stressor.
by individuals (Reed and Ones, 2006; Wipfli et al., 2008; Gaudlitz et al., 2014; Wegner et al., 2014; Akatsuka et al., 2015).

One hypothesis for this paradox is that exercise can promote tolerance to distress through what is referred to as cross-stressor adaptation (Michael, 1957) suggesting that regular exercise leads to adaptations in the stress response systems that promote decreased physiological responses to psychological stressors. These adaptations, which are believed to be primarily neurobiological, lead to a reduction in sensitivity to subsequent homolytic stressors (i.e. exercise) as well as heterolytic stressors (other than exercise, i.e. distressful psychosocial events) through the repeated successions of physiologic stress that accompanies habitual exercise (Sothmann, 2006). Hence the positive psychological benefits manifested from this physiologic phenomenon are habituation dependent. One reason for the efficacy of this condition may be that stress adaptation results in an increased catecholamine capacity in the Central Nervous System (CNS) as well as reduced levels of resting catecholamines yielding a blunted and reduced reactivity to anxiolytic and stressful events and lowered baseline anthropometrics such as HR and BP respectively (Sothmann et al., 1996; Klasperski et al., 2014; Zschucke et al., 2015; von Haaren et al., 2015). Hackney, A. (2006) offers evidence that may elucidate potential neurobiological mechanisms by observing that the aforementioned repeated exposure to exercise training results in adaptations in the neuroendocrine system, such that there is a reduction in hormonal stress response to subsequent bouts of exercise and, in many cases, reduced circulating basal stress hormone levels following. Since Hackney’s (2006) study, the examination of the neuroendocrine stress response has gained some considerable
interest among researchers and reliable data supporting the cross-stressor adaptation using these parameters is beginning to emerge.

Zschucke et al. (2015) for instance, utilized the Montreal Imaging Stress Task (MIST). The MIST is a validated stress inoculation test consisting of a series of computerized arithmetic problems that must be solved mentally, and includes a social evaluative threat component either built into the program or presented by the investigator. The difficulty and time limit of the problems are manipulated to be just beyond the individual’s ability to answer. In addition, individual, average and expected performance information is displayed for the subject to see. A final performance evaluation is given upon test completion. The study found that 30 minutes of aerobic exercise blunted a cortisol response in men subjected to a psychosocial stressor in the form of the MIST. The MIST is a validated stress inoculation test consists of a series of computerized arithmetic problems that must be solved mentally, and includes a social evaluative threat component either built into the program or presented by the investigator. The difficulty and time limit of the problems are manipulated to be just beyond the individual’s ability to answer. In addition, individual, average and expected performance information is displayed for the subject to see. A final performance evaluation is given upon test completion.

Moreover, a study by Klasperski et al. (2014) utilizing the Trier Social Stress Test, a validated instrument and a gold-standard laboratory stress task used to induce stress-arousal responses. (Kirschbaum et al., 1993; Dedovic et al., 2005; Aschbacher et al, 2013), discovered that a 12-week training protocol significantly reduced cortisol stress reactivity over a control group in response to the TSST
The transverse nature of physical exercise allows the body to become familiar with the stress response so that subsequent distressing events elicit muted physiologic reactivity to those stimuli; however, more studies are needed to elucidate the potential psychological benefits that may be harvested from this adaptation. Several studies have investigated the validity for the cross-stressor adaptation hypothesis, but as it relates to stress recovery concerning cardiovascular stress parameters (Forcier et al., 2006; Jackson and Dishman, 2006; Stubbe et al., 2007). While Jackson and Dishman (2006) did not demonstrate that the stress from acute bouts of controlled exercise reduces cardiovascular reactivity similar to Forcier et al., (2006), both investigating groups did demonstrate that repeated sessions of moderate exercise lead to adaptations which allow improved cardiovascular recovery from psychological stressors supporting the inclination that stress from purposeful exercise impacts the body’s efficacy in the recovery process from stressful events. These studies as well as others (Albright et al. 1992; de Geus et al. 1993; Calvo et al. 1996; Spalding et al. 2000; Jackson and Dishman 2006; Rimmlele et al. 2007, 2009; Klaperski et al. 2014; Childs and de Wit 2014) have not empirically investigated the cross-stressor adaptation hypothesis by specifically investigating the reactivity from physically fit individuals, but individuals classified as unfit or sedentary to various psychological stress tasks. Within the current literature, fitness levels have not been taken into account and no studies have looked at the stress buffering effect of cross stress adaptation on populations that are currently physically active. This issue in study design may be responsible for the noted discrepancies in the aforementioned studies. Even though the cross-stressor adaptation hypothesis is typically described as a within-subject hypothesis that has a clear temporal order (i.e. physical exercise does reduce the
physiological stress reactivity to psychological stressors over time within any given participant), most studies have used a between-subject design. Whether primarily out of convenience or not, the studies have not investigated participants over a specific stretch of time. When comparing participants the study design is conducted in a cross-sectional manner. However, a within-subject design with intervention over time is necessary to effectively and empirically test the cross-stressor adaptation hypothesis.

Additionally, an interesting discovery from the current literature elucidates the employment of absolute exercise compared to the utilization of relative exercise intensity in studies investigating the cross-stressor adaptation hypothesis and/or the neuroendocrine stress response. Relative intensity relates to an individual’s specific level of fitness and is based on his or her maximum capacity to do work. This can be measured both with maximum oxygen uptake or maximum heart rate. In contrast, absolute intensity is a general measurement of intensity applied to any person and is usually measured as a metabolic equivalent (MET) (Wilmore and Costill, 2004).

Because individuals have differing capacities and tolerances, a relative measurement allows for a more fair assessment of the impact that an exercise intervention will have on a measured outcome. Since HIIT requires a participant to attain 100% maximal effort measured by MHR, and MICT requires 80% intensity, the employment of relative intensity was utilized.

A popular and exceedingly cited meta-analysis documenting over 100 studies found that most studies investigating the therapeutic effects of exercise as it relates to distress incorporated submaximal exercise as the investigated intervention (Petruzzello et al., 1991). It has only been within the last decade that the role of intensity has become a
fervid matter of discussion, and out of this curiosity maximal exercise intensity has become a popular topic of some research interest. In a PubMed search of “high intensity interval training” as of March 2017, there are over 568 referenced articles solely addressing HIIT. Only two years previously (2015), there were only 280 references addressing solely HIIT, 55 in 2005, and only 10 in 1995. According to the American Psychological Association (APA) there still exists a proclivity towards researching and prescribing Moderate Intensity Steady State Training (MIT) for the mitigation of distress symptomology, but cross stressor adaptation suggest that higher levels of intensity can promote similar outcomes with less investment of time and superior anthropometric outcomes.

**Moderate Intensity Continuous Training (MICT):**

The current recommendation by the Anxiety and Depression Association of America (ADAA), National Institutes of Health (NIH), and American Psychological Association (APA), is to engage in 30 minutes of daily continuous moderate intensity aerobic activity (submaximal steady state, 60-70% VO_{2MAX}) to combat the undesirable effects of distress. Aerobic exercise is a continuous locomotive movement sufficient to increase heart rate and oxygen consumption, usually sustained for thirty minutes or more (Dietrich et al., 2004). According to The American College of Sports Medicine (ACSM), the intensity of an aerobic exercise training routine is typically measured by the use of age-predicted heart rate (220-age), workload measured in Watts, or by percent VO_{2MAX}.

When the rate of work is held constant at submaximal exercise level, HR will raise rapidly until it plateaus at an optimal rate for meeting the circulatory demands. This
plateau is referred to as “steady state” (Wilmore and Costill, 2004). The use of submaximal steady state exercise to relieve distress symptomology relies on restoring homeostasis through hemodynamic, endocrine, and metabolic adaptation that are paradoxically characteristic of acute stressors (Zschucke, 2015). Given that exercise acts as eustress stimuli, there has been an extensive amount of research addressing the therapeutic effects that exercise has on mental health (Hamer et al, 2006). Unfortunately, as explained by Wilmore and Costill, (2004) once an individual has reached $V_{02\text{max}} > 60$ ml/kg/min, performance is not improved by a further increase in submaximal training volume. Exercise allows for physiologic adaptations to allow the body to become familiar with subsequent HPA axis activation. However, extending the duration under steady state conditions does not specifically yield increased responses from the HPA axis. An additional stressor must be introduced to facilitate an additional stress response. Intensity is considered an additional stressor that leads to higher plasma cortisol and catecholamine levels. Since the cross-stressor adaptation hypothesis depends on the adaptations in stress response physiology (Childs and de Wit, 2014), an increased resilience to distress would not be expected from an increase in exercise volume at moderate intensity. HIIT may address this issue, but very few studies have addressed the effect of a high intensity interval training protocol on the physiological stress response and its potential to attenuate distress related symptomology similarly or superiorly to results currently demonstrated with MICT.

**High Intensity Interval Training (HIIT):**
In contrast to MICT, HIIT is typically a 15-20 minute aerobic activity that is comprised of short intervals of maximal effort (>90% \( V_{O2\text{MAX}} \)) followed by longer intervals of moderate intensity effort (<70% \( V_{O2\text{MAX}} \)) suspended with recovery phases between circuits lasting approximately 2 minutes.

Although HIIT has been around for decades (Tabata et al., 1996), it is currently a trending phenomenon that has recently gained celebrity by credit of fitness center boot camps, Crossfit, Tabata training, social media platforms, and generation “FIT”. HIIT has earned its significant popularity and high compliance in recreational and professional fitness programs because it can incorporate a high degree of variability to exercise bouts, works on all exercise modalities, decreases a participant’s time investment, and can be modified for individuals of all fitness levels and special conditions. The physiological, health, and performance outcomes may be the largest enticements and arguments for HIIT. ACSM cites aerobic and anaerobic fitness, blood pressure maintenance, cardiovascular health, reduction of insulin sensitivity, improved cholesterol profiles, and abdominal fat reduction and body weight management as benefits to exercise regimens that incorporate HIIT. Elmer et al., (2016) demonstrated that HIIT decreased plasma TRG (p < 0.05) when compared to moderate training. Stoa et al., (2017) demonstrated that a 12 week 4x4 walking/up-hill running protocol leads to the reduction in HbA1c by -0.58% points (p < 0.001). Quantitatively, HIIT increases measured athletic performance. Gunnarsson and Bangsbo, (2015) discovered that individuals subjected to a HIIT running protocol improved a 1,500-Km run by 6% and a 5-Km run by 4% (P>0.01) compared to a control group that maintained a MIT protocol.
HIIT has been shown to lower blood pressure (Tjønna et al, 2008; Shiraev and Barclay, 2012), supports muscle hypertrophy (Gunnarsson and Bangso, 2015), and reduces triglycerides (Shepherd et al, 2013) in a manner that is superior to moderate intensity steady state training (Racil et al 2013). Critically, it has been argued that high-intensity activity may be difficult to organize in the community and has less appeal for the sedentary and/or elderly in the long term than low-intensity aerobic training (Gibala, 2007); however, using an instructor-led gym-based, 10 week HIIT protocol vs. moderate intensity steady state training protocol study, Shepherd et al, (2015) demonstrated that adherence to a HIIT intervention was superior compared to MICT for inactive individuals. Individuals engaged in low-volume HIIT exercise report greater enjoyment and higher levels of overall energy when compared to individuals of a MICT regimen (Thum et al., 2017). The reduced time needed to complete a session coupled with the ability to introduce generous modification and modality diversity has shown HIIT to be well tolerated by most individuals, and compliance is reported to be high despite having a considerably higher rating of perceived exertion during exercise conditions (Bartlett et al., 2011; Shiraev and Barclay G, 2012).

Secretions of glucocorticoids (cortisol) and catecholmines (Norepinephrine and epinephrine) are dose dependent, and repeated successions of a maximal physical exertion from HIIT evoke physiological responses that are consistent with the release of glucocorticoids and catecholmines that occur under an intense psychological stress response. Since the aforementioned studies have demonstrated that exercise does have the capacity to create physiological adaptations that allow the body to become familiarized with and increase tolerance to subsequent additional stressors and because
HIIT induces greater physiological stress on the body than submaximal exercise, tolerance through cross stress adaptation may lead to reduction in cortisol reactivity as well as a similar or superior reduction in heterlytic stress sensitivity leading to lower perceived stress.

**Rationale:**

Biondi and Picardi (1999) examined the use of real-life stressors (academic examination, daily occupational pressures and parachuting) and though they illicit valid stress responses, the use of such stressors lend too much variability to be utilized with any degree of precision. Therefore, the use of the Trier Stress Social Test (TSST) was employed as an acute stress-inducing event. The TSST is a validated instrument and a gold-standard laboratory stress task that has been used for over 2 decades to systematically induce a stress-arousal response in order to measure differences in reactivity, anxiety and activation of the HPA or SAM axis during the task. (Kirschbaum et al., 1993; Dedovic et al., 2005; Birkett, M., 2011; Aschbacher et al, 2013; Gerber et al., 2008). Moreover, Dickerson And Kemeny, (2004) noted that salivary cortisol could be used as an indication of HPA activity due to psychosocial stress when that stimulus is based on a performance task that has social-evaluative threat and unpredictability similar to the TSST. Since free, bioactive cortisol fraction in the blood can be reliably determined through the measurement of cortisol in saliva (Klaperski et al., 2014), the
collection of cortisol through the saliva was used as a valid physiological measure of perceived stress during the administration of a TSST in all of the stress response assessments.

Additionally, it has been observed that individuals who are initially submitted to the TSST and are then given an exercise intervention show markedly lower stress response by means of salivary cortisol and heart rate to a subsequent admission of the TSST (Klaperski et al., 2014; Zschucke et al., 2015); however and unfortunately, there has been very little consensus as to the intensity of exercise that produces the most robust stress-buffering effect. It is unclear whether training at maximal or near-maximal intensity is inversely associated with the prevalence of elevated cortisol, increased catecholminergic parameters or self-reporting heightened symptoms of distress upon and after a distressing event.

MICT is the status quo protocol for complimentary treatment in mitigating the symptoms of a hyper-stimulated HPA axis from distress. Although the predominant focus has been on the cardiovascular stress response, experimental evidence predominantly supports the cross-stressor adaptation hypothesis as a mechanism behind the efficacy of MIT. Since HIIT has been demonstrated to be superior in many areas compared to MICT and assumingly elicits a stronger response from the HPA axis, a logical and reasonable assumption is that HIIT may provide similar or superior capacity in reducing the physiological response to stress compared to MIT upon a subsequent admission of the TSST after 6 weeks of exercise intervention.

The current research question of interest targets the feasibility that contrasting intensities of exercise may lead to differing levels of psychological benefits within a healthy and
physically active population. To determine the validity of this question, the aims of the proposed study were to account for fitness level of participants by excluding sedentary or individuals who were not physically active. Equally important was to utilize relative exercise intensity in exercise protocols through MHR described by the use of a VO$_{2\text{MAX}}$ testing, and design a within-subject investigation with clear temporal order. The specific aims of this study was to test the hypothesis that 6 weeks of HIIT exercise sessions will (1) reduce the glucocorticoid and catecholaminergic reactivity upon exposure to an acute psychosocial stressor; (2) impact the sympathetic recovery response through the observance of blunted physiologic measurements (BP, HR, and SA); (3) attenuate the perception of stress using self-reporting psychological inventories in participants post intervention compared to pre intervention as efficaciously when compared to MICT, but with less time investment.

CHAPTER TWO: METHODOLOGY

Experimental Design:

The study was conducted as a randomized controlled trial (RCT) containing two groups: a 6-week high intensity interval exercise training group (HIIT) and a 6-week moderate intensity continuous exercise training group (MICT). The MICT group served as a baseline for comparison for assessing the effects of the HIIT intervention, and to illustrate the effect of each intervention, each participant served as his or her own control by
comparing baseline measures with post intervention outcomes. All current regular training sessions were replaced with three weekly training sessions as indicated by the participant’s randomly assigned exercise group. Age eligibility was based on the current American College of Sports Medicine’s (ACSM) Guidelines for Exercise Testing and Prescription for exercise risk stratification and Physical Assessment Activities Readiness Questionnaire (PAR-Q) and Health History Questionnaire (HHQ) was assessed prior to conducting a maximal treadmill test. The test was administered to determine individual Maximal Heart Rate (MHR) and Maximal Aerobic Speed (MAS) at VO$_{2\text{MAX}}$ to assist in determining the degree of intensity to be prescribed at intervention. VO$_{2\text{Max}}$ was determined by attainment of 1.2 Respiratory Exchange Ratio (RER), gas exchange and HR plateau, participant’s Rating of Perceived Exertion (RPE, >17 on the Borg scale), and attainment of age predicted heart rate (220-age). No sooner than one work week prior to the intervention period (5-7 days), all subjects completed a series of base line measures including: 1) The Spielberger State Trait Anxiety Inventory (STAI), 2) Perceived Stress Scale-10 item (PSS-10), 3) BP, 4) HR, and 5) SC during the initial stress response assessment (see Figure 2.) Post-intervention, participants were reevaluated by measures 1 – 5 during the final stress response assessment within and not exceeding 1 week following the final exercise session (2-5 days). Recruitment occurred in two stages since the targeted number of participants could not run through the study at the same time for logistical reasons. The study was conducted in two separate, but identical cohorts. Cohort 1 occurred from October 2016 to December 2016, n = 19; cohort 2 occurred from January 2017 to April 2017, n = 5. The Washington State University Institutional Review Board has reviewed and approved this research for human subject participation.
Participants:

Twenty four moderately trained (spending more than 10% of their daily energy though physical activity) (Bernstein, 1999) subjects with an age, height, weight, BMI, and \( VO_{2\text{MAX}} \) of 29.25 ± 9.53 yr., 169.67 ± 11.01 cm, 66.23 ± 11.13 kg, 22.8 ± 2.39 kg/m\(^2\), 39.8 ± 6.02 ml/kg/min., respectively, participated in the study. Nine participants were men (M = 27.77 ± 5.92) and 15 participants were women (M = 30.40 ± 10.81). Thirteen percent (n = 2; HIIT, n = 1; MICT, n = 1) were in the luteal phase of the menstrual cycle during the stress response assessment. Thirty-seven participants were recruited from the general and university population by use of social media, informational flyers, e-announcements at area universities, and word-of-mouth. To ensure a balanced distribution, gender was equally distributed across the two groups and participants were randomly assigned following simple randomization procedures (computerized random numbers) to 1 of 2 treatment groups. The subjects were divided into a HIIT group (n=12) and a MICT group (n=12). A detailed flow of participation is presented in figure 2. Thirty-seven individuals expressed initial interest during the recruitment period and came into consideration for participation as they reported they were of qualifying age, did not have any physical limitations to running, and were currently spending more than 10% of their daily energy in physical activity. These individuals were contacted by telephone, given a brief explanation of the study, and
advised of their time commitment associated with the study. From this pool who met inclusion criteria, \( n = 32 \) conveyed continued interest in participation. These individuals were informed of the intervention protocols, pre-measure testing, and post-measure testing, and then reviewed for eligibility by use of a screening interview, which included administration of the Physical Activities Readiness Questionnaire (PAR-Q) and exclusion criteria. In total \( n = 27 \) participants were eligible. These individuals were invited to the research facility to complete a consent form, Health History Questionnaire (HHQ) and the PAR-Q. Anthropometric measures were collected and subjects underwent VO\(_{2}\text{MAX}\) exercise testing to determine Maximum Heart Rate (MHR) and Maximum Aerobic Speed (MAS). VO\(_{2}\text{MAX}\) testing was performed using the 12-lead Vmax Vyntus\(^\text{TM}\) CPX & ECG (Sentrysuite software), Carefusion Hoechberg, Germany. Upon completion of exercise testing, subjects were rescheduled for pre-intervention stress response evaluation and testing (PT-1). A total of \( n = 24 \) participants completed the study. Of the 27 eligible and consenting participants, 3 individuals dropped out citing time constraints at the onset of the intervention (HIIT \( n = 1 \); MICT \( n = 2 \)). See Figure 2 for a detailed participant flow.

**Inclusion criteria:**

All participants will need to meet the following entry criteria:

(a) Be between the ages of 18-45 for males and 18-55 for females
(b) Currently physically active (expending more than 10% of daily energy in activity)
(c) Be willing to be randomized
(d) Be available to attend the post intervention stress response evaluation at the same time of day the pre intervention stress response evaluation was conducted
(e) Be available for the duration of the study.

Exclusion criteria:

Any participant meeting the following criteria will be excluded from participation:

(a) Hospitalized for psychiatric disorder in last 5 years
(b) Attempted suicide within last 2 years
(c) Unable to read, understand, and provide written consent
(d) Plan to relocate from the Spokane area within 4 months
(e) Currently participating in an ongoing clinical/research study/trial
(f) Unable to perform High Intensity Interval Training due to a medical condition;
(g) Currently planning pregnancy or current pregnancy
(h) >30 BMI
(i) Consume > 21 alcoholic drinks/week
(j) Use and/or abuse of illegal substances
(k) Determined Physical Activity Readiness Questionnaire (PAR-Q) disqualifiers
Fig. 2. Participant recruitment and flow diagram within the study design

**Procedures:**

*Pre-intervention and post-intervention test*

Participants participated in two separate *stress response evaluation* sessions that were procedurally identical. However, because subjects had familiarity with the testing sequences, different panel members were exchanged for the originals during final testing. The first session (PT-1) was performed 1 week prior to the intervention and the last session (PT-2) was performed within one day preceding the final intervention session. Participants were fully informed of the experimental procedures, given a consent form,
and given an informative package with detailed study information that addressed the possible discomforts associated with participation at PT-1. At the conclusion of PT-2, participants were thanked for their participation and debriefed. To control for diurnal cortisol secretion deviation, the collection of cortisol during the PT-2 was timed so that it was taken at the same time of day as compared to cortisol collection during PT-1. In preparation of the PT-1 and PT-2, participants were instructed to refrain from smoking and eating 1.5 hours prior to testing, to refrain from consuming alcohol, coffee, milk, green or black tea, or other caffeinated drinking products 12 hours prior to testing, to refrain from exhaustive physical exercise and dental work 24 hours prior to testing, and to have a regular breakfast and lunch, but refrain from consumption of juice, soft drinks, chewing gum, red meats (especially traditionally farmed variants), shellfish, tree nuts, dairy, refined sugar, and chocolate in either meals. Participants provided a signed consent form to complete prior to beginning PT-1 and were advised that they could discontinue the study at any juncture regardless of their consent.

**Stress Response Evaluation**

The stress response evaluation sessions (PT-1 and PT-2) were comprised of a stress response assessment (lasting approximately 2 hrs.) that was broken into 3 separate and distinct phases as depicted in Fig. 3. The first phase (*preparation*) was comprised of 3 additionally separate stages collectively lasting 45 minutes. Subjects were briefed, completed forms, and rested during the first stage. Baseline measures were taken during the second stage, and preparation for the Trier Social Stress Test (TSST) occurred during the third stage. The second phase (*presentation*) consisted of the 10-minute TSST.
Subjects were asked to present an unrehearsed speech and then asked to complete a mental mathematic task in the presence of judges and multiple video cameras. In use for over 2 decades, the TSST is a standardized and valid stress inducing performance task protocol that systematically induces a stress response in order to measure reactivity, anxiety, and the activation of the hypothalamic-pituitary-adrenal or sympathetic-adrenal-medullary axis during the event (Birkett, 2011, Kirschbaum et al., 1993). It combines high levels of three (novelty, uncontrollability and socio-evaluative threat) of the four determinants of stress and reliably elicits the body’s physiological response manifested by psychosocial stress. The result of the TSST observes marked increases in SC, ACTH, catecholamines (epinephrine, norepinephrine), growth hormone, prolactin, testosterone, several immune parameters (e.g., neutrophils, eosinophils, basophils, lymphocytes, IL-6, TNF-alpha), alpha-amylase, heart rate, heart rate variability, and systolic and diastolic blood pressure levels. Increases of these biomarkers following TSST exposure have been extensively studied and used as valid measures investigating the efficacy of stress-buffering interventions.

(Dickerson and Kemeny, 2004; Birkett, 2011; Kudielka et al, 2014; Klaperski et al, 2015). The last phase (recovery) consists of 2 stages lasting a combined total of 50-minutes. During the first stage, subjects were given 40 minutes to recover from the TSST, and had BP, HR, and SC measures taken directly, 10 min, and 20 min after TSST. In the second and last stage, subjects then took a cognitive test and completed a self-reporting psychological inventory.
**Preparation**

The preparation phase was composed of 3 stages. In the first stage, all participants received an explanation of saliva sampling and were allowed to continue if they had not engaged in vigorous physical activity or had any dental work or oral injuries in the last 24 hours. As well, they were also cleared against consumption of alcohol, caffeine, nicotine, and any prescriptions or over-the-counter medicines with a pretest questionnaire. Each participant was then asked to rinse his or her mouth with water for 10 seconds, and then assigned a room. All participants were unaware of the TSST protocol. Initial levels of how participants perceived the level of stress in their lives were measured with the Perceived Stress Scale-10 (PSS-10). Upon completion of the PSS-10, all personal property, bags, reading material, and smart devices were removed from the participants and safely secured until the end of the stress response evaluation. Neutral reading material were provided, and participants were left to be alone in their assigned rooms to relax for 30-minutes.

In the second stage, baseline measures were collected. All BP measurements were collected on the same device (Omron®, HEM-907XL, Omron Healthcare, Inc. Vernon Hills, Illinois) while in an upright sitting position. Participants donned an HR device (MIO®, ALPHA, Physical Enterprises Inc. Vancouver, BC,) to determine both baseline HR and HR reactivity during the TSST. SC was collected using the SalivaBio Oral Swab (SOS) system (Salimetrics™, San Diego, CA.). All swabs were held sublingual for 1 minutes and 30 seconds, stored in 17mm X 100mm centrifuge storage tubes, and kept frozen at -20°C. Deceptive practices were employed to reduce learning effect and diminish possible contamination of natural stress responses. Participants were instructed
that they would be tested on their cognitive functions over a variety of tests and tasks. In keeping with this doctrine, participants were then provided with a blank sheet of white paper and colored pencils and asked to draw a picture of a person in 5-minutes with no further instruction.

In the third and final stage of the preparation phase, participants were informed that they would have 5-minutes to prepare for an oral presentation in which they will be tasked to convince a panel of individuals that they are the best qualified candidate to fill the vacancy for a particular job. Participants were informed that the panel consisted of experts in public speaking and the evaluation of non-verbal behavior, and that their presentation would be recorded so that a video analysis of their performance could be later evaluated. It is important to note that the procedures of PT-1 differ slightly than in PT-2. At PT-1 participants were instructed that they would be applying for a position in their own industry and area of expertise and familiarity. However, to preserve novelty and include unpredictability, participants were instructed that they would present for a position that was outside of their industry and/or area of expertise during PT-2. Upon completion of the preparation phase, participants had BP and HR measurements taken prior to being chaperoned into a separate room containing the waiting panel member to conduct the TSST presentation.

*Presentation* -

Participant stood in an upright position in front of microphone and presented to an interviewing panel seated at a table with 3 cameras situated to capture frontal, side, and rear views of the presentation. One designated member of the panel acted as the moderator and solely communicated directions to all participants. The remaining panel
members observed in silence, withholding any verbal AND non-verbal feedback, throughout the entire presentation. Each participant was unknowingly given 5 minutes to deliver his or her prepared speech. If the participant stopped talking during the presentation, the panel moderator allowed them 20-seconds of silence. Once 20-seconds had passed, if the participant had not resumed speaking, the panel moderator would prompt the participant to continue speaking by instructing: "You still have time remaining." At the end of the 5-minute presentation task, the participant was thanked and then promptly instructed that they would next perform a mental mathematical task. Each was unknowingly given 5-minutes to sequentially subtract the number 17 (at PT-1) and 37 (at PT-2) from 1022 and 20253, respectively, aloud and as quickly and accurately as possible. If a mistake was made, participants were prompted with, "That is incorrect, please start over from 1022 (or 20253 in PT-2)." until time had expired. At the conclusion of the allotted time, participants were thanked for their time and instructed to exit the room. Participants were escorted back to their room and an HR, and BP reading were recorded and saliva sample taken. HR was purposefully observed during the entire TSST and the maximum heart rate (MHR) obtained was recorded. At cessation of the immediate measures post TSST; HR monitors were removed from participants. Care was taken to disrupt familiarity with the testing procedure during the TSST. This was accomplished by exchanging the previous panel members of PT-1 for the participant’s peers during PT-2 (Cheetham and Turner-Cobb, 2016).

Recovery -

Immediately after collecting SC samples, BP, and HR measures, participants were again given a blank sheet of paper and asked to draw a picture of a person for 5-minutes. After
five minutes, the picture was collected and the participant was allowed to sit and relax for
10-minutes. BP and HR measures and SC samples were taken after this 10-minute period
and taken again following an additional 20-minute rest period. Three SC samples and HR
and BP measures were collected directly following, ten minutes post, and 20 minutes post
TSST concluding with a total rest time of 30-minutes post TSST. Administration of the
Montreal Cognitive Assessment (MoCA) was immediately given followed directly by the
Speilberger State Trait Anxiety Inventory (STAI). Participants were thanked for their
time and cooperation and briefed as to which intervention group they had been randomly
assigned. Exercise sessions began no more than 48 hours following the stress response
evaluation for the 6-week exercise intervention.
**Intervention**

After completion of PT-1, $n = 12$ participants started and completed the MICT exercise protocol and $n = 12$ participants started and completed the HIIT exercise protocol. Participants had three supervised training sessions per week with no less than 48-hours rest between exercise sessions for a total duration of 6-weeks. The 48-hour rest period
between sessions was adopted to assist in the participant’s acclimation to the physical stress relating to their randomly assigned exercise and to assist in mitigating the effects of delayed onset muscle soreness (DOMS). Beyond allowing for muscle repair upon cessation of exercise bouts, rest periods were also used to control for deconditioning effect and were strategically used to facilitate participant retention. This process was quite efficacious in that attrition was 0% once the intervention phase was initiated. Participants were counseled to increase fluid intake throughout the day so that a total daily consumption of water meet 60+ oz. They were also given dynamic and static stretches specifically targeting Quadriceps Femoris, Gastrocnemius, Biceps Femoris, Iliopsoas, and the Gluteal muscles. These stretches included: leg swings, walking lunges, skip-jumps, grapevine, butt-kickers, and soldier walk for dynamic stretches and kneeling hip flexors, standing hamstrings, standing quad, supine cow, and standing calf stretches for static stretches. Participants were also encouraged to adhere to consuming a post exercise protein and carbohydrate meal consisting of a 2:1 protein to carbohydrate ratio within 2 hours. Additionally, they were informed about ice pack and bath therapy, counseled to increase omega-3 consumption to 2g daily, shown myofascial release massage techniques using foam rollers, encouraged to engage in active recovery, and received instruction on nonsteroidal anti-inflammatory use.

RPE and heart rate controlled running modalities based on the initial VO$_{2\text{MAX}}$ tests were used in each group to determine attainment of proper protocol intensity. Allocated exercise time differed between groups whereas the least amount of time commitment was 39 minutes per session and 117 minutes per week for the HIIT protocol and the maximum amount of time commitment was 50 minutes per session and 150 minutes per
week for the MICT protocol. In total \( n = 7 \) completed all sessions (HIIT \( n = 4 \); MICT \( n = 3 \)), \( n = 10 \) completed all but one session (HIIT \( n = 4 \); MICT \( n = 6 \)), \( n = 4 \) completed all but two sessions (HIIT \( n = 2 \); MICT \( n = 2 \)), and \( n = 3 \) completed all but 3 sessions (HIIT \( n = 2 \); MICT \( n = 1 \)). Exercise was performed on the PRO FORM® Performance 400 Treadmill, (Model: PFTL59513.2, Icon health & Fitness, Inc. Logan, UT.) and the TRACKMASTER® Treadmill (Model: TMX425C, FullVision, Inc. Newton, KS.)

**MICT Group** -

In the MICT group, participants \( (n = 12) \) received 3 supervised 50 minute training sessions per week. Each training session began with a 5-minute warm-up on the treadmill at 5.28 ± 0.32 km/h so as not to exceed 70% MHR followed by five dynamic stretches (skip-jumps, butt-kickers, soldier walk –walking toe touches, grapevine, and ladders). A 30-minute steady state treadmill run at 80-85% maximum heart rate (MHR) immediately proceeded the dynamic warm up. To ensure that participant HR stayed below 85% MHR, subjects were asked to carry-on a continuous conversation for the entirety of the session and HR was monitored at 3, 6, 9, and 12 minutes or until steady state. Five minutes of cool down proceeded the steady state exercise session at 5.28 ± 0.32 km/h followed by five stretches specifically targeting Quadriceps Femoris, Gastrocnemius, Biceps Femoris, Iliopsoas, and the Gluteal muscles.

**HIIT Group** -

Participants of the HIIT group utilized a training concept similar to and based on the protocol referred to as the 10-20-30 training concept (Gunnarsson and Bangsbo, 2012).
Participants had an identical warm up as the MICT group with a treadmill speed of 5.63 ± 0.36 km/h and participated in an identical dynamic stretch protocol. A 19-minute HIIT session ensued containing 3 X 5 minute running sets with 2 minutes of active recovery between each interval. Each 5-minute set was broken into five 1-minute intervals. Each 1-minute interval was further separated into 30 second, 20 second, and 10-second periods with contrasting levels of intensity equating to <30%, <75%, and 100% (maximum effort) MHR, respectively, based on the participants previously tested VO\textsubscript{2MAX} values (See Figure 4). Sessions were HR controlled and reported via MHR and RPE. To ensure participants reached protocol intensities, treadmill grades were manipulated for each interval and treadmill speeds were administered for each 30, 20, 10-second period in reference to the subject’s appropriate MHR.

Training was conducted by the investigator and 19 trained research assistants (RA). Each participant was assigned an assistant to administer the exercise protocol. RAs recorded all training session in individual training journals. To assess compliance the measurements of the participant’s HR was collected using the MIO ALPHA heart rate device. Each participant reached his or her assigned MHR and/or RPE during each exercise session administered and no participants were excluded from analysis due to noncompliance. The HIIT protocol was a modified version of the 10-20-30 concept (Gunnarsson and Bangsbo, 2012). Participants ran at stepped increases of speed starting at 4.82 kph for 30 seconds, proceeding to 11.27 kph for 20 seconds, and culminating at 16.09 kph for 10 seconds on the PRO FORM® Treadmill. Taller individuals were exercised on the TRACKMASTER® due to its longer belt. Increasing incline of the treadmill was necessary to establish MHR and buffer training effect. Though incline was
participant dependent, modification of the incline was necessary and practiced to effect attainment of MHR on each exercise session (mean incline = 6.27%, SD = 3.82). As a limitation of that protocol, neither the treadmill nor the human body can accelerate to a prescribed speed instantaneously. To accommodate for this, the protocol was adjusted to accommodate for the treadmill spool-up period needed to attain the appropriate speed for the participant’s all-out maximal effort. The time between the end of the 30-second period and the beginning of the 10-second period, formerly noted as the 20 second period, was used to “ramp” the speed to the desired maximum aerobic speed (MAS). This typically required 5 seconds, so that the protocol was staged in 30-second, 15-second, 5-second, and 15-second periods. The last 5 seconds of the 20-second period was used for acceleration so that each participant would have a complete 10-second period of time at MAS. See figure 5.
Round 1

<table>
<thead>
<tr>
<th>MINUTE 1</th>
<th>MINUTE 2</th>
<th>MINUTE 3</th>
<th>MINUTE 4</th>
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</thead>
<tbody>
<tr>
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<td>REPEAT</td>
<td>REPEAT</td>
<td>2 MINUTE ACTIVE</td>
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<tr>
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<td></td>
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<td></td>
<td></td>
<td>RECOVERY</td>
</tr>
<tr>
<td>20 sec</td>
<td>70%</td>
<td></td>
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</table>

Round 2

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<th>MINUTE 3</th>
<th>MINUTE 4</th>
<th>MINUTE 5</th>
<th>REST</th>
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</thead>
<tbody>
<tr>
<td>30 sec</td>
<td>30%</td>
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<td>REPEAT</td>
<td>REPEAT</td>
<td>2 MINUTE ACTIVE</td>
</tr>
<tr>
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<td>20 sec</td>
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Round 3

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<th>MINUTE 3</th>
<th>MINUTE 4</th>
<th>MINUTE 5</th>
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<td>REPEAT</td>
<td>REPEAT</td>
<td>REPEAT</td>
<td>2 MINUTE ACTIVE</td>
</tr>
<tr>
<td>30%</td>
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<td>RECOVERY</td>
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<td>20 sec</td>
<td>70%</td>
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<tr>
<td>10 sec - Max</td>
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</tbody>
</table>

Fig. 4. HIIT protocol
Fig. 5. HIIT Flow compared to MICT flow
**Measurements**

**VO\textsubscript{2MAX} and MHR -**

Prior to PT-1 and PT-2 each participant underwent VO\textsubscript{2MAX} testing to determine his or her absolute MHR and MAS. A protocol was created to allow participants to reach his or her MAS by incremental increasing speed over time. The majority of cardiopulmonary testing (i.e., Astrand, Balke-Ware, Bruce, Naughton, Taylor, Weber, etc.) is either sub-maximal or step graded protocols which didn’t fit the needs of determining MAS. Instead a protocol was used that disallowed for increases in grade, but increased speed over increments of time until the participant voluntarily stopped the test or until VO\textsubscript{2MAX} indicators were attained. Figure 6 details the specifics of the modified VO\textsubscript{2MAX} protocol.

Absolute indications to terminate the test (A drop in systolic blood pressure (SBP) of more than 10 mm Hg from baseline, despite an increase in workload, when accompanied by other evidence of ischemia, moderate-to-severe angina, increasing nervous system symptoms (i.e., ataxia, dizziness, near-syncope), signs of poor perfusion (cyanosis or pallor), technical difficulties in monitoring electrocardiographic (ECG) tracings or SBP, subject’s desire to stop, sustained ventricular tachycardia, and ST elevation (> 1 mm) in leads without diagnostic Q waves (other than V\textsubscript{1} or aVR leads) were adhered to and participants were assessed using the modified cardiopulmonary test protocol.

<table>
<thead>
<tr>
<th>Time</th>
<th>3min</th>
<th>2min</th>
<th>1min</th>
<th>1min</th>
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<th>30sec</th>
<th>30sec</th>
<th>30sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>kph</td>
<td>4.82</td>
<td>9.65</td>
<td>12.87</td>
<td>14.48</td>
<td>16.09</td>
<td>17.70</td>
<td>19.31</td>
<td>20.92</td>
<td>22.53</td>
</tr>
</tbody>
</table>

Fig. 6. Modified VO\textsubscript{2MAX} protocol
**Perceived Stress Scale** -

The 10-item Perceived Stress Scale (PSS-10) inventory was utilized to assess participant’s general level of stress (Cohen, et al., 1983). The PSS-10 is a widely used and valid instrument that efficaciously indicates an individual’s overall general perception of stress (Gerber et al., 2013). The PSS-10 was applied to assess general perceived stress during the past month. It measured the frequency with which participants had the perception of overloading, uncontrollability, and unpredictability. Answers to the scale are given on a 5-point Likert scale, ranging from 0 to 4 (never to very often, respectively). Scores are obtained by reversing the responses (e.g., 0=4, 1=3, 2=2, 3=1, and 4=0) to four positively statements (statements 4, 5, 7, and 8) and then summing the remainder points across the scaled statements. PSS-10 scores range from 0 to 40, with high scores indicating higher subjectively perceived stress levels and lower scores indicating the antithesis. Though scores differ across varying demographics, average PSS-10 scores have risen from an average of 12.07 ± 5.89 to 15.52 ± 7.44 since 1983. Individuals who score’s exceed 20 are considered to be under distress (Cohen and Janiki-Deverts, 2012). Since the sample size was < 100, a Cronbach’s alpha was replace for the mean with standard deviation. In the present sample the mean score was 14.08 ± 5.08 (HIIT 12.85 ± 4.47; MICT 14.9 ± 5.48).

**The Spielberger State-Trait Anxiety Inventory** -

The Spielberger State-Trait Anxiety Inventory (STAI) is a commonly used instrument to measure an individual’s perception of stress following a stressful event and task (Kirschbaum et al., 1993). The STAI is a self-report instrument. It was designed to
assess levels of state anxiety and trait anxiety, through 40 items scored by a Likert-scale. State anxiety can be defined as a transient momentary emotional status that results from situational stress. This allowed us to investigate the effect the TSST had on individual’s ability to cope with and recover from a single transient stressful event. Equally important, trait anxiety represents a predisposition to react with anxiety in stressful situations. This allowed us to determine internal consistency across both scales, but specifically allowed for any observed inconsistencies among personality traits of the participant population. Since the sample size was < 100, a Cronbach’s alpha was replace for the mean with standard deviation for “trait” anxiety of 38.26 ± 9.49 (HIIT 36.5 ± 6.00; MICT 40.18 ± 12.28). The STAI was conducted preceding the TSST and at the end of PT-1 and PT-2.

Over the past 35 years, salivary cortisol has been the most widely used biomarker to measure the stress response in stress-focused studies (Kirschbaum and Hellhammer, 1989). Catecholamines, unlike cortisol, cannot easily be measured in saliva. However, researchers have been able to non-invasively measure sympathetic nervous system activation through known responses such as blood pressure and heart rate using by-proxy methods. (Peterson et al., 2014; Victor et al., 2011)

*Blood Pressure*

Evidence has demonstrated a relationship exists between psychosocial stress and heightened BP response (Carroll et al., 2003; Matthews et al., 2004). As a good by-proxy measure of catecholaminergic response to stress, BP measures were compared to
response and recovery measures related to PT-1 and PT-2 to elucidate the intervention effect. BP was measured consistently using the identical automatic blood pressure monitor system (Omron HEM907XL, Omron Healthcare, Inc. Vernon Hills, Illinois). BP was measured in a standing position after rest, 1 minute preceding the onset of the TSST (both PT-1 and PT-2) and 2 minutes proceeding the TSST (See Fig. 3).

Heart Rate -
In a meta-analysis, Forcier et al., (2006) presented that physical fitness attenuates cardiovascular reactivity and improves recovery from acute psychological stressors. As an indicator of a valid proxy measure of sympathetic activation and release of catecholamine, heart rate was monitored in an upright standing position at baseline after rest, from 1 minute prior to the onset of the TSST (both PT-1 and PT-2), throughout the TSST, and 2 min after the cessation of stress exposure (see Fig. 3). MHR during TSST was noted by two observers and recorded. HR was effectively measured in beats per minute (bpm) using a wireless wrist monitor/watch (MIO® ALPHA, Physical Enterprises Inc. Vancouver, BC.). For internal consistency, as exampled with PSS-10, STAI, and BP, participants were matched for resting HR during randomization. In the experimental population the mean score was 71.5 ± 12.3 (HIIT 70.1 ± 12.3; MICT 72.8 ±12.2).

Cortisol -
(Salmon P., 2001; Rimmle et al., 2009; Klaperski et al., 2014) have demonstrated salivary cortisol to be a valid measure of the HPA axis to stress reactivity. Salivary cortisol was used to assess the response of the endocrine system to stress. To minimize
any anticipation bias, baseline saliva samples were collected after 30 minutes of rest and before participants were informed about the nature of the TSST (see Fig. 3). Additionally, to further minimize any bias, deceptive practices were employed in conveying the nature of the test. In total 8 saliva samples were gathered per participant. Four samples were collected prior to the exercise intervention and 4 additional samples were collected post intervention (30 minutes after rest, directly after administration of TSST, 10 minutes, and 20 minutes post TSST) using a commercially available sampling device (SOS, Salimetrics™, San Diego, CA.). Samples were then stored at −28 °C. Free salivary cortisol levels (nmol/l) were analyzed using HS Salivary Cortisol EIA kit from Salimetrics™ using a time-resolved fluorescence immunoassay plate reader and 4-parameter non-linear regression curve fit. The intra-assay coefficient of variation was between 4.0% and 6.7%, and the inter-assay coefficients of variation ranged between 7.1% and 9.0%. In the present study, cortisol concentrations were measured in duplicate, yielding a mean coefficient of variation between duplicate analyses of 5.0%. Samples were read at 450 nm.

Statistical Analysis -

Demographic and baseline characteristics were compared using t-test analysis. To examine if a significant difference existed between HIIT and MICT amongst PSS-10, STAI, and MHR measures pre and post-intervention a mixed-design (repeated measures with a between subject factor) ANOVA was conducted. A paired samples t-test was conducted independently for HIIT and MICT to elucidate any significant differences of SBP, HR, and SC within each group. A mixed-design ANOVA with repeated measures
was used to determine the main effect of SBP, HR, and SC between groups. To examine whether exercise had an effect on participant’s global perception of stress as a stand-alone independent variable, PSS-10 and STAI test score were analyzed for differences between PT-1 and PT-2 across all participants using a paired samples t-test. In addition, MHR response to stress were utilized to examine whether exercise (in general) had an effect on participant stress perception using a paired samples t-test. A paired samples t-test was additionally performed to assess systematic differences in overall perceived stress within each group using MHR, PSS-10, and STAI responses to stress during the TSST at PT-1 compared to PT. To determine if any correlation existed between recorded exercised heart rate and PSS-10 score a Pearson’s coefficient correlation was used. Data was collected in 2016 and 2017 and analyzed in March 2017 using SPSS 24 according to the per protocol analysis principle.
CHAPTER THREE: ANALYSIS

Demographics and baseline differences:

In total, 34 individuals were assessed for participation during a four-week recruitment. Twenty-seven individuals remained after inclusion/exclusion criteria and were equally distributed and randomized. Thirteen randomized to the HIIT group, and 14 randomized to the MICT group. Gender (8 men, 16 women) was equally distributed and randomized (HIIT n = 5; MICT n = 4). An independent-samples t-test was conducted to determine whether the following baseline characteristics were different across the two treatment groups: age, body mass index (BMI), resting HR (RHR), resting BP (RBP), Perceived Stress Scores (PSS-10), State anxiety and Trait anxiety from the Speilberger State Trait Anxiety Inventory (STAI), Maximum Heart Rate (MHR) in the Trier Social Stress Test (TSST). There was no significant difference across baseline measures when comparing HIIT protocols to MICT protocols. See Table 1.
Comparison of HIIT to MICT:

A mixed-design (repeated measures with a between subject factor) ANOVA was conducted to compare the PSS-10, STAI, and MHR pre and post-intervention measures from HIIT and MICT protocols. The main effect of PSS-10 was not significant $F(1,22) = 4.49$, n.s. Although MHR varied amongst groups (See fig. 6), there was no significant difference with the main effect of MHR $F(1,22) = .341$, n.s. The main effect of STAI, State/Trait was not significant $F(1,22) = .645$, n.s and $F(1,22) = 1.45$, n.s. respectively (See fig. 7-9). Ignoring all other variables, these effects elucidate that HIIT PSS-10, STAI, and MHR measures were not significantly different than MICT measures across both groups pre and post-exercise intervention. This suggests that HIIT is as effective as MICT in reducing perception of stress and HR response to acute stressful events. See Table 2.

Table 1. Baseline Characteristic Distribution. Data are expressed as mean with standard deviation.
<table>
<thead>
<tr>
<th>MEASURES</th>
<th>HIIT M ± SD</th>
<th>MICT M ± SD</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>MHR (bpm)</td>
<td>119.00 ± 18.92</td>
<td>115.25 ± 25.13</td>
<td>.341</td>
<td>.565</td>
</tr>
<tr>
<td>PSS-10</td>
<td>11.42 ± 4.38</td>
<td>12.58 ± 5.37</td>
<td>.117</td>
<td>.736</td>
</tr>
<tr>
<td>State</td>
<td>29.50 ± 6.30</td>
<td>30.08 ± 9.25</td>
<td>.645</td>
<td>.430</td>
</tr>
<tr>
<td>Trait</td>
<td>33.33 ± 5.19</td>
<td>34.08 ± 10.80</td>
<td>1.48</td>
<td>.242</td>
</tr>
</tbody>
</table>

Table 2. Comparison of HIIT and MICT to MHR, PSS-10, and State/Trait anxiety (STAI).

Fig. 6. HR responses to TSST at PT-1 and PT-2
Fig. 7. PSS-10 responses pre and post-intervention.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIIT</td>
<td>13</td>
<td>11.91</td>
</tr>
<tr>
<td>MICT</td>
<td>15.17</td>
<td>13.67</td>
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</table>

Fig. 8. STAI – State Anxiety Scores pre and post-intervention.

<table>
<thead>
<tr>
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<th>Pre</th>
<th>Post</th>
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</thead>
<tbody>
<tr>
<td>MICT</td>
<td>39.83</td>
<td>30.41</td>
</tr>
<tr>
<td>HIIT</td>
<td>35.83</td>
<td>29.5</td>
</tr>
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</table>
Effect of total exercise on PSS-10, MHR, and STAI measures

To determine if exercise alone, as an independent variable (3X week for 6 weeks), lowers MHR, PSS-10, and STAI measures from pre-intervention to post-intervention, a paired samples t-test was conducted on each measure. There was a significant difference in the pre intervention MHR, PSS-10, and STAI scores compared to post intervention scores suggesting that exercising 3x week for 6 week reduces perceived stress. See Table 3.
<table>
<thead>
<tr>
<th>MEASURES</th>
<th>Pre-intervention M ± SD</th>
<th>Post-intervention M ± SD</th>
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<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>MHR (bpm)</td>
<td>126.29 ± 22.55</td>
<td>116.63 ± 21.63</td>
<td>2.14</td>
<td>.043*</td>
</tr>
<tr>
<td>PSS-10</td>
<td>13.95 ± 4.98</td>
<td>12.00 ± 4.82</td>
<td>3.80</td>
<td>.001*</td>
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<tr>
<td>STAI - State</td>
<td>37.21 ± 10.51</td>
<td>29.79 ± 7.75</td>
<td>3.62</td>
<td>.001*</td>
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<tr>
<td>STAI - Trait</td>
<td>37.54 ± 9.93</td>
<td>33.71 ± 8.30</td>
<td>3.50</td>
<td>.002*</td>
</tr>
</tbody>
</table>

Table 3. Pre and post-intervention measures on exercising 3X week for 6 weeks. *denotes significance at < 0.05 level

**Effect of each group on PSS-10, MHR, and STAI measures:**

Additionally, a paired samples t-test was conducted on the HIIT group to determine if post PSS-10, MHR, and STAI measures were significantly different from pre measures. This set of analysis was repeated with the MICT group. There was a significant difference in the pre/post STAI - State scores between each groups (p < .05) suggesting that HIIT and MICT lowered State anxiety levels. However, HIIT did not have a significant difference on Trait scores, but MICT demonstrated significant differences in scores (p < .05) suggesting that MICT may lead to lower levels of trait anxiety compared to HIIT. There was no significant difference in pre MHR and post MHR measures in the MICT condition, but there was a significant difference in the HIIT condition (p < .05) suggesting that HIIT may blunt activation of the SAM system and MICT may have little impact and change on it. Additionally, there was no significant difference between pre and post PSS-10 scores in the HIIT condition, but there was significant difference between pre and post PSS-10 scores in the MICT condition suggesting that HIIT didn’t reduce over-all stress perception, but MICT was successful (p < .05) at reducing participant’s over-all perception of stress. See Table 4.1 & 4.2
HIIT Condition

<table>
<thead>
<tr>
<th>MEASURES</th>
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<th>Post-intervention M ± SD</th>
<th>t</th>
<th>p</th>
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<tbody>
<tr>
<td>MHR (bpm)</td>
<td>130.33 ± 16.13</td>
<td>119.00 ± 18.92</td>
<td>2.87</td>
<td>.015</td>
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<tr>
<td>PSS-10</td>
<td>13.17 ± 4.86</td>
<td>11.42 ± 4.86</td>
<td>1.88</td>
<td>.087</td>
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<tr>
<td>STAI - State</td>
<td>35.83 ± 7.60</td>
<td>29.50 ± 6.30</td>
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<tr>
<td>STAI - Trait</td>
<td>36.50 ± 6.00</td>
<td>33.33 ± 5.19</td>
<td>1.72</td>
<td>.113</td>
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</table>

Table 4.1. Effect of HIIT on MHR, PSS-10, and State/Trait anxiety STAI

MICT Condition

<table>
<thead>
<tr>
<th>MEASURES</th>
<th>Pre-intervention M ± SD</th>
<th>Post-intervention M ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>MHR (bpm)</td>
<td>122.25 ± 27.71</td>
<td>115.25 ± 25.13</td>
<td>.853</td>
<td>.412</td>
</tr>
<tr>
<td>PSS-10</td>
<td>14.75 ± 5.19</td>
<td>12.58 ± 5.37</td>
<td>4.42</td>
<td>.001</td>
</tr>
<tr>
<td>STAI - State</td>
<td>38.58 ± 13.00</td>
<td>30.08 ± 9.25</td>
<td>2.36</td>
<td>.038</td>
</tr>
<tr>
<td>STAI - Trait</td>
<td>38.58 ± 12.95</td>
<td>34.08 ± 10.80</td>
<td>3.62</td>
<td>.004</td>
</tr>
</tbody>
</table>

Table 4.2. Effect of MICT on MHR, PSS-10, and State/Trait anxiety (STAI).

Effect of HIIT and MICT on Systolic BP:

Mean Systolic BP (SBP) values were reported for each phase during the stress response assessment. Pre-intervention and post-intervention measurements of SBP were collected at identical times in the day. A paired samples t-test was conducted independently for HIIT and MICT to elucidate any significant differences of SBP within each group.

**HIIT**

A paired samples t-test was conducted to compare SBP values in pre-intervention and post intervention conditions. There was no significant difference in SBP measures from pre to post-intervention in any of the 5 measure-collecting periods (See Table 5.1) which would suggest that HIIT may not have an impact as previously believed on blunting SAM
system activation. In fact, an increase in reactivity was observed with the group post-intervention testing (See Fig. 10); however, this may be explained through the maintenance of cardiac output and total peripheral resistance, which is elucidated in greater detail in the discussion.

<table>
<thead>
<tr>
<th>MEASURES</th>
<th>Pre-intervention M ± SD</th>
<th>Post-intervention M ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>117.17 ± 15.56</td>
<td>120.42 ± 9.71</td>
<td>-.884</td>
<td>.396</td>
</tr>
<tr>
<td>Pre – TSST</td>
<td>129.58 ± 13.28</td>
<td>136.92 ± 23.70</td>
<td>-1.25</td>
<td>.236</td>
</tr>
<tr>
<td>Post – TSST</td>
<td>131.83 ± 15.63</td>
<td>128.00 ± 17.82</td>
<td>.665</td>
<td>.520</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>119.67 ± 9.75</td>
<td>124.83 ± 13.03</td>
<td>-1.72</td>
<td>.113</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>110.50 ± 13.85</td>
<td>114.17 ± 34.68</td>
<td>.566</td>
<td>.583</td>
</tr>
</tbody>
</table>

Table 5.1. Pre and post intervention HIIT SBP values.

Effect of HIIT on Systolic BP Measures

Fig. 10. HIIT SBP measures pre and post intervention for each measure-collecting period during stress response assessment.

MIC'T
A paired-samples t-test was conducted to compare SBP values in pre-intervention and post intervention conditions. There was no significant difference in SBP measures from pre to post-intervention in any of the 5 measure-collecting periods in the MICT group suggesting that MICT has similar impacts on the SAM system as does HIIT (See table 5.2). MICT SBP followed the same pattern as that seen of HIIT SBP. See fig. 11.

<table>
<thead>
<tr>
<th>MEASURES mmHg</th>
<th>Pre-intervention M ± SD</th>
<th>Post-intervention M ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>115.55 ± 12.16</td>
<td>144.45 ± 12.68</td>
<td>.395</td>
<td>.701</td>
</tr>
<tr>
<td>Pre – TSST</td>
<td>123.91 ± 11.56</td>
<td>132.18 ± 16.97</td>
<td>-1.85</td>
<td>.095</td>
</tr>
<tr>
<td>Post – TSST</td>
<td>124.36 ± 16.97</td>
<td>128.09 ± 14.90</td>
<td>-.854</td>
<td>.413</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>118.81 ± 16.03</td>
<td>122.09 ± 10.71</td>
<td>-.939</td>
<td>.370</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>116.55 ± 10.15</td>
<td>119.00 ± 15.29</td>
<td>-.536</td>
<td>.604</td>
</tr>
</tbody>
</table>

Table 5.2. Pre and post-intervention MICT SBP values.

Fig. 11. MICT SBP measures pre and post intervention for each measure-collecting period during stress response assessment.
**HIIT and MICT**

A mixed-design ANOVA with repeated measures was performed to compare mean HIIT and MICT Systolic BP measures across the 5 measure collecting periods within the stress response assessment. Additionally, when comparing SBP measures for both groups there was no significant difference in HIIT and MICT SBP measures across all measure-collecting periods (see table 5.3) suggesting that HIIT exercise has a similar impact on blunting the SAM system response to stress that MICT demonstrates. As can be seen in Fig. 12, mean SBP for both groups matched nearly identical with exception to post TSST measurements.

<table>
<thead>
<tr>
<th>MEASURES mmHg</th>
<th>MEAN ± SD HIIT</th>
<th>MEAN ± SD MICT</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>119.45 ± 9.56</td>
<td>116.15 ± 12.49</td>
<td>.716</td>
<td>.482</td>
</tr>
<tr>
<td>Pre-TSST</td>
<td>135.09 ± 23.96</td>
<td>133.92 ± 17.83</td>
<td>.137</td>
<td>.892</td>
</tr>
<tr>
<td>Post-TSST</td>
<td>125.55 ± 16.42</td>
<td>131.85 ± 16.43</td>
<td>.936</td>
<td>.359</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>123.00 ± 11.94</td>
<td>124.46 ± 11.77</td>
<td>.301</td>
<td>.766</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>117.91 ± 13.33</td>
<td>120.46 ± 14.82</td>
<td>.440</td>
<td>.664</td>
</tr>
</tbody>
</table>

Table 5.3. Mean HIIT and MICT SBP measures.
Fig. 12. HIIT SBP compared to MICT SBP across all 5 measure-collecting periods during stress response assessment.

**Effect of HIIT and MICT on HR:**

Mean HR values were reported for each phase during the stress response assessment. Pre-intervention and post-intervention measurements of HR were collected at identical times in the day. A paired samples t-test was conducted independently for HIIT and MICT to elucidate any significant differences of HR within each group, and a mixed-design ANOVA with repeated measures was performed to compare mean HIIT and MICT Systolic HR measures across the 6 measure collecting periods within the stress response assessment.

**HIIT**

A paired-samples t-test was conducted to compare HR values in pre-intervention and post intervention conditions. There was only significant difference in Pre – TSST and TSST
HR measures from pre to post-intervention, and Rest, Post – TSST, +10 min Post, and +20 min Post were not significantly different in the HIIT (See table 6.1) suggesting that HIIT may offer SAM axis activation blunting during psychosocial stressors. Fig. 13 illustrates mean HIIT HR values over time.

<table>
<thead>
<tr>
<th>MEASURES (bpm)</th>
<th>Pre-intervention M ± SD</th>
<th>Post-intervention M ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>71.00 ± 12.71</td>
<td>71.17 ± 10.95</td>
<td>-.053</td>
<td>.959</td>
</tr>
<tr>
<td>Pre – TSST</td>
<td>77.83 ± 13.20</td>
<td>86.50 ± 16.76</td>
<td>-2.59</td>
<td>.025*</td>
</tr>
<tr>
<td>TSST</td>
<td>130.33 ± 16.13</td>
<td>118.00 ± 18.53</td>
<td>3.06</td>
<td>.011*</td>
</tr>
<tr>
<td>Post – TSST</td>
<td>78.33 ± 14.01</td>
<td>79.75 ± 15.41</td>
<td>-.334</td>
<td>.745</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>73.91 ± 14.85</td>
<td>74.92 ± 11.50</td>
<td>-.209</td>
<td>.839</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>73.33 ± 14.92</td>
<td>69.83 ± 9.01</td>
<td>.853</td>
<td>.412</td>
</tr>
</tbody>
</table>

Table 6.1. Pre and post-intervention HIIT HR values.

![Effect of HIIT on HR Measures](image)

**Effect of HIIT on HR Measures**

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Pre - TSST</th>
<th>TSST</th>
<th>Post - TSST</th>
<th>Recovery 10</th>
<th>Recovery 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre intervention</td>
<td>71</td>
<td>77.83</td>
<td>130.33</td>
<td>78.33</td>
<td>73.91</td>
<td>73.33</td>
</tr>
<tr>
<td>Post intervention</td>
<td>71.17</td>
<td>86.5</td>
<td>118</td>
<td>79.75</td>
<td>74.92</td>
<td>69.83</td>
</tr>
</tbody>
</table>

Fig. 13. HIIT HR measures pre and post intervention for each measure-collecting period during stress response assessment.
A paired-samples t-test was conducted to compare HR values in pre-intervention and post-intervention conditions. There was no significant difference in HR measures from pre to post-intervention in any of the 6 measure-collecting periods in the MICT group suggesting that MICT has similar minimal if any impact at reducing SAM system activation during stress (See table 6.2). In fact, MICT HR followed the same pattern as that seen of HIIT HR. See fig. 14.

<table>
<thead>
<tr>
<th>MEASURES (bpm)</th>
<th>Pre-intervention M ± SD</th>
<th>Post-intervention M ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>71.83 ± 11.99</td>
<td>75.33 ± 12.47</td>
<td>-1.09</td>
<td>.298</td>
</tr>
<tr>
<td>Pre – TSST</td>
<td>76.67 ± 19.2</td>
<td>80.42 ± 19.26</td>
<td>-.605</td>
<td>.558</td>
</tr>
<tr>
<td>TSST</td>
<td>122.25 ± 27.71</td>
<td>115.25 ± 25.13</td>
<td>.853</td>
<td>.412</td>
</tr>
<tr>
<td>Post – TSST</td>
<td>87.00 ± 26.45</td>
<td>83.33 ± 14.73</td>
<td>.567</td>
<td>.582</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>71.46 ± 15.37</td>
<td>75.25 ± 10.77</td>
<td>-1.15</td>
<td>.275</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>74.17 ± 18.03</td>
<td>73.92 ± 12.42</td>
<td>.053</td>
<td>.958</td>
</tr>
</tbody>
</table>

Table 6.2. Pre and post-intervention MICT HR measures
HIIT AND MICT

A mixed-design ANOVA with repeated measures was performed to compare mean HIIT and MICT Systolic HR measures across the 6 measure collecting periods within the stress response assessment. Comparing HIIT and MICT, there was not a significant main effect of HR across all 6 measure collecting periods demonstrating that HIIT HR was similar to MICT HR during the stress response assessment. Additional values for each period can be found in Table 6.3. Fig. 15 illustrates that difference between groups at all measure collecting periods.
### Table 6.3: HIIT HR compared to MICT HR across all 6 measure collecting periods during stress response assessment.

<table>
<thead>
<tr>
<th>MEASURES (bpm)</th>
<th>MEAN ± SD HIIT</th>
<th>MEAN ± SD MICT</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>71.17 ± 10.95</td>
<td>75.33 ± 12.47</td>
<td>.551</td>
<td>.466</td>
</tr>
<tr>
<td>Pre – TSST</td>
<td>86.5 ± 16.76</td>
<td>80.42 ± 19.26</td>
<td>3.176</td>
<td>.088</td>
</tr>
<tr>
<td>TSST</td>
<td>118.00 ± 18.53</td>
<td>115.25 ± 25.13</td>
<td>.341</td>
<td>.565</td>
</tr>
<tr>
<td>Post – TSST</td>
<td>79.75 ± 15.41</td>
<td>83.33 ± 14.73</td>
<td>.432</td>
<td>.518</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>74.92 ± 11.50</td>
<td>75.25 ± 10.77</td>
<td>.235</td>
<td>.632</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>69.83 ± 9.01</td>
<td>73.92 ± 12.42</td>
<td>.272</td>
<td>.607</td>
</tr>
</tbody>
</table>

**Effect of HIIT and MICT on SC:**

Mean SC values were reported for each phase during the stress response assessment excluding the pre-TSST phase. Pre-intervention and post-intervention measurements of SC were collected at identical times in the day for each participant. A paired samples t-test was conducted independently for HIIT and MICT to elucidate any significant
differences of SC within each group, additionally, a paired sample t-test was conducted for each sampling period using exercise alone as an independent variable to compare pre and post intervention measures. A mixed-design ANOVA with repeated measures was performed to compare mean HIIT and MICT SC measures across the 4 measure collecting periods pre and post intervention within the stress response assessment.

**COMBINED HIIT AND MICT**

A paired samples t-test was conducted at each collection period to elucidate difference between pre intervention SC levels and post intervention SC levels while participants underwent the stress response assessment. There was significant difference (p < .001) in SC in each of the 4 periods sampling periods (See table 7.1) that were taken suggesting that exercise at or above 80% MHR is efficacious at blunting HPA reactivity to standardized psychosocial laboratory stressors. See fig 16.

<table>
<thead>
<tr>
<th>MEASURES</th>
<th>Pre-intervention M ± SD</th>
<th>Post-intervention M ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>µg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>1.57 ± .611</td>
<td>.839 ± .435</td>
<td>6.73</td>
<td>&lt; .0001</td>
</tr>
<tr>
<td>Post – TSST</td>
<td>1.39 ± .493</td>
<td>.832 ± .385</td>
<td>6.51</td>
<td>&lt; .0001</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>1.56 ± .585</td>
<td>.991 ± .630</td>
<td>3.52</td>
<td>.002</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>1.34 ± .470</td>
<td>.822 ± .393</td>
<td></td>
<td>&lt; .0001</td>
</tr>
</tbody>
</table>

Table 7.1. Pre and post-intervention SC measures with exercise at or above 80% MHR as an independent variable.
A paired samples t-test was conducted to compare SC values in pre-intervention and post intervention conditions (See Table 7.2). There was a significant difference \((p < .005)\) in all 4 measure-collecting periods. Fig. 17 illustrates SC measures over time for pre and post intervention during the stress response assessment.

**Table 7.2.** Pre and post-intervention HIIT SC measures.

<table>
<thead>
<tr>
<th>MEASURES</th>
<th>µg/dl</th>
<th>Pre-intervention (M \pm SD)</th>
<th>Post-intervention (M \pm SD)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>1.46 ± .439</td>
<td>.693 ± .367</td>
<td>6.03</td>
<td>&lt; .0001</td>
<td></td>
</tr>
<tr>
<td>Post – TSST</td>
<td>1.21 ± .463</td>
<td>.675 ± .249</td>
<td>5.62</td>
<td>&lt; .0001</td>
<td></td>
</tr>
<tr>
<td>+10 min Post</td>
<td>1.42 ± .541</td>
<td>.838 ± .331</td>
<td>3.54</td>
<td>.005</td>
<td></td>
</tr>
<tr>
<td>+20 min Post</td>
<td>1.31 ± .503</td>
<td>.829 ± .359</td>
<td>3.51</td>
<td>.005</td>
<td></td>
</tr>
</tbody>
</table>
A paired-samples t-test was conducted to compare SC values in pre-intervention and post intervention conditions. There was significant difference in SC measures from pre to post-intervention in all but the measurement taken 10 minutes after the TSST (+10 min Post) in the MICT group suggesting that MICT has similar impacts on the HPA axis as does HIIT (See table 7.3). Fig. 18. demonstrates a mean SC comparison over time.

### Table 7.3. Pre and post intervention MICT SC measures.

<table>
<thead>
<tr>
<th>MEASURES</th>
<th>Pre-intervention M ± SD</th>
<th>Post-intervention M ± SD</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>1.68 ± .749</td>
<td>.985 ± .464</td>
<td>3.83</td>
<td>.003</td>
</tr>
<tr>
<td>Post – TSST</td>
<td>1.56 ± .472</td>
<td>.989 ± .440</td>
<td>3.95</td>
<td>.002</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>1.70 ± .616</td>
<td>1.14 ± 8.18</td>
<td>1.93</td>
<td>.079</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>1.37 ± .453</td>
<td>.815 ± .439</td>
<td>3.56</td>
<td>.004</td>
</tr>
</tbody>
</table>

**Fig. 17.** HIIT SC measures in µg/dl pre and post intervention for each measure-collecting period during stress response assessment.

**Fig. 18.** demonstrates a mean SC comparison over time.
Fig. 18. MICT SC measures in µg/dl pre and post intervention for each measure-collecting period during stress response assessment.

**HIIT AND MICT**

Mean salivary cortisol levels (µg/gl) are presented in Table 7.4. A mixed-design ANOVA with repeated measures was performed to compare mean HIIT and MICT Systolic HR measures across the 4 measure collecting periods within the stress response assessment. Comparing HIIT and MICT, there was no significant difference in cortisol levels between the two groups for each of the 4 measure collecting periods, neither Rest, $F(1,22) = .095, \text{MSE} = .014, p = .761$; Post TSST, $F(1,22) = .065, \text{MSE} = .006, p = .802$; + 10 min from Post, $F(1,22) = .005, \text{MSE} = .002, p = .945$; +20 min from Post, $F(1,22) = .155, \text{MSE} = .020, p = .698$. Fig. 19. depicts the mean SC over time.

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Post - TSST</th>
<th>Recovery1</th>
<th>Recovery2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre intervention</td>
<td>1.68</td>
<td>1.56</td>
<td>1.7</td>
<td>1.37</td>
</tr>
<tr>
<td>Post intervention</td>
<td>0.985</td>
<td>0.989</td>
<td>1.14</td>
<td>0.815</td>
</tr>
</tbody>
</table>

**Effect of MICT on SC Measures**
<table>
<thead>
<tr>
<th>MEASURES µg/dl</th>
<th>MEAN ± SD HIIT</th>
<th>MEAN ± SD MICT</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>.693 ± .367</td>
<td>.985 ± .464</td>
<td>.095</td>
<td>.761</td>
</tr>
<tr>
<td>Post – TSST</td>
<td>.675 ± .249</td>
<td>.989 ± .440</td>
<td>.065</td>
<td>.802</td>
</tr>
<tr>
<td>+10 min Post</td>
<td>.838 ± .331</td>
<td>1.14 ± 8.18</td>
<td>.005</td>
<td>.945</td>
</tr>
<tr>
<td>+20 min Post</td>
<td>.829 ± .359</td>
<td>.815 ± .439</td>
<td>.155</td>
<td>.698</td>
</tr>
</tbody>
</table>

Table 7.4. HIIT SC compared to MICT SC across 4 measure-collecting periods during stress response assessment.

![HIIT vs MICT SC Measures](image)

Fig. 19. HIIT SC compared to MICT SC measures in µg/dl across 4 measure-collecting periods during stress response assessment.

**HR intensity and perceived stress correlation:**

A Pearson’s product-moment correlation coefficient was computed to elucidate the relationship between the intensity of HR during exercise and the perception of stress by means of self-reported psychological measures (PSS-10 and STAI). There was no correlation between HR and self-reported psychological measures (PSS-10; r(22) = -.339,
demonstrating that beyond 80% MHR, increased HR intensity does not appear to foster superior stress-buffering and emotional salutogenic properties.

CHAPTER FOUR: DISCUSSION

This is the first study to investigate the effects that the intensity of an exercise program has on the HPA and SAM axis response and self-reported perception of stress/anxiety to a validated laboratory stress procedure using a randomized control trial. The key findings of the present study is that (1) higher than normal levels of perceived stress are positively associated with elevated salivary cortisol (SC) and catecholamine responses [heart rate (HR) and systolic blood pressure (SBP)] associated with the hyper-activation of the physiological stress response through the HPA and SAM axis of the sympathetic nervous system during the exposure of a standardize laboratory stressor (TSST), and that this psychological and physiological response is blunted with only 18 sessions of exercise over a period of 6 weeks (3 session/wk.).

It was also shown that (2) exercise intensities above the threshold of 80% MHR sustained did not lead to correlated reductions in physiologic response in terms of HR, SBP, and SC nor mitigated psychological perception in terms of the PSS-10 and STAI when exposed to a standardized laboratory stressor. Additional findings demonstrated that (3) the laboratory stressor utilized (TSST) was efficacious in inducing a stress response in all participants in PT-1 as well as in PT-2 and that (4) exercise alone as its own independent variable lead to significant differences in PSS-10, STAI, MHR measures at PT-2 when
compared to PT-1; however there was no significant difference in SBP within and between groups.

In line with previous finding (Klaperski et al., 2014; Federenko et al., 2004), post-intervention cortisol responses during PT-2 were reduced compared to pre-intervention cortisol responses during PT-1; however, in contrast to (Klaperski et al., 2014) HR response remained constant with the MICT group, but noticeably decreased with the HIIT group. The above findings should be regarded with caution due to limitations, but in the following, the key findings are discussed in greater detail. The cross stressor adaptation hypothesis suggests that regular exercise leads to neurobiological adaptations in the stress response systems that promote decreased physiological responses to similar physiological stressor, but as well to psychological stressors (Michael, 1957).

Since post-interventions PSS-10 scores were equal, there is no clear statistical evidence that either intensity (HIIT nor MICT) is preferable or superior at reducing perceived stress with the sample size (n=24). However, across the population, exercising three times a week did have an impact on lowering perceived stress with MICT training clearly demonstrating superior outcomes on stress perception.

**Perceived Stress Scale – 10 item:**

This is the first study that uses information from Cohen’s Perceived Stress Scale - 10 item (PSS-10) to measure perception of stress in individuals engaging in a prolonged HIIT exercise compared MICT exercise routine over time. Data from the study confirms a correlation between physical exercise and lower perceptions of stress as reported on the PSS-10 the most widely used psychological instrument used to measure the perception of
stress (Gerber et al., 2013; Cohen et al., 1994). Administration at the very beginning of
the stress response assessment at PT-1 and PT-2 before resting and any tasks allowed for
the clearest and apparent perception of the participant since anticipatory stress was at its
lowest. Overall, evidence could be found that perception of stress significantly changed
over time between exercise groups. While exercise (above the threshold of 80% MHR)
was significantly different from pre intervention measures to post intervention measures,
the HIIT group did not experience a significance difference within group measures;
however the difference between the two time periods did approach significance. There
was a significant difference in PSS-10 scores within the MICT group over time. The
conclusion of this observation supports previous research (Oaten and Cheng, 2006;
Puterman et al., 2010) that exercise lowers stress perception. This study demonstrated
further that MICT compared to HIIT regimens might offer better stress-buffering
capacity to lower these perceptions of stress.

**Speilberger State Trait Anxiety Inventory:**

The Speilberger State Trait Anxiety Inventory (STAI) was categorized into its two
divisions being state anxiety and trait anxiety measures.

**State**

Data from this study confirms a link between physical exercise and state anxiety scoring
responses to stress. State anxiety, anxiety based on an immediate and/or acute
circumstance or condition, was significantly different within group comparison and in
comparison of exercise (above 80% MHR) as an independent variable. There was no
significant difference between groups comparison suggesting that either intensity of
exercise was equally efficacious at promoting a reduction in state anxiety from the administration of an acute stressor. In support of previous studies (Steptoe et al., 1989; Brown et al., 1995), this study suggests that physical exercise leads to significant differences in state anxiety scores over time. Furthermore, the data contributes to previous work using state anxiety scoring to measure enjoyment of higher intensity exercise (Kilpatrick et al., 2015; Whitney A., 2016) and elucidating that high intensity exercise also leads to stress tolerance in conjunction with high enjoyment scores. In summation, the results for state anxiety scores supports that no difference exists between groups, but exercise at or above 80% of MHR reduce state anxiety over time.

**Trait**

The results from the study suggest that exercise may have the capacity to effect trait anxiety levels in addition to state anxiety. This observation was in contrast to initial assumptions that trait dispositions would not be modified by exercise. The neurobiological adaptations that blunt HPA and SAM axis activation proposed from cross stress adaptation may also impact an individual’s overall trait for anxiety and resiliency to stress. As trait anxiety scores relate to an individual’s enduring characteristic and relative disposition to feel stress, worry, and discomfort (Spielberger et al., 1994), it was not predicted to observe significance over time in comparisons within groups and between groups. Considering exercise (at or above 80% MHR) as its own independent variable, there was an observed significant difference in trait anxiety scores at PT-2 when compared to PT-1. Within subject comparison elucidated that individuals of the MICT group significantly reduced trait anxiety while individuals of the HIIT group did not. As expected, there was no significant difference between MICT when compared to HIIT.
This observation requires further investigations as it has typically been noted that relative dispositions and characteristics of anxiety are products of parsimonious-based thought patterns created by belief systems (Himle et al., 1982; McNally, 1989; Paulus M. and Angela J, 2012; Fletcher and Sarkar, 2016); however, it appears that blunted neuroendocrine and sympathoadreno reactivity may support the alteration of perception to a degree that imparts the ability to effect internal dialogue and remodeling of thoughts that lead to belief systems and ultimately resiliency to stress and anxiety through mental fortitude. Due to the nature of measurement for both PSS-1- and STAI being self-reporting, an accurate and quantitative inference is difficult to make between the PSS-10 and STAI and the cross stressor adaptation hypothesis. Nonetheless, it was clear that a significant difference in perception was observed from pre intervention to post intervention within groups suggesting that the mechanism may be related to cross stressor adaptation. Furthermore, it is also clear that one group was not superior over another in altering perception given that there was no significant difference in both PSS-10 and both facets of the STAI when comparing HIIT and MICT post intervention scores and pre intervention scores suggesting that both intensities of exercise size have equal stress-blunting capacity related to perception of stress as reported by the PSS-10 and STAI.

**Maximum Heart Rate:**

The study confirms the link that exists between exercise and heart rate response to stress. As an independent variable, exercise (at or above 80% MHR) contributed to a significant difference in MHR during TSST from PT-1 to PT-2. There was no significant difference in MHR between groups suggesting that HIIT and MICT may have equal capacity at
blunting the SAM axis during stress. In further support, MHR reactivity and recovery patterns from stress were similar compared between groups and compared within groups; however, HR was significantly different within the HIIT groups when comparing PT-1 to PT-2 but not significantly different within the MICT group when comparing PT-1 to PT-2 suggesting that HIIT contributed to significant lower MHR measures during the TSST over time and MICT did not. These findings support cross stressor adaptations by offering evidence that additional physiological stress by means of high intensity levels of exercise may lead to superior reactivity to heterolytic stressors. It should be noted that over time HR values taken at each measure collecting period were shown to have significant difference only during the pre-TSST and TSST measures for the HIIT group. All other periods were not significantly different. Therefore, if cross stressor adaptation is the cause of this observation it is only valid during anticipatory stress and stress inoculation. In contrast, MICT group participant’s measures across all measure collecting periods were not significantly different but appeared to be approaching significance during TSST measurement suggesting this intensity of exercise may possess stress-buffering capacity during stress inoculation similar to that of HIIT. Participant’s general health and fitness status may have contributed to the observations noted in MICT. Participants were non-sedentary and generally healthy individuals. Training effect may not have occurred at levels of exercise not exceeding participant’s pre-study exercise regimens for MICT group participants thus supporting the argument that cross stressor adaptation should lead to superior results in stress resiliency.
Systolic Blood Pressure:

Catecholaminergic vasoconstriction is a typical physiological change from the sympathetic nervous system that follows stress inoculation (Knight et al., 2001; Kvetnansky, 2012). In contrast to the belief that exercise would reduce systolic blood pressure during the post intervention stress response assessment (Georgaides et al., 2000), an increase in systolic blood pressure was observed in both groups. In comparisons between groups, differences in values where minimal and demonstrated no significant difference. The largest difference occurred post TSST, and was also not significant. This observation may be explained to relating directly to the observed cardiac changes in both groups.

Heart rate response significantly decreased in pre-TSST and TSST measurements from participants of the HIIT group. Though no TSST SBPs were taken, PT-2 pre-TSST SBP was measured and found to be elevated to a corresponding muted HR response. Arterial blood pressure is a product of Cardiac output and peripheral vascular resistance. Cardiac output is based on heart rate and stroke volume (Porth, 2011). The observations of heightened SBP can be explained by the mechanism of cardiac output maintenance. Since cardiac output must be maintained, a reduction of HR would solicit an increase in blood pressure. This can explain the observations for the two time periods where HR was significantly decrease during the second stress response assessment and the corresponding increase in heart rate that was observed.

Nevertheless, though not significantly different, it does not explain the increase that was observed with the MICT group and is in not in line with the majority of the existing experimental studies in which participation in an exercise program improved blood
pressure reactivity and recovery from stress (Blumenthal et al., 1990; Georgaides et al.,
2000; Hamer et al., 2006). All in all, the data from this study does not support the cross
stressor adaptation hypothesis as it relates to blood pressure suggesting that physical
exercise may not have the capacity to blunt reactivity of the SAM axis to stress.

**Salivary Cortisol:**

The findings experimentally confirm what had been hypothesized in other studies
(Salmon P., 2001; Rimmerle et al., 2009; Klaperski et al., 2014) in that exercise is
efficacious at blunting HPA axis reactivity to stress by measure of salivary cortisol.
Though not intended as a specific aim of this study, this randomized control study was
able to significantly reduce salivary cortisol stress reactivity in 6 weeks of exercise
training compared to the previous study’s 12 weeks of exercise inoculation. In a
comparison of the effects of the exercise training intensity, I discovered that exercises of
high intensity do not have any clear advantage over moderate intensity exercise (above
MHR 80%) in terms of cortisol reactivity. Participants of the moderate intensity group
significantly reduced their cortisol reactivity to stress, as did participants from the high
intensity group. The groups did not differ regarding change in cortisol reactivity over the
study period; however, HIIT group demonstrated greater recovery capacity when
compared to MICT and a difference in cortisol levels between groups existed that was not
significant, but observable.

Given these observations, my findings support the assumptions that the neuroendocrine
response to stress may be mediated by cross stressor adaptations, in agreement with
previous studies (Salmon P., 2001; Rimmele et al., 2009; Klaperski et al., 2014).
Contributing to these studies (Gerber et al., 2008) reported vigorous physical activity reduces adrenocortical reactivity to stress. Though this study did not demonstrate a significant difference between the high intensity group compared to the moderate intensity group, a non-significant trend was observed towards this outcome.

**Strengths and limitations:**

It can be assumed that the findings from this study have a high internal validity. This is because of X features within the methodology: 1) A randomized, controlled intervention design was used with each participant acting as their own control; 2) A validated and standardized procedure was applied to induce stress in a laboratory environment with the use of the TSST; 3) In response to this stressor, three physiological measures (HR, SBP, SC) were utilized to indicate the sympathetic reactivity and response of the HPA and SAM axis; 4) Actualized HR thresholds were determined by use of cardiopulmonary exercise testing by means of a VO$_{2\text{MAX}}$ test and utilized these measures to confirm appropriate exercise intensity; 5) The reactivity and the recovery from stress was evaluated. These attributes reinforce the assumption that my findings demonstrate a positive correlation between exercise and the body’s physiological response to stress, in particular as it relates to the role that intensity of exercise plays in blunting the of this stress response. However, one limitation that deserves greater investigation and emphasis is the individual responses to the TSST. Clear differences were observed within participants on psychosocial responses to stress that translated to observed agitation and emotional discomfort, increased measures within physiological stress responses parameters, and elevated PSS-10 and STAI scores. A superior method of
accounting for individuals with high sensitivity and low stress resiliency is to categorize each into a separate group post intervention. Not only would this offer a more complete understanding of the effects of exercise on global stress perception, but it would also elucidate correlations that exist between exercise and stress response for individuals who are not classified or have been classified with a clinical variant of anxiety/stress but have low stress resiliency and/or coping skills.

Group size was an additional limitation to the study. While the study offered significant evidence demonstrating that exercise reduces global perception of stress and associated physiological responses, an observed trend existed in the change from pre to post intervention between the HIIT group compared to the MICT group in physiological parameters, but not psychological parameters. This trend suggests that HIIT may possess a more robust capacity to reduce the associated physiological response to stress, but may not change an individual’s perception of the intensity of the related stress. A larger sample size would be needed to help elucidate this observation.

Salivary cortisol increases within 10 minutes of a stressor and peaks within 20 minutes (Van Eck et al., 1996). Cortisol samples were taken directly following the TSST, again at 10 minutes, and lastly at an additional 20 minutes, thus allowing sampling to occur 10 minutes and 30 minutes post TSST. It was suspected that this time frame would offer sufficient time to explicate a recovery curve associated with the cessation of the TSST. Additional sampling periods at 20 minutes, 40 minutes, and 50 minutes post TSST should have been added to create a more robust and coherent recovery curve.
Outlook:

Health outcomes are negatively affected by chronic hyper-elevations of the HPA and SAM axis and the proceeding recovery from activation (Chrousos, 2009). This study has demonstrated that engaging in regular physical exercise appears to attenuate the activation of the sympathetic nervous system during stress inoculation and reduces the HPA and SAM axis reactivity to a stress. Given the rising epidemic of sedentarism in America (U.S. Department of Health and Human Services, 2012) it is increasingly important for professionals in the medical fitness industry to identify modalities of exercise that are sustainable by effectively including enjoyable and time efficiency perspectives that ultimately lead in the manifestation of significant health benefits. Despite empirical evidence showing significant adaptations and potentially higher enjoyment in response to HIIT compared to MICT (Kilpatrick et al., 2015; Whitney A., 2016), acceptance of high intensity interval training is not universal (Thum et al., 2017). It is a fast growing trend that may well become a normative option because of return on time investment. Although, this study did not empirically demonstrate that HIIT is superior compared to MICT at blunting HPA and SAM axis activation upon psychosocial stressors, it is clear that exercise above the threshold of 80% MHR does significantly reduced stress perception and neuroendocrine and sympoandroreno activation with as little as 18 bouts of exercise over a period of 6 weeks. This study has also demonstrated that this response can be accomplished in 19 minutes of activity compared to 30 minutes of activity lending to an alternative form of exercise that offers similar stress-buffering benefits in 37% less time than traditional moderate intensity forms of aerobic exercise. It should be noted that large amounts of HIIT have been associated with hyper-activation of the HPA axis.
(Buono et al., 1986; McGuigan et al., 2004), a condition that is commonly associated with comorbidities including metabolic syndrome (Siddiqui et al., 2015), obesity (Vicennati et al., 2014), cardiovascular disease (Kaplan et al., 1996), memory dysfunction (Sapolsky R., 1992) and depression and anxiety (Mackin and Young, 2004). Care must be afforded that an attempt to reduce reactivity to stress does not lead to alternative negative health outcomes. Whether cross stressor adaptation forms the basis of the observations noted in the study or whether the stress-buffering capacity is a by-product of other changes that are related to exercise induced health should be further investigated.
REFERENCES


34. Centre for Studies on Human Stress; (2015) Fernand-Seguin Research Centre of Louis-H. Lafontaine Hospital, Quebec, Canada


APPENDIX A

PAR-Q

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

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1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

2. Do you feel pain in your chest when you do physical activity?

3. In the past month, have you had chest pain when you were not doing physical activity?

4. Do you lose your balance because of dizziness or do you ever lose consciousness?

5. Do you have a bone or joint problem (example, back, knee or hip) that could be made worse by a change in your physical activity?

6. Is your doctor currently prescribing drugs (example, water pills) for your blood pressure or heart condition?

7. Do you know of any other reason why you should not do physical activity?

If you answered YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.

- Take part in a fitness appraisal — this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- If you are feeling well because of a temporary illness such as a cold or a fever — wait until you feel better or
- If you are or may be pregnant — talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME ____________________________

SIGNATURE ________________________

DATE ____________________________

SIGNATURE OF PARENT or GUARDIAN (for persons under the age of majority)

WITNESS _________________________

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.
### HEALTH HISTORY QUESTIONNAIRE—Please Print

#### PERSONAL INFORMATION
- **Name:**
- **DOB:**
- **Gender:** M F
- **Address:**
- **City:**
- **State:**
- **Zip Code:**
- **Place of Employment:**
- **Work Phone:**
- **Cell Phone:**
- **Marital Status (circle one):** Single Married Divorced Widowed
- **Name of Personal Contact:**
- **Phone:**
- **Education (circle highest level):** Elementary High School College Graduate

#### PHYSICIAN INFORMATION
- **Primary Care Physician:**
- **Phone:**
- **Other Physicians:**
- **Phone:**
- **Reason for Last Visit:**
- **Date of Last Physical:**
- **Location:**

#### DRUG, MEDICATION, AND DIETARY SUPPLEMENT LIST

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<th>Name</th>
<th>Dose and Frequency</th>
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#### PERSONAL HEALTH HISTORY

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<th>Condition</th>
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<td>High Blood Pressure</td>
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<td>High Cholesterol</td>
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<td>Abnormal Electrocardiogram (ECG)</td>
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<td>Blood Clots or Thrombophlebitis</td>
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<td>Blood Disorders / Problems</td>
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<td>Anemia</td>
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<td>Heart Valve Problems / Murmur</td>
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<td>Rheumatic Fever</td>
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<td>Heart Disease or Heart Attack</td>
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<td>Vascular Problems (PVD)</td>
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<td>Varicose Veins</td>
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<td>Lung Disease (emphysema, COPD)</td>
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<td>Asthma</td>
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<td>Pulmonary Hypertension</td>
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<td>Seizures / Epilepsy</td>
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<td>Sinus / &quot;Mini&quot; Bruker</td>
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<td>Liver Problems / Hepatitis</td>
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<td>Diabetes</td>
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<td>Thyroid Problems</td>
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<td>Cancer</td>
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<td>Lymphedema</td>
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<td>Allergies</td>
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<td>Skin Problems</td>
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<td>Arthritis</td>
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<td>Gout</td>
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<td>Osteopenia/Osteoporosis</td>
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<td>Low Back Pain</td>
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<td>Kidney Disease Or Stones</td>
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<td>Gall Bladder Problems</td>
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<td>Pancreatitis</td>
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<td>Collitis</td>
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<tr>
<td>Irritable Bowel Syndrome</td>
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<td>Bladder Infections</td>
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<td>Eating Disorders</td>
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<td>Polio</td>
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<td>Migraines / frequent headaches</td>
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<td>Anxiety/Depression</td>
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<td>Fatiguing Spells</td>
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<td>Concussion</td>
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<td>Vertigo</td>
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<td>Alcohol/Drug Abuse</td>
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<td>Vision Problems (Glaucoma)</td>
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<td>Pregnancy</td>
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<td>Other</td>
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Health History Questionnaire
**FAMILY HISTORY:** If any of your immediate family (parents, siblings) has had any of the conditions listed under the previous section, please list here.

1. 
2. 
3. 
4. 
5. 
6. 

**OPERATIONS OR HOSPITALIZATIONS**

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**CURRENT TREATMENT(S)**

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**ALLERGIES**

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**RISK FACTOR ASSESSMENT** Please check any statements that are true for you.

- Your systolic blood pressure is $\geq 140$ mm Hg and diastolic blood pressure is $\geq 90$ mm Hg.
- Your total cholesterol is $> 200$ mg/dL OR you don’t know your cholesterol numbers.
- Your HDL cholesterol is $< 40$ mg/dL if you are a male or $< 50$ mg/dL if you are a female.
- Your triglycerides are $> 150$ mg/dL.
- You fasting blood glucose has been $> 100$ mg/dL or a physician told you that you are pre-diabetic.
- You have a close relative who had a heart attack or heart surgery before age 55 (father or brother) or age 65 (mother or sister).
- Your waist $> 40$ inches if you are a male or 35 inches if you are a female.
- You smoke or quit smoking within the last 6 months or you are exposed to environmental tobacco smoke.
- You do not participate in at least 30 minutes of moderate physical activity on at least three days per week for at least three months.
- You are $> 20$ pounds overweight.

**SIGNS OR SYMPTOMS** Please check yes if you have experienced:

- Shortness of breath at rest or with usual activities
- Dizziness or fainting during exercise
- Burning, squeezing or heaviness in your, chest, jaw, arms, back, especially during exertion
- Rapid or irregular heart beats
- Swelling of ankles or hands
- Unusual fatigue with usual activities
- Pain or cramping in calves, thighs, or buttocks during physical activity
- Recurrent vomiting

Signature: ___________________________ Date: ___________________________
APPENDIX C

Perceived Stress Scale

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate by circling how often you felt or thought a certain way.

Name ____________________________ Date ____________
Age ________ Gender (Circle): M F Other ________________________________

0 = Never  1 = Almost Never  2 = Sometimes  3 = Fairly Often  4 = Very Often

1. In the last month, how often have you been upset because of something that happened unexpectedly? .............................. 0 1 2 3 4
2. In the last month, how often have you felt that you were unable to control the important things in your life? .............................. 0 1 2 3 4
3. In the last month, how often have you felt nervous and "stressed"? ........... 0 1 2 3 4
4. In the last month, how often have you felt confident about your ability to handle your personal problems? .............................. 0 1 2 3 4
5. In the last month, how often have you felt that things were going your way? ................................................................. 0 1 2 3 4
6. In the last month, how often have you found that you could not cope with all the things that you had to do? .............................. 0 1 2 3 4
7. In the last month, how often have you been able to control irritations in your life? ............................................................ 0 1 2 3 4
8. In the last month, how often have you felt that you were on top of things? 0 1 2 3 4
9. In the last month, how often have you been angered because of things that were outside of your control? .............................. 0 1 2 3 4
10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them? ............. 0 1 2 3 4

Please feel free to use the Perceived Stress Scale for your research.

Mind Garden, Inc.
info@mindgarden.com
www.mindgarden.com

References
APPENDIX D

STAI

SELF-EVALUATION QUESTIONNAIRE

Please provide the following information:

Name__________________________ Date________ S________

Age__________________________ Gender (Circle) M F T________

DIRECTIONS:

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

1. I feel calm....................................................... 1 2 3 4
2. I feel secure ................................................... 1 2 3 4
3. I am tense ...................................................... 1 2 3 4
4. I feel strained .................................................. 1 2 3 4
5. I feel at ease ................................................... 1 2 3 4
6. I feel upset ..................................................... 1 2 3 4
7. I am presently worrying over possible misfortunes ... 1 2 3 4
8. I feel satisfied ................................................ 1 2 3 4
9. I feel frightened ............................................. 1 2 3 4
10. I feel comfortable ....................................... 1 2 3 4
11. I feel self-confident ................................. 1 2 3 4
12. I feel nervous ............................................. 1 2 3 4
13. I am jittery ................................................... 1 2 3 4
14. I feel indecisive .......................................... 1 2 3 4
15. I am relaxed ................................................ 1 2 3 4
16. I feel content .............................................. 1 2 3 4
17. I am worried ............................................. 1 2 3 4
18. I feel confused ........................................... 1 2 3 4
19. I feel steady ............................................. 1 2 3 4
20. I feel pleasant ........................................... 1 2 3 4

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100
SELF-EVALUATION QUESTIONNAIRE
STAI Form Y-2

Name __________________________ Date __________________

DIRECTIONS
A number of statements which people have used to describe themselves are given below.
Read each statement and then circle the appropriate number to the right of the statement to
indicate how you generally feel. There are no right or wrong answers. Do not spend too
much time on any one statement but give the answer which seems to describe how you
generally feel.

21. I feel pleasant .................................................. 1 2 3 4
22. I feel nervous and restless .................................. 1 2 3 4
23. I feel satisfied with myself .................................... 1 2 3 4
24. I wish I could be as happy as others seem to be. .... 1 2 3 4
25. I feel like a failure ............................................. 1 2 3 4
26. I feel rested ...................................................... 1 2 3 4
27. I am “calm, cool, and collected” .......................... 1 2 3 4
28. I feel that difficulties are piling up so that I cannot overcome them 1 2 3 4
29. I worry too much over something that really doesn’t matter 1 2 3 4
30. I am happy ...................................................... 1 2 3 4
31. I have disturbing thoughts .................................... 1 2 3 4
32. I lack self-confidence ........................................ 1 2 3 4
33. I feel secure .................................................... 1 2 3 4
34. I make decisions easily ..................................... 1 2 3 4
35. I feel inadequate .............................................. 1 2 3 4
36. I am content .................................................. 1 2 3 4
37. Some unimportant thought runs through my mind and bothers me 1 2 3 4
38. I take disappointments so keenly that I can’t put them out of my mind 1 2 3 4
39. I am a steady person ....................................... 1 2 3 4
40. I get in a state of tension or turmoil as I think over my recent concerns and interests 1 2 3 4

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