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PHYSIOLOGICAL STRESS RESILIENCE: OPTIMIZING COGNITIVE PERFORMANCE IN RESERVED OFFICERS TRAINING CORPS DURING EXERCISE

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Table of Contents

Chapter I: Introduction 1

Purpose 3

Research Question 3

Research Subquestions 4

Research Hypotheses 4

Research Subquestion Hypotheses 4

Significance of Study 4

Assumptions 4

Delimitations 5

Limitations 5

Operational Definitions 5-7

Chapter II: Review of Literature 7

Exercise and Cognition 7

Executive Function 9

Executive Function and Exercise 11

Factors Affecting Executive Function During Exercise 17

Exercise Intensity 18

Exercise Duration 20

Cognitive Change Models 22

Inverted U Theory 22

Cognitive Energetic Model 23

Reticular Activating Hypofrontality (RAH) Theory 24

Physiology and RAH Interactions 27
Physiological Responses 27
Physiological Responses to Exercise 27
Neurostransmitters 30
The Prefrontal Cortex and Catecholamines 32
Cortisol 34
Brain Derived Neurotropic Factor (BDNF) 39
Blood Lactate 42
Cerebral Oxygenation 46
Temperature Regulation 52
Summary 57
Chapter III: Methodology 58
Experimental Design 58
Participants 58
Dietary Restrictions 58
Inclusion Criteria 59
Exclusion Criteria 59
Questionnaires and Documentation 60
Health Insurance Portability and Accountability Act 60
Health Status Questionnaire 60
Menstrual History Questionnaire 60
Physical Activity Readiness Questionnaire 61
Anthropometric Measurements 61

Study Measurements 61
Executive Function 61
Heart and Respiration Rate, Skin Temperature 62
Cerebral Oxygenation 62
Blood Oxygen Saturation 62
Study Timeline 62
Visits 63
Statistical Analyses 63
Chapter IV: Results and Discussion 65
Participant Characteristics 65
Absolute Exercise Intensity and Executive Function 65
Relative Exercise Intensity and Executive Function 66
Relationships Between Executive Function and Exercise Intensity 67
Discussion 68
Chapter V: Conclusions 74
Significance of Study 74
Future Directions 75
References 77

Tables and Figures

Table 1: CEDAR OWAT Test Descriptions in order of Administration 85

Table 2: Demographic Characteristics of Study Sample 86

Table 3: Means for Executive Function by Stage 87

Table 4: Multiple Comparisons for Absolute Exercise Intensity 88

Table 5: Multiple Comparisons for Cerebral Oxyengation Measures 89

Table 6: Multiple Comparisons for Relative Exercise Intensity 90

Table 7: Means for Physiological Variables by Stage 91

Figure 1: CEDAR OWAT..... 92

Figure 2: Tissue Saturation and Heart Rate Reserve..... 93

Figure 3: Tissue Saturation and Heart Rate Reserve..... 94

List of Appendices

Appendix A: Health Insurance Portability and Accountability Act 95

Appendix B: Health Screen Questionnaire 98

Appendix C: Menstrual History Questionnaire 103

Appendix D: Physical Activity Readiness Questionnaire 107

Abstract

Understanding the etiology of psychological and physiological stress resilience is imperative to human performance optimization. Previous studies examining how physiological systems interact with cognitive performance are not only quite limited, but have routinely examined performance immediately following exercise, the present study was designed to simultaneously and systematically examine the relationship between cognitive performance, specifically executive function, and high-intensity exercise in 15 ROTC cadets. Linear mixed modeling and Tukey's multiple comparison tests were conducted to evaluate significant differences in mean scores at varying levels of exercise intensity. Results indicated that both absolute and relative levels of exercise intensity are highly predictive of changes in both executive function scores and degree of cerebral oxygenation. Specifically, decrements in cognition begin to occur at intensities higher than 70% of HRR. This study is an important first step in identifying key factors and associated mechanisms indicative of positive adaptation to physiological stress (i.e., exercise), enabling the identification of potential targets for training or the modification of protocols to optimize performance in high risk occupations.

Chapter I: Introduction

Military personnel often engage in complex cognitive tasks during physically demanding and high-stress scenarios. These cognitive demands can include complex navigation, operational decision making, knowing when and when not to engage with an enemy as well as memorization of codes, layouts, and operational procedures. Thus, the ability to concurrently exercise while undergoing cognitive demands are essential components of success in military populations (Jeanick Brisswalter, Collardeau, & René, 2002; Lucas et al., 2012).

It has been generally accepted that cognition can be altered during exercise (Jeanick Brisswalter et al., 2002; Reilly & Smith, 1986). For example, experiments in the 1970s and 80s found completing a fatiguing bout of exercise caused transient decrements in cognitive and motor performance (Wrisberg & Herbert, 1976) and that high-intensity treadmill running decreased the ability to interpret topographical maps (Hancock & McNaughton, 1986). These studies initiated more than 200 studies in the 1990s aimed at elucidating the relationship between cognition and exercise. (Arcelin, Delignieres, & Brisswalter, 1998).

However, much of the research has been divergent. Recent reviews and meta-analyses have attempted to uncover potential factors which may attribute to these diverse findings (Chang, Labban, Gapin, & Etnier, 2012a). Their outcomes were that the type of cognitive test, time of test administration, fitness level of participants, exercise intensity and the type of task are all moderators of cognition during exercise (Chang et al., 2012a). Of these, high fitness level may contribute to an enhancement of cognitive function during exercise (J. Brisswalter, Arcelin, Audiffren, & Delignieres, 1997; Jeanick Brisswalter et al., 2002), while task administration and exercise duration are time-dependent (Chang et al., 2012a). Exercise intensity has been shown to potentially follow an inverted-U (Yerkes & Dodson, 1908), such that intensity ranges of 40-60%

elicited the most significant improvements in cognitive performance while intensities below or above this range exhibited decrements in cognitive performance (Reilly & Smith, 1986).

While, experimental considerations (e.g., type of cognitive test, time of test administration, fitness level of participants, exercise intensity) impact the interpretation of the effects of exercise on cognition, illuminating mechanisms that alter cognition during exercise have been derived from different arousal theories. Most recently, the reticular activating hypofrontality theory (RAH) has been proposed to explain the cognition-exercise relationship (Audiffren, 2016; Chang et al., 2012a; Sanders, 1983). Exercise facilitates both central and peripheral responses to exercise, causing a cascade of hormonal, metabolic and electrical responses (Ball, 2015; A. C. Hackney & Lane, 2015; Meeusen, Piacentini, & De Meirleir, 2001). The brain utilizes a finite amount of resources (e.g., oxygen, lactate, and glucose) that become limited as exercise intensity increases. Consequently, the brain begins to re-allocate or shift resources to maintain arousal and movement for purposes of sustaining exercise and simultaneously degrades cognitive processes (Dietrich & Audiffren, 2011).

This has been shown in more complex, higher order cognitive processes that rely on executive function (Dietrich & Sparling, 2004b) and may be a link with exercise intensity, such that low to moderate exercise may enhance cognition (A., Kavussanu, Willoughby, & Ring, 2013; Lucas et al., 2012) while higher exercise intensities result in performance decrements (González-Alonso et al., 2004; Kahina Oussaidene et al., 2015; S. Qian et al., 2015; Shaowen Qian et al., 2013) However, additional data suggests that cognition may be improved during exercise at higher intensities (Pesce & Audiffren, 2011; Tempest, Davranche, Brisswalter, Perrey, & Radel, 2017).

These findings bring to light important discrepancies that may be a result of differences in the way cognition and exercise are operationally defined and measured across studies. More importantly, these differences can have important implications for how changes in performance are interpreted. In other words, inconsistencies in the manner in which both cognition and exercise are defined, assessed, and evaluated may significantly impact interpretation of findings in addition to limit its generalizability. Consequently, it seems both logical and prudent to systematically examine the relationship between cognition and exercise intensity in a manner that most closely reflects demands placed on the populations of interest. For instance, often the type of cognition that is required of individuals in high-risk occupations (e.g., SWAT and Special Forces Operators) involves complex, higher order thinking while simultaneously engaging in high intensity exercise.

Purpose

The purpose of this investigation was to examine executive function during various exercise intensities in military personnel between the ages of 18-30 years. The study was designed to determine 1.) whether cognition is enhanced or degraded during high intensity exercise, and 2.) to identify measures that may be indicative or particularly sensitive to these alterations. More importantly, while previous studies have routinely examined cognitive performance immediately following exercise this study will assess the relationship between executive function and exercise intensity simultaneously. The examination of the exercise-cognition relationship focusing on the assessment of higher order processing, specifically executive function across exercise intensities, may provide an essential first step in understanding this relationship.

Research Question

1. How is executive function affected across exercise intensity?

Research Sub-questions

1. Is there a threshold of exercise intensity in which executive function begins to decline?
2. Are there physiological measures related to changes in executive function across exercise intensities?
3. Are there individual differences indicative of resilience to executive function decline?

Research Hypothesis

There will be a significant change in executive function affected by exercise intensities.

Research Sub-Hypotheses

1. There will be a significant positive relationship between executive function and low to moderate exercise intensities.
2. There will be a significant negative relationship between executive function and high exercise intensities.

Significance of Study

The exercise-cognition relationship is vital to understand the integrated mechanisms that underlie military demands. This potential interaction serves as the backbone of their operational success, and within military populations, this success or failure may likely depend on an outcome of a mission. Therefore, determining potential thresholds of executive function changes and the physiological measures that may be indicative of these changes is of the utmost importance and may allow for targeted training protocols to optimize human operational performance.

Assumptions

1. Participants will provide candid and accurate information while completing the included study documentation.
2. Participants will be free of injury or disease.
3. Participants will follow all pre-testing guidelines, as outlined in Chapter 3.

4. Participants will perform each of the included tests with their maximum effort.
5. Equipment for measuring executive function and physiological measurements will produce valid and reliable measurements.

Delimitations

1. The findings of this proposed study will only apply to active ROTC cadets between the ages of 18-30.
2. Participants with recent musculoskeletal injury or a previous injury that may affect testing will be excluded.
3. Participants will be recruited from the University of Oklahoma's Army Reserve Officers Training Corps (OU Army ROTC)

Limitations

1. Participants will continue their normal daily ROTC activities and will be required to minimize high intensity exercise within a 24-hour period of participants' study visits.
2. Participants are volunteers and may not provide an accurate representation of the entire population.
3. The study will examine aerobically trained individuals and may not be indicative of the population.
4. Participation will be limited to persons who are currently active members of the OU Army ROTC.

***Operational Definitions*:**

Blood Oxygen Saturation (SpO_2): The amount of oxygen that is bound to hemoglobin and is based off 100%, such that every available heme molecule is full of oxygen.

Cedar Operator Workload Assessment Tool (OWAT): A tablet version of NASA's Multi-Attribute Task Battery-II designed to evaluate human performance during a multitasking paradigm was used to assess executive function.

Cerebral Oxygenation (brain O_2): The amount of oxygenation supplied to the brain.

Executive Function (EF): A domain of cognition specifically encompassing inhibitory control, working memory, and cognitive flexibility.

Executive Function Exercise Test (EFET): The EFET trials consisted of the completion of a graded exercise test (performed on a treadmill) with and without concurrent executive function assessment. Participants wore a weighted backpack a vest (35 lbs. combined) intended to simulate the weight and movement restrictions of military PPE.

Graded Exercise Test (GXT): The GXT will serve as the maximal exercise test conducted. It consists of 2-minute stages beginning at 0% incline increasing in incline by 2% each subsequent stage. Participants will walk at 3.5 mph until 16% incline is reached. Every subsequent stage following 16%, speed will increase by 0.5 mph.

Heart Rate Reserve (HRR): HRR is calculated as the highest 30-second heart rate achieved minus the last 30-seconds of resting heart rate recorded during the maximal graded exercise test = ($HR_{max} - HR_{rest}$).

Hit Rate: A measure of accuracy that quantifies the number of 'correct' responses to target stimuli while accounting for the number of false positive and missed responses. Hit Rate is calculated as Hit / (Hit + False Positive + Miss), with miss being defined as an incorrect, or non-response to a given stimuli.

Precision Average: The rate at which participants correctly respond to the stimuli being presented across all three OWAT tasks. Precision is calculated as Hit / (Hits + False Positives).

Reticular Activating Hypofrontality theory (RAH): Theory posited to explain the cognition-exercise relationship in which allocation of resources occurs limiting top-down cognitive processes at higher exercise intensities.

Chapter II: Review of Literature

The purpose of this study is to examine the understanding of individual factors that may be associated with physiological stress resilience. Previous work has examined *psychological* stress resilience, however, much less is understood in terms of *physiological* stress resilience, specifically pertaining to concurrent exercise and cognitive function. This proposed study is essential in identification of key measures and mechanisms which may be indicative in alterations and adaptations to physiological stress and long-term will allow the examination of stress and the psychophysiological relationships with cognition, brain function and physiological mechanisms which the body regulates to find empirically based approaches to optimize human performance.

Exercise and Cognition

Cognition

Almost all aspects of human performance, no matter the medium, incorporates some level of cognition. Cognition is a general term that comprises many aspects of brain functions, such as memory, intellect, language, perception, learning, and executive function. Within the context of sport and military operations, performance is often related to the ability of individuals to make decisions and engage in complex critical thinking while simultaneously participating in intense physical work (Bailey et al., 2008; Del Giorno, Hall, O'Leary, Bixby, & Miller, 2010). As such, cognition, specifically executive function, has been identified as an important factor in understanding of athletic performance (Jeanick Brisswalter et al., 2002) and thus, is likely to be imperative in military and first responder performance as many of the tasks in theses professions

also involve simultaneous physical and cognitive demand (Bailey et al., 2008). Therefore, understanding the interaction and, ultimately, the limitations of the cognitive demand via executive function and physical exercise is an integral aspect of performance enhancement.

Research has been less than clear in determining the effects of exercise on cognition. In a meta-analysis conducted by Chang et al. (2012) it was found that an acute session of exercise facilitated a small effect ($d = 0.097$) on cognitive performance (Chang et al., 2012a). Their findings were derived from results based on three categories: 1. Cognition assessed during exercise 2. Cognition assessed immediately post-exercise and 3. Cognition assessed over 1-minute post-exercise.

Assessment of cognition *during* exercise revealed that exercise intensity did not significantly alter effects of the cognitive task administration ($p > 0.05$) (Chang et al., 2012a). Specifically, low to very hard intensities showed a positive effect size while very low intensity exhibited a negative effect of cognitive performance. The time of task administration did significantly alter the effects of cognitive performance ($p < .001$). Tests that were administered within the first 10 minutes of exercise elicited no significant effects ($d = .060$), while administration between 11 to 20 minutes of exercise elicited negative effects ($d = -.182$), and 20 minutes or more of exercise resulted in positive effects ($d = 0.261$) on cognitive performance (Chang et al., 2012a). A meta-analysis on cognitive performance and exercise results were examined by Lambourne and Tomporowski which noted similar findings as the Chang et al. meta-analyses (Lambourne & Tomporowski, 2010). Specifically, positive effects on cognitive performance were exhibited when it was assessed after the first 20 minutes of exercise while negative effects seen within the first 20 minutes. Cognitive performance assessed immediately post-exercise revealed that low to moderate exercise intensities exhibited a positive effect of

cognition (d= 0.169) while hard to intense exercise intensities did not exhibit a significantly different effect from baseline assessment (d = 0.003 and -0.158, respectively). When cognition was assessed after a delay (at least 1 minute after the cessation) of acute exercise, very light exercise elicited a negative effect of cognitive performance (d = -0.113) while moderate (d = 0.202), hard (d = 0.268), very hard (d = 0.465) all exhibited positive effects of cognitive performance. Current synthesis of the literature suggests a small improvement of general cognition during exercise, with the greatest improvements seen after 20 minutes of low to moderate aerobic exercise.

Executive Function

Executive function is an important component of cognition that encompasses specific elements of higher order mental processes essential for critical thinking, concentration, and the execution of decisions when 'auto-pilot' would be detrimental to performance (Diamond, 2013). These higher level cognitive functions control lower level processes and goal-directed behaviors (Alvarez & Emory, 2006). It has generally been accepted that inhibitory control, working memory, and cognitive flexibility are the main components that comprise executive function (Alvarez & Emory, 2006; Diamond, 2013; Miyake et al., 2000).

Inhibitory control has been described as the ability to control one's attention, behavior, thoughts and/or emotions to override an internal or external lure. This control facilitates a reaction and subsequent behavioral action instead of solely relying upon impulsivity (Diamond, 2013). Inhibition can be broken down into sub-components, including selective attention, self-control and interference control. Together, inhibition is often referred to as 'inhibitory control'. A common example of inhibitory control is a 'go- no-go task'. In this task a series of X's and O's are flashed on a screen. The participant is instructed to click the mouse each time an X flashes on the screen

and not to click anything when you see an O. These are presented very quickly, which forces an individual's inhibitory control, clicking for an X and not an O.

Working memory, the second component of executive function, involves cognitively possessing information and working with that information, such that the information is no longer visually present (E. E. Smith & Jonides, 1999). Working memory includes both verbal and non-verbal (commonly referred as visual-spatial) components. It should also be noted that working memory is essential for reasoning in that it relies on the ability to link connections and disentangle verbal, spatial and visual elements and recombine them in new ways (Diamond, 2013). Additionally, working memory is different than short term memory. While working memory involves the manipulation of information, short term memory is solely holding and remembering information. This specific difference is also pertinent within the brain, for working memory resides within the dorsolateral prefrontal cortex (Alvarez & Emory, 2006). A commonly used example of working memory can be seen in a reordering task. For instance, an individual may be asked to reorder numbers that they have previously seen (e.g. 5,8,3,9) and are asked to reorder them in numerical order (3,5,8,9) (Diamond, 2013).

Cognitive flexibility, the third component of executive function, builds upon both inhibition and working memory. The ability to change spatial and interpersonal perspective is the main tenet of cognitive flexibility (Diamond, 2013). This framework provides the integration with the first two components of executive function. An individual's prior perspective provides framework for a memory of that perspective. Working with that memory in the present time and altering that memory involves the integration of working memory, inhibition and cognitive flexibility. Thus, being adjustable to different demands and being advantageous of sudden and unexpected events are both keys of cognitive flexibility. Cognitive flexibility can be either verbal

and categorical in nature (Robinson, Shallice, Bozzali, & Cipolotti, 2012) such that it is similar to both working memory and inhibition by possessing a verbal and spatial nature. A commonly used test to assess cognitive flexibility is the Wisconsin Card Sorting Test (Diamond, 2013). In this test, cards are presented on top (e.g. 3 red circles, 1 blue diamond, 2 yellow squares, 4 green stars). A card is then presented below these cards (2 red stars). The participant can be asked to match this card based on its number, shape, or color. Doing this requires the ability to be flexible and switch between the stimuli presented.

Though executive function are important for daily living, sport and military personnel are often required to engage in tasks that are very physically demanding while undergoing complex cognitive tasks, including inhibition, working memory and cognitive flexibility (Bailey et al., 2008). The competing demand of cognitive and physical exercise requires the ability to possess cognitive flexibility, working memory and inhibitory control; thus, the link between physical activity and executive function is fundamentally tied with performance (Jeanick Brisswalter et al., 2002).

Executive Function and Exercise

There are a several ways in which executive function can be assessed and understood in terms of its location within specific brain regions. Identification of the brain regions that executive function occurs allows for the understanding of the effect of concurrent executive function tasks and exercise. Positron Emission Tomography (PET), functional magnetic resonance imaging (fMRI) and near infrared spectroscopy (NIRS) and transcranial doppler (TCD) have all been utilized in understanding the brain regions that are responsible in potentially determining the role of executive function and exercise responses. Together, these tools have implicated the prefrontal cortex (PFC) as the primary location for which executive function resides (Jeanick Brisswalter et

al., 2002; Del Giorno et al., 2010; Dietrich & Sparling, 2004a; Lucas et al., 2012; McMorris, Sproule, Turner, & Hale, 2011).

The Chang et al. (2012) meta-analysis showed that exercise accounted for significant positive effects on executive function tasks. Positive effects were seen for simple math and attention based executive function tasks while there were no significant findings for verbal working memory. There was a negative effect seen for spatial working memory, meaning that verbal working memory improved while spatial working memory declined.

Working memory is often tested due to its relationship with occupations that rely on it during exercise, specifically cognitively demanding sports and military personnel. McMorris et al. conducted a meta-analysis to investigate the speed and accuracy of working memory tasks as a result of intermediate intensity exercise (McMorris et al., 2011). Intermediate exercise intensity was defined as 50-75% of either VO_{2max} or Heart Rate Reserve (HRR). The synthesis of their findings revealed a large positive effect for overall working memory tasks, when assessed during and post-exercise. However, a low to moderate negative effect size was seen for accuracy of working memory tasks during intermediate intensity exercise, denoting a decay in accuracy performance. The negative effect (decay in working memory accuracy) may be attributed to an increase in overall stress that the brain is undergoing, one that plausibly may increase specific aspects of executive function, but may inhibit the accuracy of working memory, in part due to the overall stress upon the system.

In a 2013 study, Quelhas et al. also examined working memory during moderate exercise. In experiment one, 24 male participants exercised at different exercise intensity ranging from 95 to 170 watts with a heart rate ranging from 80-160 beats per minute (A. et al., 2013). Participants were instructed to pedal at 95 watts (63 rpm), 165 watts (83 rpm), 155 watts (78 rpm) and 170

watts (85 rpm). These stages represented the time points for each working memory task 'block' and were chosen to represent the different stages of a bicycle race. These blocks changed every two minutes along with the working memory task, such that the presentation of the stimuli during the tasks were alternated between 2.5 (fast) to 5 seconds (slow). Findings from this experiment showed that when the task was presented fast to the participants, working memory showed improved performance; however, when combined as total working memory task (combining fast and slow), performance deteriorated (A. et al., 2013). This finding is interesting to note in that the speed of the stimuli facilitated a deterioration in working memory performance.

In the second experiment, 120 men and women were split amongst three groups (very low, low or moderate intensity (5, 50, and 60 watts, respectively) that corresponded with a heart rate of 120-130 beats per minute (A. et al., 2013). These data suggested that working memory was improved during the moderate intensity (60 watts) but was not significantly changed in the other two conditions.

Overall, these studies suggest in part that general working memory may be improved during moderate exercise intensity, but the speed of which the tasks are presented seem to influence the overall performance. Thus, there may be underlying mechanisms that cause a decay in working memory accuracy, which in part, may be influenced by the speed of which the stimuli of the tests are presented. Overall, this may be a great oversimplification of previous studies that have not identified speed of presentation of stimuli as a potential modifier in executive function performance.

Task switching, a derivative of cognitive flexibility, has also been assessed during submaximal aerobic exercise. Investigating both young and old participants, Pesce and Audiffren examined their reaction times using task switching tests both at rest and during exercise, using an

exercise intensity of 60% HRR, lying within the same moderate exercise intensity (Pesce & Audiffren, 2011). One hundred male and female participants (n = 53 between 16-24 and 47 between 65-74) were subdivided into either sport with 'low cognitive demand' (e.g. swimming, gymnastics, running, rowing) or 'high cognitive demand' (e.g. orienteering and soccer). Participants had their HRR% previously computed by the authors, and then were asked to perform 20 minutes of aerobic exercise at 60% HRR. The first 10 minutes of the exercise the participants read and listened to their task instructions with the tasks divided either a low or high demand switching task. The targets were either matched (low) or mismatched (high); meaning that the participant had to respond in a different manner, relying on a higher level of executive function for both cognitive flexibility and inhibition due to the needed control to answer and match targets as they were presented to the participant. The second half of the aerobic exercise was then combined with the task switching test. Their findings revealed that aerobic exercise did elicit an increase of task switching performance as denoted by a decrease in reaction time. However, an interesting observation in their findings was that a moderator was seen between the types of task switching tests. When moderating for the low vs high demand tasks, the ability to 'switch' between objects in the task and answer appropriately was smaller in time (increased performance) for the younger versus older participants. This was also seen during exercise versus the resting condition and, specifically, within the athletes during the high demand task versus those in low demanding tasks, suggesting that cognitively demanding sports and occupations facilitate improved executive function at intermediate/moderate aerobic exercise when conducted simultaneously.

The effects of exercise on inhibitory control has also been examined during exercise. Lucas et al. (2012) examined the effects of exercise on inhibitory control (Lucas et al., 2012). 13 (25 ± 4 years) and 9 (62 ± 3 years) underwent a staged protocol in which executive function was measured

at rest, followed by an 8-minute stage at 30% of HRR followed by an 8-minute stage at 70% HRR. During rest the executive function task (Stroop Task) was performed in blocks, starting from simple (block 1) and progressing in difficulty to difficult (block 4). During the two exercise stages, blocks 1 and 4 were performed in alternating fashion. At rest, the younger group possessed faster reaction times in both the simple and difficult tasks and was also exhibited in both the 30% and 70% HRR stages with both the simple and difficult blocks. While the younger group was faster across all time points, the pattern of responses was similar between groups. Thus, both groups saw a significant improvement of executive function (inhibitory control) at both low (30% HRR) and moderate (70% HRR) exercise intensity.

The ability to examine multiple executive function domains has also been examined during sub-maximal aerobic exercise. Tempest et al. (2017) recently examined the effects of inhibitory control and working memory on two different intensities, heavy (70% of VO_{2max}) or very low intensity (30% VO_{2max}) in a counterbalanced order. Both protocols consisted of 60 minutes in duration during which the participants completed executive function tasks, broken down into 'task blocks. Each block lasted 6 minutes and was performed 10 total times with each block consisting of 2 minutes of no task, 2 minutes of the inhibitory control task and 2 minutes of the working memory task.

Their analyses revealed that the inhibitory control saw a significant decrease in reaction time from block 1 to block 10 ($p < 0.001$) in the heavy exercise condition, but there was no significant change within the very low condition ($p > 0.05$). The working memory tasks revealed that performance declined from block 1 to block 10 ($p < 0.001$) while the very low condition saw no significant change in either direction ($p > 0.05$). Thus, these findings suggest that during extensive aerobic exercise above ventilatory threshold elicits a positive reaction of inhibitory

control (via a reduction of reaction time) while working memory shows a decline in performance (denoted by the number of errors during the task)

Del Giorno et al. (2010) also examined multiple executive function domains, assessing inhibitory control (via attentional processing) and cognitive flexibility (Del Giorno et al., 2010). 31 college aged participants (17 male, 13 female) with an average VO_{2max} of 46.5 (+-9.0 mL·kg–1·min–1) completed two sessions counterbalanced and randomized at either 75% or 100% of their ventilatory threshold (VT) for 25 minutes. The two domains were assessed in succession (inhibitory control and cognitive flexibility) pre- during and post-exercise and were separated by the number of successful trials in each task.

Their findings showed that the 100% VT group elicited a significant increase in the number of 'false alarms', total errors and 'preservative errors' when compared against the 75%VT group. These data suggest that the higher intensity group exhibited a decrement of inhibitory control and cognitive flexibility as compared with the moderate intensity group.

Similar findings have also been found when examining working memory and inhibition versus general cognition assessments in both treadmill and cycling conditions (Dietrich & Sparling, 2004b). In their first experiment, working memory was compared against a general cognition test and a control group who performed both tests while sitting. Participants completed a between participant's design, randomly assigned to a treadmill or cycling condition each of which consisted of 50 total minutes of exercise at 70-80% of maximal heart rate (HR_{max}). The tests were administered at the 25-minute mark, which has been identified as a time point in which positive cognitive benefits have been seen. However, both the running and cycling conditions showed a decrement in the working memory task, but not the general cognition task (denoted by number of errors throughout test, $p < 0.05$).

Their second experiment used 8 endurance trained runners who completed 65 minutes of treadmill running at 70-80% of their HR_{max}. Protocol differences from their first experiment included the overall time, test administration (40 minutes into the protocol instead of 25) and the type of neurocognitive tests. However, these tests still focused on working memory and general cognition. The test was assigned randomly and in counterbalanced fashion. The findings from this experiment revealed a significant increase in the amount of errors produced in the working memory task ($p < 0.05$)

Collectively these studies do not elucidate the effects of executive function during exercise. There are some data that show an improvement in executive function at low intensity domains (Del Giorno et al., 2010; Tempest et al., 2017) while other data shows an increase of executive function at both low and moderate intensity domains (A. et al., 2013; Lucas et al., 2012). Attempts to determine the reason for the discrepancies in findings has been described within arousal models, most notably the Inverted U Theory. However, more recent research has proposed the RAH theory to explain possible improvements at specific exercise intensities and why there may be underlying performance decay in specific aspects of executive function. These are key aspects to try and uncover. In sport and military, concurrent demands of executive function during intense physical demands relies on the ability of an individual to possess a high level of inhibitory control, working memory and cognitive flexibility. As we will see in this next section, there are several potential moderators that may begin to explain these differences and further understand the current gap in research that necessitates the present study.

Factors Affecting Executive Function During Exercise

The main moderators that have been identified to affect executive function during exercise are exercise intensity, exercise duration, exercise modality, the type of executive function assessed and overall study design (Browne et al., 2017).

Exercise Intensity

Exercise intensity has been linked due to the increased metabolic, mechanical and biochemical responses that occur, including hormonal and intramuscular responses in addition to cardiovascular feedforward and feedback mechanisms. Ultimately, these responses have been shown to cause regional shifts in brain activation, especially in the motor cortices which may at the expense of other brain regions (Dietrich & Sparling, 2004b). An increase of exercise intensity facilitates an increase of arousal in brain activity (Kubitz & Mott, 1996) and has been shown to increase regardless of aerobic fitness level (Magnié et al., 2003). However, the discrepancy may potentially lie in the scant available literature that determines the metabolic load of different exercise intensities that may be optimal for executive function performance during exercise. In the meta analyses conducted by Chang et al., the most significant increase of cognitive performance was seen within high intensity exercise; however, many of these studies did not explicitly examine executive function (Chang et al., 2012a).

Labelle et al. (2013) had participants perform aerobic exercise at either 40, 60 or 80% of their peak power output for 6.5 minutes while simultaneously performing an inhibitory control task. Their data found no significant effect (positive or negative) on inhibitory control during the low to moderate exercise conditions, but did reveal a significant decrement of inhibitory control performance from the 60 to 80% conditions (Labelle, Bosquet, Mekary, & Bherer, 2013).

A 2015 study by Whyte et al. examined inhibitory control pre- and post-exercise that consisted of high intensity interval agility running (Whyte, Gibbons, Kerr, & Moran, 2015). In

their two exercise conditions, an inhibitory control task was performed counterbalanced with a general cognition task. Their results exhibited a significant decay in inhibitory control from pre- to post-high intensity exercise. However, the scores on the general cognition test did not significantly differ.

Inhibitory control has also been examined during high intensity (M. Smith et al., 2016). In this study, participants completed 10 minutes of treadmill running at moderate (70% HRR) and high (90% HRR) intensities on two separate sessions at least 48 hours apart conducted in a counterbalanced fashion. Once participants reached the required HRR%, they then completed 100 trials of an inhibitory control task (lasting approximately 2-minutes in duration). Their findings exhibited a significant decay in inhibitory control via an increase in reaction time and error rate at the high intensity exercise domain.

Lo Bue-Estes et al. examined the effects of working memory tasks during high intensity exercise utilizing trained collegiate women (Lo Bue-Estes et al., 2008). Their study utilized a staged protocol, such that working memory tasks were performed between increased levels of difficulty (denoted by percentage of VO_{2max}) during each 2-minute stage. Participants performed 2-minute stages at 25, 50, 75 and 100% of VO_{2max} with 90 seconds between each stage in which a working memory task was assessed. Their analyses revealed that working memory was significantly decayed across each stage and returned to baseline levels within 3-minutes of cessation of maximal exercise (Lo Bue-Estes et al., 2008).

Each of the previous studies examined a domain of executive function in trained subjects (Labelle et al., 2013; Lo Bue-Estes et al., 2008; M. Smith et al., 2016; Whyte et al., 2015) These findings seemingly contradict previous synthesis of the literature, which found an overall positive effect of cognitive performance when assessed during exercise (Chang et al., 2012a). However,

the biggest discrepancy within exercise intensity is by what definition is exercise intensity defined and utilized. The American College of Sports Medicine defines 'high intensity' as anything over 80% of VO_{2max} or HR_{max}. Based on this definition, much of the research that has found positive effects may fall into a lower intensity domain (A. et al., 2013; Chang et al., 2012a; Lucas et al., 2012).

Exercise Duration

Exercise duration has also been identified as a potential moderator of executive function performance during exercise. Recent meta analyses found that negative effects were seen in overall cognition when assessed within the first 20 minutes of exercise while positive effects were seen after 20 minutes of exercise (Chang et al., 2012a; Lambourne & Tomporowski, 2010)

Pesce et al. found a positive effect on cognitive flexibility when cycling at 60% of HRR for 20 minutes (Pesce & Audiffren, 2011), while Lucas et al. found an improvement in inhibitory control during 16 minutes of cycling at moderate intensity (70%HRR) (Lucas et al., 2012). However, Tempest el al. found that low and high intensity exercise facilitated an improvement in reaction time during an inhibitory control task, however, working memory was decayed during 60 minutes of cycling at high intensity which was determined as 10% above ventilatory threshold (Tempest et al., 2017).

Additionally, Del Giorno et al. found a decrement in working memory when assessed during 30 minutes of cycling at ventilatory threshold (Del Giorno et al., 2010). Dietrich and Sparling also found a decrement in working memory during aerobic exercise (Dietrich & Sparling, 2004b). What it is interesting of note is the difference in study designs. Dietrich and Sparling conducted two experiments. In their first experiment, participants either ran or cycled (randomized prior to their trial) at 70% of their age predicted HR_{max} for 45 total minutes, with the working

memory task being assessed after the first 20 minutes. In both conditions, a decrement in working memory performance was determined by a significant increase in error rate. In their second experiment, they chose the running condition and increased the duration to 65 total minutes and increasing the working memory task to 40 minutes in duration. This experiment also revealed a significant increase in error rate across the duration of exercise compared with a control group as well as throughout each block of trials throughout the duration of exercise.

While these findings are similar, they may be the cause for differential findings, as their intensity may not be indicative of high intensity as defined by Browne et al. (Browne et al., 2017). These findings seem to contradict those synthesized by Chang et al. as well as Lambourne and Tomporowski, which may allude to the differences in the type of cognition assessed. Additionally, each of the study designs employed have different in duration, intensity, assessment and modality. Thus, these findings have not elucidated how executive function changes throughout exercise intensity.

In sum, simple reaction time and simple decision-making performance is improved during acute aerobic exercise. It is unclear as to executive function tasks during aerobic exercise. Much of the evidence, however, points to a performance decline of executive function during acute aerobic exercise (Audiffren, 2009; Davranche & McMorris, 2009; Del Giorno et al., 2010; McMorris et al., 2011), specifically at shorter durations. Additionally, it seems that higher intensities attenuate executive function performance. Due to variety of executive function tasks utilized, exercise modalities and intensities used; the evidence is unequivocal in determining the relationship between exercise and executive function. This gap in the literature is important to elucidate to determine the effect on exercise intensity and executive function and measures that

may potentially be involved with the alteration of executive function at different exercise intensities due to their integration with military, first responder and athletic performance.

Cognitive Change Models

Inverted-U Theory

Several models have attempted to understand the relationship between cognition and exercise. In its essence, these models have attempted to tie two fundamental principles that are evoked from the interaction of exercise and cognition, stress and arousal. Stress and arousal are intrinsically linked, which has caused difficulty in determining their individual components (Yerkes & Dodson, 1908) as both are aspects of the mobilization of energy within the human body. It's been proposed that stress is a response governed by hormonal influence while arousal is guided by cortical or autonomic responses. This interlinking between these two important components of both exercise and cognition has proven difficult to understand. Thus, there have been a few primary models aimed at elucidating this relationship.

The inverted-U hypothesis, which began initially to understand human arousal, shifted to an attempt to also understand the complex interaction within cognition and exercise. In its essence, the inverted-U hypothesis suggests that exercise will influence the effect of cognitive performance, such that moderate intensity will elicit the greatest benefit from a cognitive performance standpoint (Chang et al., 2012a). In 1908, Yerkes and Dodson performed an experiment on mice under three conditions. Specifically, they varied the levels of electric shock to the mice in an effort for the mice to perform a task (Yerkes & Dodson, 1908). Their findings exhibited that when stimulation can be increased to elicit an improvement in performance (to a given threshold), however, once past that threshold, performance deteriorates (Mair, Onos, & Hembrook, 2011). Thus, at a given level of arousal, cognitive performance may increase until a point in which that arousal becomes

to great and a given cognitive outcome then deteriorates back to baseline (forming an Inverted-U). At optimal levels of performance stress is then minimized and performance is increased while at suboptimal levels performance decreases due to a successive increase in stress (Sanders, 1983). However, this would imply that an increase of demanding tasks would then facilitate a lower optimal level of arousal.

However, studies assessing neurotrophic responses (e.g. brain derived neurotrophic factor) have shown significantly higher levels to high intensity exercise versus low intensity exercise. However, if this model is true, then it contradicts the findings presented by Chang et al. As a reminder, Chang et al. found an overall positive effect of cognitive performance when assessed during exercise at hard (d = .130) and maximal (d = .138) exercise, with the highest response occurring via executive function tasks (d = .260).

Cognitive Energetic Model

More recently, the cognitive energetic model was proposed to explain this complex relationship by accounting for stress and arousal, coupled with cognition and exercise (Sanders, 1983). Specifically, Sanders determined that the entrance of stress occurs if effort is either overloaded or falls short to adjust the energy requirements of the body. Thus, stress and effort work together to attempt to facilitate equilibrium at high energetic demands.

Sanders' theory lies within three mechanisms: Arousal, activation and effort (Dietrich, 2008; Sanders, 1983). These three components rely on the aspect of arousal and activation being intertwined, which while they act concurrently, they are independent in their characteristics. For instance, Sanders' model implies that an acute bout of exercise increases both arousal and activation, modulating both sensory input and motor output. While this is in part true, there is also a time in which activation may be more specific and occur not in line with arousal, such that

activation refers to the ability of the system to engage motor processes while arousal acts as a stress response via the endocrine system(Dietrich & Audiffren, 2011).

Reticular Activating Hypofrontality (RAH) Theory

Dietrich and Sparling (2004) investigated the effects of aerobic exercise on two different cognitive tasks. In each experiment, they found that the task which examined executive function elicited a decrement in performance while the task that assessed general intellect was unaffected. The complexity of the brain underscores the impact of it on human performance. Each test assessed in the study required sustained mental effort and no participants reported a significant difference in task difficulty (Dietrich & Sparling, 2004b). Thus, two tests that seemingly were the same in difficulty and length facilitated controversial results. This is in many ways, why the two models seemed on the surface to represent fundamental operational mechanisms to explain cognition and exercise, yet this, along with several other findings, directly conflict with their theories.

While previous research has delineated between time of assessment, previous models did not account for changes between pre, during and post-exercise. Because there was no account for the differences of the brains operational systems, all processes were a global entity. This, however, has been shown to not be the case.

Dietrich and Audiffren's RAH model is rooted within three fundamental energetic principles: 1. That the brain receives a constant but limited amount of resources (chiefly glucose and oxygen). 2. Human movement requires a large amount of metabolic resources that, are in part, allocated to the brain (motor, sensory and autonomic) regions. This metabolic requirement largely depends on the amount of muscle tissue involved with a given movement and the intensity of that movement. 3. Neural processing is competitive in nature (Dietrich & Audiffren, 2011). Thus, using principle 1 as a guideline, if neural processing increases in one region it then facilitates a

shift in metabolic resources from a region that at that given time has less of a requirement in neural processes. Thus, due to Principle 2, a large amount of resources is required to sustain human movement, which then leads to an overall reduction in total resources available.

From a neurocognitive standpoint, these three principles are then guided by physiological implications, being that 1. Catecholamines respond and mediate arousal of human movement (exercise) which modules information processing. 2. The brain operates two independent cognitive domains, the implicit and explicit systems. 3. Exercise presents a very high demand to the organism and as such can cause the brain to be 'taxed', in turn causing a downregulation of brain regions that become 'irrelevant' to specific motor skills and tasks required to complete the exercise (Dietrich & Audiffren, 2011). This theoretically happens in a hierarchical manner such that highest cognitive demands would downregulate to areas that support basic human movement and function (Dietrich & Audiffren, 2011). This then implicates the prefrontal cortex as becoming limited during exhaustive and taxing exercise.

From a physiological perspective, one can agree that human motion (exercise and physical activity per sport or military requirements) is exacerbated by the amount of muscle used, intensity and duration. This is then deconstructed in terms of the effects of exercise on executive function between during and post-exercise, which completely alters findings (Dietrich, 2006) because at the cessation of exercise alters the competitive resources that the brain underwent during concurrent exercise and executive function.

This is understood when taking into consideration that a task may not be, or more likely, is not, characteristically explicit or implicit in nature. It is an oversimplification to state that a given task (motor or otherwise) requires the engagement of only one brain region. In fact, previous research has shown that several brain regions can be active within a given task or skill (Magnié et

al., 2003; Otten, 1996; Polich & Kok, 1995; Pontifex & Hillman, 2007). For instance, language, a very complex task, can be practiced ad nausea such that a brain representation of language can be imprinted and become implicit in nature. In a study by Delignieres in 1994, novices and expert fencers performed choice reaction time tasks during different cycling intensities (Delignieres, Brisswalter, & Legros, 1994). The results exhibited that non-experts saw a significant decrement in choice reaction time performance as exercise intensity increase, while the expert group saw an improvement in performance from 20-60% of maximal aerobic power with a non-significant change in performance from 60-80% of maximal aerobic power (Delignieres et al., 1994). In short, the novices both perceived and executed the task in an explicit way while the experts completed the task as an implicit mechanism, denoting by both perception, effort and cognitive performance.

Delignieres findings can also be supported by Mishkin's 1984 review in which evidence has clearly shown that shifts of both neural activation and control can and are shifted due to practice of a given motor skill or tasks, such that a shift can start within a prefrontal region, denoting an explicit component and shift its activation (over time and with significant practice) to the basal ganglia, denoted as an implicit region (Mishkin, 1984).

With practice, the brain can build implicit representations of a given task or skill. As Dietrich and Audiffren propose, this is the brain's "divide and conquer principle" (Dietrich & Audiffren, 2011), meaning that the brain possesses an ability to shift a complex motor skill or task as an ingrained, implicitly driven component that requires less resources to be allocated to the prefrontal cortex which spares competitive resources. Automation of a task or skill is then less dependent upon higher order functions (explicit) via the prefrontal cortex and then shifts those to an implicit driven domain such that the organism may operate more efficiently.

However, any task that requires several cognitive representations (e.g. executive function) relies upon the explicit domain. Any motor or skilled tasks that requires sensimotor control (e.g. sports or military scenarios in which fast, stimulus driven responses occur) cannot be controlled by the explicit system, thus as this occurs, the RAH model proposes that this form of exercise requires a substantial resource allocation for those implicit demands and downregulates the allocation for the explicit domain. Human movement in and of itself requires a large amount of neural and metabolic resources. Increasing the intensity and/or duration of this movement limits the amount of resources. If, physical fitness is an indicator of executive function and concurrent exercise, then theoretically operating at a given workload would allow for more efficient implicit control and potentially spare metabolic resources needed to sustain concurrent executive function and physical activity.

Physiology and RAH Relationships

Physiological Responses

Acute exercise elicits a substantial response of the physiological components of the human body which elicits several sub-systems that respond to exercise. Hormone responses are one of the major elements in the body's response to an acute exercise bout. These occur to facilitate cardiovascular adjustments, activate metabolic pathways, maintain hydration status and is in part an essential component of the reactivity to stress. Hormonal responses have been proposed as highly interrelated which has been explained by the 'hormonal exercise response model' by Hackney and Lane (A. C. Hackney & Lane, 2015).

During the onset of exercise, an initial phase that is centered mainly on feedforward responses are facilitated chiefly by increases of catecholamines within the brain and circulation.

The systemic response of elevation in catecholamines occurs as a facilitation of the sympathetic 'spillover effect' (Christensen & Galbo, 1983). This phase is the onset of both NE and E release.

Within a minute after the onset of exercise, the second phase begins, defined, in part, by the release of thyrotropin-releasing factor, corticotropin-releasing factor and growth hormone releasing factor (A. C. Hackney & Lane, 2015). This is in response of the anterior pituitary glands stimulation to release hormones via hypothalamic stimulation which causes these 'tropic hormones' to be released into systemic circulation (A. C. Hackney & Lane, 2015). This hormonal cascade is brought about by the hypothalamic-pituitary-adrenocortical activity, causing an end release of cortisol.

The third phase of the HERM model begins within a few minutes post-onset of exercise. This is marked by the augmentation of the hormone release from the pituitary gland via the sympathetic-adrenal axis. This facilitates peripheral endocrine glands as secondary facilitators to pituitary regulation (A. C. Hackney & Lane, 2015). This is led by feedback mechanisms, rather than the feedforward mechanisms in the first two phases of the model. This shift is posited by the primarily neural factors whereas the third phase is marked by an increase in humoral and hormonal influences that regulate the global response to exercise, previously termed as the internal milieu (Christensen & Galbo, 1983). This milieu is part of an overall modulator component based on exercise duration and intensity, which is in part facilitate by substrate availability (Anthony C. Hackney, 2006).

The reticular activating process consists of two basic arousal/energetic systems: the noradrenergic and dopaminergic pathways (Meeusen & Piacentini, 2001). Acute exercise activates these two monoamine systems which in-turn facilitates the release of norepinephrine and dopamine within several brain regions (Audiffren, 2016). The RAH model is based on a basic

tradeoff between the allocation of metabolic resources. Thus, as individuals perform gross motor tasks (e.g. locomotion) the brain must shift its resources to the regions that control those movements such that it may then limit the available resources within other regions (specifically the PFC). This is the basis of the model, in that the deactivation of the PFC (supported by a reduction of neural activity) coupled with a decrement of regional blood flow, oxygenation and glucose uptake.

While a majority of the literature presented suggests a decrement of executive function performance during acute exercise, Lucas et al., Pesce and Audiffren, and Quelhas et al. have all shown positive effects (A. et al., 2013; Lucas et al., 2012; Pesce & Audiffren, 2011). The RAH model would posit that this is due to limitations of exercise duration, lack of high intensity domains and the potential of physical fitness as a moderator that may not facilitate a limitation of executive function performance. However, if the RAH model is to be accepted, even if altered, then it should be effective regardless of the physical fitness level of the participants, and may just occur at an increased relative level versus untrained individuals (Tempest et al., 2017). In theory, physical fitness status would facilitate a decrement of executive function performance during exercise at a higher relative (and absolute) intensity (Kahina Oussaidene et al., 2015).

The RAH model is not without limitations. A given brain region that is involved during the execution of concurrent cognitive task and exercise should facilitate an increase of cerebral blood flow within both the PFC and additional regions involved with that task (Audiffren, 2016). This principle, known as neurovascular coupling, would argue that this voluntary activation would facilitate an increase of activation (i.e. that an increase of neural activity is associated with an increase of blood flow to that region); the RAH model on the other hand, would consider a decrement of neural activity within a given region that is not critical to the concurrent tasks at

hand, and would do so in a top-down manner rather than a flexible one (Audiffren, Tomporowski, & Zagrodnik, 2008). However, it may be that these components are not mutually exclusive. Thus, there may be flexibility or a potential decrement, which may in part be due to a regional shift in activation which is directly related to neurovascular coupling. This ties into another potential limitation of the model. The ability to maintain effortful exercise (e.g. high intensity exercise) requires self-regulation, in part, by overcoming the urge to stop exercise or decrease the intensity at which the exercise is being performed (Audiffren & André, 2015). If, as the RAH model suggests, a simple top-down decrement beginning with the PFC, then this would potentially include sub-regions that oversee these self-regulatory processes. Thus, the current question is such that is there a decrement of executive function performance during exercise, and if so, when does that occur and is that occurrence linked with any specific markers that would contribute with this decline?

Physiological Responses to Exercise

Neurotransmitters

It has been well established that exercise presents a significant stressor upon the human body. In response to this stress, the endocrine and nervous systems work 'in concert' (Meeusen & Piacentini, 2001) so that movements and physiological processes are controlled. The ability of these systems to regulate and coordinate functions is, in part, influenced by the central dopaminergic, noradrenergic and serotonergic systems (Meeusen & Piacentini, 2001). The essence of the reticular activating process is the upregulation of the arousal systems, chiefly the noradrenergic and dopaminergic pathways (Audiffren, 2016). These systems facilitate the main amine neurotransmitters, including the catecholamines dopamine, epinephrine (i.e. adrenaline) and norepinephrine (i.e. noradrenaline). All three of these catecholamines are derived from tyrosine.

Their synthesis occurs with the catalyzation of tyrosine hydroxylase. This is also their rate limiting step, making it an integral criterion in determination of catecholaminergic neurons within the brain (Purves D, 2001).

Norepinephrine (NE) is synthesized by dopamine B-hydroxylase, which catalyzes NE from dopamine. Specifically, dopamine is transported by vesicles into the adrenergic terminals where it is then converted into NE. This conversion occurs within predominantly within sympathetic ganglion cells. Epinephrine (E) is present at lower levels within the brain and occurs via the synthesis of phenylethanolamine-N-methyltransferase which is only present within E secreting neurons. These neurons are found specifically within the rostral medulla (Purves D, 2001).

As previously mentioned, exercise is considered a significant stressor upon the human body. In response to this stress, catecholamines are released. NE is considered both a neurotransmitter and a hormone due to the ability of NE to be synthesized within nerve fibers as well as within the adrenal medulla. On the other hand, E is typically considered a hormone due to its synthesis and secretion occurring from the adrenal medulla (Zouhal, Jacob, Delamarche, & Gratas-Delamarche, 2008). This is significant, because the adrenal medulla is under sympathetic control (which is chiefly why NE and E increase during exercise) and this activity is typically via the sympathoadrenal system. It is then understood to consider NE and E as 'indexes' of sympathetic nervous system and adrenal activity (Zouhal et al., 2008).

In response to exercise, catecholamines are released and use their specific membrane receptors to facilitate their activity. NE primarily activates α-receptors while E primarily activates β-receptors. Stimulation of α-receptors constricts small blood vessels within the bronchial mucosa while relaxing the smooth muscles of the intestinal tract, while, on the other hand, β-receptors relaxes the bronchia smooth muscles, facilitating lung bronchi dilation and also increasing heart

rate and contractility (Zouhal et al., 2008). An overarching view is that catecholamine responses (specifically NE and E) stimulate respiratory, cardiac, metabolic and thermoregulatory mechanisms.

Dopamine, the third of the three catecholamines, is produced primarily within the corpus striatum, specifically within the caudate and lentiform nuclei. The corpus striatum receives input from the substantia nigra. The substantia nigra plays a vital role for the coordination of human movement. The dopaminergic cells at the substantia nigra send their output up to the caudate. Once dopamine is released from the caudate, it binds to dopamine receptors as well as β-adrenergic receptors facilitating dopamine as a neurotransmitter, playing an important role within the sympathetic ganglia.

The Prefrontal Cortex and Catecholamines

In terms of cognitive processes, it's likely that catecholamines exhibit an impact on cognition, especially NE, as it is released to the hippocampus via the locus coeruleus (LC). The LC is the largest source of NE release within the brain, as it houses multiple noradrenergic nuclei within it. The LC has widespread projections throughout the brain, but, specifically has projections into the PFC (Audiffren, 2016). The LC, as the main component of the brain to release NE, is unique in that it also receives input from the forebrain regions including the PFC. The integration of the PFC and LC theoretically allows for top-down regulation of arousal via the noradrenergic system. Regulation of arousal from a stressor via NE is complex and it may be differentially recruited under varying conditions which may include concurrent exercise and cognitive tasks. Previous findings have shown that 'low' and 'excessive' levels of NE may be associated with impairments within working memory while moderate levels have been associated with effective working memory (Robbins & Arnsten, 2009)

Upon its release, NE can bind to three receptor families (α-1, α-2 and β). Primarily, it's been thought that the α-1 and β receptors are postsynaptic receptors while the α-2 can operate both as pre- and post-synaptic receptor (Berridge & Spencer, 2017). Data has indicated that α-1 and β receptors are necessary for stress related arousal which suggests that NE is an integral neurotransmitter by its modulatory effects within the stress response to exercise (A. F. T. Arnsten, 1998 Spencer, 2015 #234).

Thus, in the case of moderate levels of NE and facilitated working memory, the supposition is that NE is released and binds with α-2 receptors within the PFC to support working memory; and that this is degraded at extreme levels of NE due to the binding of α-1 receptors (Robbins & Arnsten, 2009) while impaired working memory has been associated also with low NE release and signaling to the PFC, which may be reversed due to the activation of α-2 receptors within the PFC, further supporting their implication as post-synaptic receptors that support working memory (A. F. T. Arnsten, 1998).

Another main executive function, cognitive flexibility, has also exhibited similar findings as working memory in terms of NE expression. Specifically, depletion of NE within the PFC has been linked with decrements of cognitive flexibility via set shifting tasks (McGaughy, Ross, & Eichenbaum, 2008). This may in part be due to evidence suggesting that α-1 receptor activation is linked with improved set shifting (Spencer, 2015) acting potentially as a key modulator of NE's influence on cognitive flexibility while α-2 and β receptors may exhibit a minimal effect of NE expression and cognitive flexibility (Lapiz & Morilak, 2006). It appears that NE may exhibit an inverted-U function on both working memory and cognitive flexibility, though the response process may be based differently upon rate of NE release and receptor. This inverted-U response may alter the improvement or impairment of executive function via the PFC. Thus, it may be an

oversimplification to classify NE simply as an 'excitatory' or 'inhibitory' neurotransmitter but instead consider that NE is a neuromodulator that can exhibit diverse actions depending on rate of release, level of concentration and receptor affinity within a specific region of the brain as well as systemically.

Concerning catecholamine effects at difference exercise intensities, prior findings have also shown an inverted-U function, very similar to NE, such that the catecholamines in summation seems to operate in response to cognition during exercise similarly (Audiffren, 2016; Cools & D'Esposito, 2011). The impairment of the PFC during concurrent cognitive and physical demands may occur due to the following: 1. NE levels are either too low or high 2. DA is either to low or too high and 3. A decrement in cognitive function during concurrent cognitive and physical demands via RAH mechanisms, meaning that at higher exercise intensity may be altered due to catecholamine concentrations (Amy F. T. Arnsten, 1998; Audiffren, 2016).

Cortisol

Cortisol is the main glucocorticoid hormone in the human body and it has both acute and chronic implications. Cortisol is produced by the adrenal cortex, released in response as the final product of the hypothalamic-pituitary-adreno-cortical (HPA) axis (Henckens, van Wingen, Joels, & Fernandez, 2012). The HPA axis consists of several structures within and outside of the central nervous system that together facilitate hormone responses to the energetic demands of the body, mainly that of stress. The first hormone released, corticotropin-releasing hormone (CRH) is released from the paraventricular nucleus in the hypothalamus where it is secreted across the anterior pituitary where it binds with its target receptors to stimulate adrenocorticotropic hormone (ACTH). ACTH is released into the blood where it binds to its receptors on the adrenal cortex. At this point, the adrenal cortex then secretes cortisol in response to this binding. Typically, one can

describe cortisol's actions as a facilitator of metabolic regulation such that it stimulates different components to increase the availability of glucose. In the periphery one can see this as an increase of gluconeogenesis and lipolysis, while in the brain cortisol can cross the blood brain barrier. Within the brain, cortisol can bind to either type I (mineralocorticoid) or type II (glucocorticoid) receptors. Type I receptors are predominately within the amygdala, hippocampus and prefrontal cortex while type II are diverse throughout the cerebrum. It is plausible that the diverse nature of type II receptors is what allows for cortisol to bind up to 10-fold versus type I receptors and plausible to understand the myriad of effects of cortisol as a stress hormone.

It's widely accepted that elevated resting levels of cortisol is an indicator of chronic stress that may have a negative impact upon performance (Taverniers, Van Ruysseveldt, Smeets, & von Grumbkow, 2010). Furthermore, it's been shown that chronic elevated levels of cortisol can downregulate the HPA axis which leads to impairments in stress coping and cognitive function including executive function (Chen et al., 2017). Various forms of exercise increase cortisol levels such that these elevations correlate as an intensity duration relationship (higher and longer) (A. C. Hackney & Viru, 1999; Hill et al., 2008). Thus, as Chen et al. have termed the 'exercise-glucocorticoid paradox' it seems that exercise modulates the stress response, thereby potentially improving both exercise and cognitive performance while limiting potential downregulation of processes indicative of cognitive and physiological impairments (Chen et al., 2017).

Acute exercise >60% of VO_{2max} exhibits a significant secretion of cortisol in trained individuals (Bloom et al., 1976) while others have seen these significant increases ~80% of VO_{2max} while seeing a reduction in cortisol at lower exercise intensities (Hill et al., 2008). These findings suggest that there may be intensity and training factors that alter cortisol's effect on the body,

although some data has concluded that higher elevations in cortisol may improve acute performance (Morgan III et al., 2001).

To determine the effects of cortisol response at different exercise intensities, Hill et al. assessed 12 recreationally trained men to perform 30 minutes of cycling at either 40, 60 or 80% of their VO_{2max} (Hill et al., 2008). Controlling for diet, circadian rhythms and time of day, which are all known to be largely variable in cortisol's response, the results exhibited that only the 60 and 80% conditions evoked significant increases in cortisol, which are akin to previous findings (Browne et al., 2017).

Hackney et al. investigated the cortisol responses to both moderate and high intensity exercise throughout a 24-hour period. At running or cycling either at moderate (60 minutes of aerobic work) or high intensity (intervals of 2-min), cortisol levels were found to be significantly elevated in both conditions for up to 3 hours post-exercise in the morning but only immediately post-exercise in the afternoon (A. C. Hackney & Viru, 1999). Additionally, both conditions exhibited significantly lower levels throughout the night compared to the control condition. Interestingly, the high intensity condition also exhibited significantly elevated levels when compared against the moderate group in the morning but not afternoon while exhibiting significantly lower levels throughout the night when compared against the moderate intensity group. This landmark study alludes to the intensity and duration effects of cortisol and allude to the potential element of increased levels of cortisol at higher intensities over duration, suggesting that potentially intensity may be the greatest contributor to cortisol responses.

It also appears that physical fitness and training status may also play a role in cortisol response to acute exercise. A study by Luger et al. compared untrained versus trained runners, both completing treadmill running at 50, 70 and 90% of VO_{2max} (Luger et al., 1987). Their findings

showed that at each stage trained runners had a higher overall cortisol level, but post-exercise revealed that the trained runners also exhibited attenuated levels of cortisol compared with the untrained group.

In terms of cognitive performance, specifically that of executive function, is not elucidated as to the impact of cortisol levels. Cortisol levels have been previously measured during a task of working memory (McCormick, Lewis, Somley, & Kahan, 2007). Specifically, salivary cortisol was measured four times throughout a working memory task during submaximal exercise. Their findings exhibited that cortisol levels possessing an individual response, with the only moderate correlations between working memory task scores and cortisol levels ($r = -0.25$ to -0.35) for women while the men saw non-significant correlations across testing time points (McCormick et al., 2007). However, this task was done in undergraduate students and was completed during a resting condition without exercise. A study by Elzinga et al. exhibited different findings (Elzinga, Bakker, & Bremner, 2005). 16 college aged females underwent working memory tasks pre- and post-stress challenges. Their findings showed that participants improved their working memory after exposure to a mentally stressful challenge while seeing a significant increase in cortisol response (41%) during the stress exposure. This suggests that cortisol may improve working memory tasks when done at rest or during cognitively stressful scenarios.

Schmidt-Kassow et al. examined the relationship between cortisol and working memory during treadmill walking. Their results exhibited that improved working memory was positively correlated with elevated levels of cortisol during the walking condition but not during the resting condition (Schmidt-Kassow et al., 2014). Contrary to these findings, Taverniers et al. examined the cortisol responses in Special Operations candidates during either a controlled (60 minutes of aerobic exercise) or a simulated high stress scenario like the United States' Military's Survival,

Evasion, Resistance and Escape (SERE) school (Taverniers et al., 2010). The high stress group exhibited significantly greater cortisol responses and lower (decreased) working memory task scores. Both studies present different limitations. Schmidt-Kassow estimated their percentage of effort with a target of approximately 50% of VO_{2max}, which they denote as lower than previous investigations. Additionally, they utilized short term memory via learning polish language (Schmidt-Kassow et al., 2014). On the other hand, the Taverniers study did not identify intensity domains of either condition or quantify the level of induced stress to garner a potential 'level' of which decrements in EF occurred (Taverniers et al., 2010). Additionally, tests were conducted pre- and post-exposure and were not conducted during either condition.

During exercise cortisol is released, and it's thought that during its increase in secretion it modulates arousal by limiting CRH and ACTH as a negative feedback loop. However, as exercise intensity (or duration) increases cortisol becomes unable to inhibit both CRH and ACTH which then in turn may potentially limit cognitive performance (Lambourne & Tomporowski, 2010). This may be plausible as it's been shown to have an inverted-U effect similar to that of NE and E which may also be representative of the findings in Taverniers et al. (Lupien & McEwen, 1997; Taverniers et al., 2010).

This sharp conflict in information may play a key role in understanding potential performance variables and that cortisol may play a significant role within exercise and cognition. Specifically, two questions arise with the present hypothesis. First, is the inhibition of CRH and ACTH a negative factor that may degrade executive function at higher exercise intensities and is its initial increase correlated with enhancements in executive function?

Additional research has revealed that cortisol has been linked with brain derived neurotropic factor (BDNF) in that they both have an impact upon the neurogenesis of the brain

(Rojas Vega et al., 2006). This has been shown, as previously stated, that chronically elevated levels of cortisol have been linked with chronic stress. In addition, this chronic stress due to increased cortisol levels has also been associated with the dysfunction of neural plasticity and neurogenesis and thus, lower levels of BDNF (Kishino et al., 2001).

Brain Derived Neurotropic Factor (BDNF)

Brain Derived Neurotropic Factor (BDNF) is one of the main neurotropic, or growth, factors present within the human body. BDNF serves as a modulator of neuronal and synaptic growth where it exhibits its effects chronically and systemically within the brain where it impacts cognitive performance (Piepmeier & Etnier, 2015). Since BNDF can cross the blood brain barrier, it's assumed that peripheral circulating BDNF is transported into the brain where it contributes to neural plasticity (Pan, Banks, Fasold, Bluth, & Kastin, 1998). This link of BDNF expression is thought to be increased through exercise such that it acts throughout the brain (though it's been noted to show the largest amounts within the hippocampus). The hippocampus sits above the medial temporal lobe and facilitates memory, learning and emotions. Many neurons within the hippocampus that project to the PFC with evidence exhibiting the relationship between the PFC and the hippocampus. Thus, it's suggested that the PFC and the hippocampus work in concert to facilitate working memory, one of the central executive functions.

BDNF has previously been positively linked with exercise, such that higher intensity exercise exhibits the greater increases in BDNF levels (Ferris, Williams, & Shen, 2007; Griffin et al., 2011; Schmolesky, Webb, & Hansen, 2013). This is key as BDNF signaling is a requisite for hippocampal plasticity (Cotman, Berchtold, & Christie, 2007). Specifically, exercise facilitates plasticity to occur by enhancing the potentiation of both high frequency and theta stimulation within the dentate gyrus, which reduces the theta stimulation threshold required to induced long-

term potentiation which is the synaptic component of learning and memory (Farmer et al., 2004). To confirm BDNF's implication in learning and memory, previous research has blocked the signaling of BDNF by infusing anti-TrkB antibodies (TrkB is the receptor for BDNF) resulting in downregulation in learning and memory (Vaynman, Ying, & Gomez-Pinilla, 2004) further suggesting that BDNF must be present for learning and memory to occur in the hippocampus.

The hippocampus and PFC have long been known to have associations (Jin & Maren, 2015). Specifically, several forms of projections between the two structures have been identified, including both mono and polysynaptic projections that primarily project into the medial portion of the PFC. This pathway has shown to implicate working memory such that the interaction between the hippocampus and PFC is critical for performance of complex tasks (Baddeley, 2003) and this activity can occur from the HPC to the PFC and vis versa (Jin & Maren, 2015).

The responses of BDNF to exercise has previously been examined. In a 2016 study, 10 males underwent a high intensity interval exercise protocol on a treadmill running at 100% of the VO_{2peak}. Participants ran 1-minute interspersed with 1-minute of passive rest, participants ran this interval until either 1.25 or 2.5 kilometers in distance was reached. Assessment of BDNF exhibited significant increase from pre- to post-exercise, however, returned to basal levels within 1-hour post-exercise.

BDNF has been examined in learning (Hotting, Schickert, Kaiser, Roder, & Schmidt-Kassow, 2016) and memory (Griffin et al., 2011), however, there is conflicting evidence as to BDNF's role within executive function during exercise. Hotting et al. examined the effects of exercise on learning and memory during exercise (Hotting et al., 2016). In their study, 81 college aged participants (41 female) completed either a high (80% HR_{max}) or low (<75% HR_{max}) for 30 minutes after learning words from a different language. Their results exhibited significant

increases in BDNF during the high intensity group showing a positive correlation between BDNF and memory retention, which is a large component of HPC function. However, this was not seen immediately post-exercise, as immediately post-exercise revealed a decrement in acute memory (less words were recalled). In turn, 24 hours later the high intensity group recalled significantly more words compared with the control group. This suggests that BDNF's implications on the impact of cognition during exercise may be delayed and exhibit more of a chronic effect.

In terms of acute effects of BDNF on exercise and executive function, Ferris et al. examined BDNF during two different cognitive tests during exercise, one aimed at general cognition and the other focusing on executive function (Ferris et al., 2007). Participants completed 30 minutes of cycling at either 10% above or 20% below ventilatory threshold. To determine the effects of BDNF on acute exercise, participants took an executive function task prior to and post exercise. Their results indicated that during the graded exercise test, BDNF levels rose significantly and that while executive function task scores increased, it did not result in significant increases and thus was not strongly correlated with BDNF levels.

To assess executive function, Griffin et al. had 47 college aged males performed a maximal graded exercise test. They were assessed by general cognition that was followed by the Stroop Word-Color Task, commonly used as a measure of executive function. Their findings showed significant increases in BDNF from pre- to post-exercise ($p < 0.05$) there was not any significant differences in the Stroop task during this same testing time points ($p > 0.05$) (Griffin et al., 2011). A second experiment within their study examined the effects of 3 and 5-week training regimens on BDNF levels and general cognition. These findings exhibited significant increases in BDNF in both training time points, with both eliciting increases in BDNF that were sustained up to 90-minutes post exercise at the end of both the 3rd ($p < 0.05$) and 5th ($p < 0.05$) weeks of training

(Griffin et al., 2011). In comparison, only the 5-week time point revealed significant improvements in the general cognition task ($p < 0.05$) when compared with the pre-test assessments.

A more recent study conducted by Hung et al. examined the effects of task switching (a form of cognitive flexibility) and serum levels of BDNF (Hung, Tseng, Chao, Hung, & Wang, 2018). 20 college male students underwent 30 minutes of running or badminton at 60% HRR. Their assessment of task switching and BDNF levels were assessed prior to and post-exercise. Their findings revealed significant increases in BDNF levels for both groups ($p < 0.05$) with badminton exhibited significant elevations compared with the running group ($p < 0.05$). While it was non-significant, the badminton group showed trends toward significance in global switch costs ($p = 0.052$) indicative of inhibitory control and cognitive flexibility (Hung et al., 2018).

There are limitations to the previous studies that may potentially alter the findings of BDNF, cognition and exercise. These limitations include small sample sizes which may have affected the statistical outcomes. Additionally, these studies either did not assess executive function domains during exercise and have not been under standardized intensity domains and while it can be deduced that BDNF is increased during acute moderate and high intensity exercise. While its relationship with executive function has yet to be elucidated, it's role in exercise intensity and potential enhancement of cognitive components make it a measure that needs to be assessed to determine its relationship

Blood Lactate

It has well been established that lactate is the product of glycolysis (George Austin Brooks, Fahey, & White, 1996). During exercise, working muscle can utilize lactate in several ways, including the Cori Cycle, as well as intra and intercellular lactate shuttles (G. A. Brooks, 2007). At lower exercise intensities, blood lactate levels remain stable as part of the glycolytic flux rate

(George Austin Brooks et al., 1996). This is due to the oxidization of lactate by the muscle cells and tissues, keeping its production, oxidation and removal in balance. In effect, this represents the cardiorespiratory systems ability to meet the demands of the active muscle. However, as exercise intensity increases the rate of lactate concentration begins to increase which exceeds its rate of utilization and removal. This inflection point has been termed the Onset of Blood Lactate Accumulation (OBLA) (Chmura, Nazar, & Kaciuba-Uścilko, 1994).

Lactate not only exerts its effects peripherally within muscle tissues and cells but also exists and is utilized within the brain (G. A. Brooks, 2007). Specifically, it's been suggested that astrocytes, which are non-neuronal glial cells in the brain, provide the basis for cellular signaling. This occurs similarly to the shuttles that exist within muscle, such that an exchange between the production and removal of lactate occurs via monocarboxylate transport proteins (MCTs) (G. A. Brooks, 2007). Specifically, MCT1 acts as a primary transporter between the mitochondria allowing lactate to be oxidized within the cell (G. A. Brooks, 2007).

To establish lactates role as an energy source for the brain, Van Hall et al. infused lactate systemically to identify its ability to be utilized within the brain, assessed by catheter insertion (Van Hall et al., 2009). By infusing lactate, the researchers were able to control the amount of lactate increased throughout the infusion period and identify the amount of lactate and glucose uptake and utilization at rest and during exercise. Their results exhibited that lactate into the brain was completely oxidized and further exhibited that this utilization resulted in net cerebral lactate oxidation of approximately 25% of that total energy demand during exercise (Van Hall et al., 2009). Thus, during exercise, the brain is activated which is often denoted by an increase in both neuronal and blood flow activity. This enhancement occurs such that an increase of the requirement for metabolites to assist in this demand must also increase.

Hashimoto et al. (2017) aimed to determine the effects on acute exercise bouts on lactate and its potential correlation with executive function by assessing executive function pre- and post-exercise (Hashimoto et al., 2017). 14 college-aged males performed two acute bouts of high intensity interval exercise (HIIE) on a recumbent bike. These intervals were performed in the following fashion: 4 minutes of cycling at 80-90% of their maximal power output (pre-determined by an initial maximal exercise test) followed by 3 minutes at 50-60% of power output. Rest was 60 minutes in duration and split these two bouts, with executive function and physiological measurements occurring in 10-minute increments prior, in-between, and post-exercise.

Their findings exhibited improved executive function (via the Word Color Stroop Task) from pre-test to post-HIIE 1 ($p < 0.01$) that was elevated for 40-minutes between bouts ($p < 0.05$) (Hashimoto et al., 2017). Executive function was also significantly increased from pre-test to post-HIIE 2 ($p < 0.05$) though there was no significance between bouts ($0 > 0.05$). Interestingly, lactate uptake significantly increased from pre- to post-HIIE 1 but was not significantly different from HIIE 1 to HIIE 2(Hashimoto et al., 2017). While we have discussed in previous sections BDNF and catecholamines, this article is interesting in that it also examined these measures in conjunction with lactate. Similar to Van Hall (2009), Hashimoto et al. also utilized a retrograde catheter into the right jugular vein to assess brain lactate uptake. This, in combination with BDNF and catecholamine response, exhibited that BDNF was significantly increased post-HIIE 1 ($p < 0.05$) and post-HIIE 2 ($p < 0.05$) but was not significantly different from HIIE 1 to HIIE 2 ($p > 0.05$). Additionally, the a-v difference of lactate was lower post HIIE 2 versus HIIE 1, showing a similar trend as executive function and BDNF. The authors suggest that several of these interacting factors may modulate cerebral metabolism which in turn may alter executive function after bouts of HIIE (Hashimoto et al., 2017).

The activation of the brain is multifactorial in nature. A combination of feedforward and feedback signals integrate to sustain exercise and cognitive control. This activation is seen as an increase in cerebral blood flow expressed as an enhancement of oxygenation. This activity of the brain is contrary to what occurs within the muscle, as throughout the exercise intensity spectrum, a decrement of muscle oxygenation can often be seen. What is intriguing, is that a reduction of cerebral oxygenation has been seen to occur at higher exercise intensities, though it has not always been correlated with a decrement in executive function (Quistorff, Secher, & Van Lieshout, 2008). The interaction of lactate coincides and is related to glucose kinetics, as it's posited that lactate as a fuel in part acts to spare glucose. In fact, during exercise, the brain may take up as much as 15-25 mmol of lactate, which has been shown to enhance executive function per aforementioned studies. While it's yet to be elucidated as to the specific mechanisms which may control the blood brain barrier that allows for the transport of lactate, it's been suggested that it may be coupled with β-2 receptors, which as previously discussed, is a receptor for epinephrine (Quistorff et al., 2008).

Previous research has shown that certain aspects of cognition show a biphasic pattern in their response, specifically that reaction time as a surrogate for improved cognition is enhancing between 60-80% of 'maximal workload', while any intensity domain above this is then associated with a decrement in cognitive performance (Chmura & Nazar, 2010). Chmura et al. have established this domain as the 'threshold of psychomotor fatigue' (Chmura et al., 1994). In their more recent investigation, they aimed to determine if choice reaction time also exhibits a similar response in trained athletes (Chmura & Nazar, 2010). 13 professional soccer players (VO_{2max} 57.2 ± 1.6 ml/kg) underwent a stage maximal treadmill test (6km/hr. with an 2km increase every 3-minutes) and choice reaction time tests during the last minute of each stage. Their findings exhibited the greatest cognitive performance denoted as the shortest reaction time at ~87.9% of

maximal workload (15.8 ± .17 km/hr.). Additionally, this correlated on average 2% above the onset of blood lactate accumulation (OBLA). However, there were no statistically significant interactions of accuracy of responses ($p > 0.05$) throughout the test (Chmura & Nazar, 2010).

This threshold is a very intriguing concept as it relates to the RAH theory. The workload level of the OBLA is higher than the 'breaking point' of the anaerobic threshold that has been previously seen (Chmura et al., 1994) and may also coincide with the concept of critical power such that the transition from tolerable to severe intensity domains may indicate the onset of anaerobic dominance such that peripheral fatigue presents itself as an accumulation of hydrogen, inorganic phosphate and rise in lactate. If this is the case, then what happens to executive function and does the brain operate in a similar fashion as skeletal muscle in this regard? Specifically, does the alteration of metabolite concentrations associated with this workload level attenuate executive function during exercise?

Cerebral Oxygenation

The most basic concept of brain activity arises from two components, oxygenation and electrical activity. From a metabolic standpoint, oxygenation presents the ability of blood to deliver both oxygen and glucose within the brain. Prior investigations had concluded that cerebral blood flow increases from rest to moderate exercise intensities, including from the decline in oxygenated hemoglobin (HbO_2) (Kahina Oussaidene et al., 2015), the respiratory compensation point (Tempest, Eston, & Parfitt, 2014) with no further increase of oxygenation from moderate to VO_{2max} (González-Alonso et al., 2004; Tempest et al., 2014) while others have shown cerebral oxygenation decline at high to maximal intensities (E. E. Smith & Jonides, 1999; A. W. Subudhi, Miramon, Granger, & Roach, 2009a; Andrew W. Subudhi et al., 2011). To understand potential interactions that may be exhibited by no further increase of oxygenation, Gonzalez-Alonso et al.

examined cerebral hemodynamics across exercise intensity domains (rest to maximal exercise) (González-Alonso et al., 2004). Brain activity seems to be enhanced by exercise (Dalsgaard et al., 2004). However, the question arises is oxygen uptake is compromised during maximal exercise.

During exercise, systemic circulation via blood flow occurs to deliver oxygen and nutrients while also removing metabolites. A secondary mechanism of circulation is blood pressure regulation to ensure that perfusion pressure is met and regulated. As exercise commences, the autonomic nervous system increases its output via cardiovascular (heart rate, cardiac output), metabolic and sympathetic output all increase in different magnitudes. The cerebral oxygenation response has traditionally been defined as biphasic, such that from rest to ~ 60% VO_{2max} followed by either a leveling off or decline as intensity increases. A recent review points to potential relationships between peripheral hemodynamics during low to moderate intensity exercise while high and maximal exercise intensity may be coupled with peripheral feedback mechanisms, including changes within arterial blood gases (Rooks, Thom, McCully, & Dishman, 2010)

To assess this, Gonzalez-Alonso et al. examined cerebral hemodynamics in both a control and heat stress condition to determine if brain metabolism becomes impaired such that it elicits a decrement in oxygen delivery and uptake, specifically at maximal exercise and whether fatigue (facilitated sooner via heat stress condition) alters these brain metabolic reactions (González-Alonso et al., 2004). Conducting two maximal tests on a cycle ergometer in either a control (normal skin and core temperature) or hot (skin and core temperature) conditions, participants cycled at a rate which elicited VO_{2max} within approximately 5 minutes, reaching 'max' within 10 minutes. Findings exhibited that while heat stress accelerates brain extraction of oxygen, glucose and lactate, there is no alteration of brain activity at exhaustion (reflected in changes of both oxygenation and blood flow). Furthermore, what's seen is that metabolism (oxygen, glucose,

lactate) extraction within the brain increases across exercise intensity despite oxygen (and glucose) delivery decrements, such that metabolism has a linear increase with an increase in exercise intensity and the extraction increases to compensate for the decrease in substrate delivery (González-Alonso et al., 2004).

To determine specific brain regions and how they may be affected by exercise, Oussaidene et al. examined the oxygenation responses of the prefrontal cortex in both trained and untrained individuals. Utilizing a max protocol on a cycle ergometer, the researchers monitored oxygenation via NIRS, specifically looking at the left prefrontal cortex region (Kahina Oussaidene et al., 2015). Trained versus untrained men exhibited similar outcomes, such that both groups exhibited an increase of oxygenation up to a 'threshold' followed by a significant decline ($p < 0.05$). Similarly, this threshold, which they term the cerebral oxygenation threshold, occurred at a similar VO_{2max} and W_{max}. However, total change in oxygenation increased in the trained ($p < 0.05$), but not the untrained group ($p > 0.05$) (Kahina Oussaidene et al., 2015).

To determine regional differences of cerebral oxygenation Tempest et al. examined 8 regions within the prefrontal cortex (dorsal and ventral) and their responses during a maximal ramp protocol on a cycle ergometer in untrained individuals (Tempest et al., 2014). 25 recreationally trained participants (23 males) completed an incremental maximal ramp protocol on a cycle ergometer (increasing 20W/minute). Assessments of prefrontal cortex oxygenation were examined throughout warmup, exercise and cessation of exercise.

Their findings presented the change in oxygenation was greater in ventral rather than dorsal regions up to respiratory compensation point, (hyperventilation) while they both remained stable from the respiratory compensation point to max, save for region 4 (ventral region) which increased while region 1 (dorsal) declined. In addition, the change in deoxygenation of hemoglobin (ΔHHb)

was stable to the ventilatory threshold, and increased from the VT to max, while total blood volume increased throughout exercise.

The alteration of the amount of oxygen present may also play a role in alteration of cerebral oxygenation. To assess this, an investigation by Subudhi et al. (2009) examined prefrontal, premotor and motor cortices oxygenation during two bouts of incremental maximal exercise protocols (A. W. Subudhi et al., 2009a). 23 participants underwent a ramp protocol (25W/min) on a cycle ergometer under during hypoxic and normoxic conditions. To accurately determine the effects of exogenous oxygen on cerebral oxygenation, these two conditions were blinded and counterbalanced. Under the hypoxic condition, prefrontal oxygenation was reduced by 11.1 ± 14.3% at rest ($p < 0.01$) and was further attenuated at maximal exercise intensity (26.5 ± 19.5%, $p < 0.01$). While correlations between regions in the hypoxic condition were high ($r^2 = 0.61$), deoxygenation was greater in prefrontal rather than premotor and motor regions ($p = 0.05$). In the normoxic condition, bilateral changes occurred for oxygenated and deoxygenated hemoglobin as well as total hemoglobin were highly correlated ($r^2=0.94$, $p < 0.01$) throughout exercise intensities, save for max, which saw an increase of oxygenated and total hemoglobin for the right, but not left, prefrontal region ($p < 0.05$).

The authors attributed their findings of higher cerebral deoxygenation in multiple brain regions, including the motor cortex, which may have been exhibited by an overall lower power output at max (A. W. Subudhi et al., 2009a). It is interesting to note, that overall tissue saturation index (TSI), which is the global value of cerebral oxygenation via NIRS, was not significantly affected in the normoxic condition until 75% of maximal power output. However, about 75%, there was a significant reduction in TSI ($p < 0.05$) (A. W. Subudhi et al., 2009a). These findings under normoxic conditions support previous findings and add to the literature suggesting that

oxygen may limit prefrontal and motor cortex oxygenation throughout exercise. This is of importance, as it seems that there may be a relationship between oxygen availability within the brain and overall exercise performance.

To determine the effect of cerebral oxygenation may have on cognition during exercise, 12 male participants underwent two different exercise conditions; one control condition cycling only at 20 watts while the experimental bout consisted of stages at 40, 60 and 80% of VO_{2max} (Ando, Kokubu, Yamada, & Kimura, 2011). Each stage lasted 6 and a half minutes and the cognitive test, the flanker task in this study, was assessed 3 minutes into each stage (Ando et al., 2011). Across each exercise stage, HHb increased significantly ($p < 0.05$) while the control condition saw significant decrements from the 2nd to 3rd stage ($p = 0.05$). Interesting of note is that total hemoglobin content ($p > 0.05$) or error rate ($p > 0.05$) in the cognitive task did not significantly change during any time point in either condition (Ando et al., 2011). However, the reaction time (denoted as premotor time) decreased from rest to the 60% stage ($p < 0.05$). The authors suggested that deoxygenation occurs at higher intensities, indicative of an increase in cerebral activity while no significant changes in the cognitive task performance may suggest that increased prefrontal activation may support test execution at higher exercise intensities (Ando et al., 2011).

In a more recent study, Schmit et al. (2015) observed the effects of concurrent cognitive demands while cycling at high intensity (Schmit et al., 2015). 15 untrained participants (10 men) underwent initial ramp cycling max test (10W/30s) to obtain maximal exercise. Participants then underwent a control condition consisting of the cognitive test, and in counterbalanced fashion, an experimental condition cycling at 85% of their maximal aerobic power while undergoing 20 minutes of a cognitive task (modified Erikson Flanker Task) (Schmit et al., 2015). Two data collection zones occurred: the initial period of the first 100 trials and the terminal period, the last

100 trials performed before the cessation of the protocol. Utilizing electromyographic (EMG) activity of the hands to analyze the reaction time to the given task allowed the researchers to obtain pre-motor time, similar to that of the Ando et al. study (Ando et al., 2011; Schmit et al., 2015).

Their analyses revealed longer reaction times for incongruent trials ($p < 0.001$), frequency of errors ($p < 0.01$), and partial EMG errors ($p = 0.01$) from the initial to terminal periods for the exercise condition. Oxygenated hemoglobin decreased linearly from the initial to terminal periods ($p < 0.05$) in the exercise condition while cerebral oxygenation also significantly dropped during the terminal period when compared against the same time-point of the control condition ($p < 0.05$) (Schmit et al., 2015). These data suggest that a relationship between decreased oxygenation may impact the effect of cognition during exercise.

To assess the impact of executive function, Tempest et al. (2017) found that working memory (2-back test) and incongruent trials of the Erikson Flanker Task significantly decreased across 60 minutes of cycling at 10% above ventilatory threshold (Tempest et al., 2017). Particularly interesting is that cerebral oxygenation increased during the first 3 stages of the 60 minutes ($p < 0.05$) but stabilized throughout the duration of exercise in the prefrontal cortex, but not the motor cortex (Tempest et al., 2017). Conversely, Ando et al. (2013) observed that executive function (via the Erikson Task) was improved during 10 minutes of cycling at 60% of VO_{2peak} while cerebral oxygenation decreased throughout exercise ($p < 0.001$) (Ando et al., 2013).

Taken together, these data suggest that exercise intensity may very well be a moderator for executive function during exercise observed by changes in cerebral oxygenation. It appears, however, that while there may be a biphasic response to oxygenation under different exercise intensity it may occur under different scenarios. For instance, as previously stated the operational definitions of intensity zones differed between studies. Schmit et al. (2105) utilized maximal

aerobic power; Ando et al. (2013) VO_{2max} percentage and Tempest et al. (2017) assessed exercise intensity based on ventilatory threshold (Ando et al., 2011; Schmit et al., 2015; Tempest et al., 2014). These differing intensity domains coupled with different cognitive tasks makes it difficult to operationalize the effects of cognition during exercise and the impact of cerebral oxygenation. Lastly, the fitness level of the participants varied throughout studies. If we are to take the findings from Oussaidene et al. (2015) it would suggest that trained individuals present a different oxygenation response at higher exercise intensities and that may be presented when combined with concurrent cognitive demanding tasks based around executive function (Kahina Oussaidene et al., 2015).

The hypothesis for the proposed study would lean on tenants from the above findings that may allude to the RAH theory. Specifically, cerebral oxygenation will increase from low to moderate exercise intensities that may be coupled with enhancement in executive function while at a potential intensity 'threshold' cerebral oxygenation will either level off or decrease which may be indicative of potential decrements in executive function.

Temperature Regulation

Only a fraction of the chemical energy consumed ends up converted and used as mechanical energy. In fact, Whipp and Wasserman determined that in cycling, one of the most physically efficient tasks, approximately 20% of converted energy is utilized for external work (Whipp & Wasserman, 1972). This heat within the muscle occurs through the metabolic process, which facilitates the ability for the muscle to contract and relax as the basis of human movement. Thus, as the metabolic rate of the muscle increases from exercise, it also increases the amount of heat release. While the temperature of the muscle cell vastly exceeds that of the center of the body, it does facilitate an overall increase of temperature which is denoted by increases in both core and

skin temperatures (Kenny et al., 2003). This is due to the muscle releasing heat through both conductive and convective mechanisms between the working muscles and the blood as well as surrounding tissue and compartments.

These physiological factors have been the basis for overall heat production, and therefore elevated skin and core temperature serving as modulating factors of exercise performance (Flouris & Schlader, 2015). Specifically, the body's ability to balance the rate of metabolic heat production leading to temperature balance is pushed to its limits as seen as increases in both skin and core temperature. Core temperature is typically ~37° C and is very tightly regulated (35-41°C). During incremental exercise, this temperature is raised and in response, the brain, skin, and blood vessels possess receptors that send afferent signals in response to thermoregulatory centers in the hypothalamus (Periard, Racinais, & Sawka, 2015). These centers are integrated with receptors combines to affect sweating and blood pressure responses. These receptors can be both behavioral and physiological.

From a behavioral perspective, one perceives these changes in thermal responses within the body as affective (comfort and pleasure) and discriminative (sensation). Thermal sensation is dictated by skin temperature and is largely independent of core temperature (Flouris & Schlader, 2015). Temperature is sensed by transient receptor potential (TRP) channels. Previous work has shown that TRP V3/4 may be large contributors to the sensation of increased thermal stimuli (Caterina, 2007). The ability of the brain to sense these changes occurs in the upper brainstem where thermoregulatory centers reside. Interestingly, and where the impact of potential cognitive factors may be altered due to changes in thermoregulatory patterns is that the brainstem combines the sympathetic and parasympathetic tracts. This may alter catecholamine concentrations (Malcolm, Cooper, Folland, Tyler, & Sunderland, 2018) as well as elicit changes in cerebral

oxygenation (Nielsen & Nybo, 2003). While it's generally accepted that large increases in thermal stress (typically seen as exogenous heat and/or humidity) (Flouris & Schlader, 2015), temperature regulation concerning cognition and exercise is less understood.

Passive heating is an often-utilized method as it attempts to parse out the effect of exercise on cognition (Taylor, Watkins, Marshall, Dascombe, & Foster, 2016). In a 2011 study, Gaoua et al. examined the effects of passive heating on attention and memory task performance. 16 participants (11 men) underwent a control condition (20°C, 40% relative humidity (RH)) and experimental (50°C, 50% RH) conditions (Gaoua, Grantham, Racinais, & El Massioui, 2012). A third condition mirrored the experimental and added head cooling as an attempt to determine if sensory input altered cognition after this acute bout of passive heat exposure.

Utilizing the Cambridge Battery, the researchers examined seven different attention and memory tasks (Gaoua et al., 2012). After the acute bout of passive heat exposure, their data exhibited no significant differences in any of the attention tests in both reaction time and accuracy between conditions or from pre- to post-exposure. However, in the spatial span task, the experimental condition elicited significantly lower memorization ($p = 0.007$) when compared with the control and head cooling conditions. Additionally, pattern recognition tasks were significantly lower in the hot condition ($p = 0.042$) but no significant differences were noted between the head cooling and control conditions.

To determine if the effects of cognition and passive heat exposure were related to temperature regulation, participants were passively exposed to 50°C and 30% RH for 15 minutes while simple reaction time was measured (Gaoua et al., 2012). Measuring skin and core temperature, the authors found that increased skin temperature was associated with cognitive

decrements in reaction time ($p < 0.05$) but that these findings were independent of core temperature.

A potential accumulation of fatigue may also be facilitated within increased thermoregulatory conditions. A study by Qian et al. in which 20 men were exposed to normothermic (25°C, 60% RH) or hyperthermic (50°C, 60% RH) conditions. After exposure, they underwent 20-minutes of a continual cognitive assessment (S. Qian et al., 2015). Their data revealed no significant differences in reaction time under either condition across all stages of the task. However, significant interactions occurred with regards to reaction time and RPE, such that a higher RPE was related to reaction time in the hot condition ($r = 0.634$, $p = 0.005$). Additionally, the hot condition increased cerebral blood flow (CBF) within the brainstem and surrounding regions while decreasing activity within the prefrontal cortex (S. Qian et al., 2015).

In an attempt to delineate thermal strain on cognition, Caldwell et al. (2011) examined different amounts of body armor in Australian Defense Forces (Caldwell, Engelen, van der Henst, Patterson, & Taylor, 2011). Walking on a treadmill for 2 hours while undergoing cognitive assessments, the study exhibited that core temperature increased across all conditions (control, partial armor and full armor), however, none of the tests assessed revealed any significant interactions (Caldwell et al., 2011). Another study during treadmill walking utilizing several different cognitive tests (e.g. verbal, spatial control, digit span) under three different conditions. A control condition (25°C, 65% RH), hot condition (35°C, 65% RH) and hot condition (35°C, 65% RH) with increased core temperature were examined. Their results exhibited only the digit span task being significantly impaired ($p = 0.05$) in the hot condition but not in the hot + core temperature condition (Caldwell et al., 2011).

One reason for these confounding findings may be due to either the task demands, or that core temperature may not be the only cause of altered cognition as presented previously (Gaoua et al., 2012). Taylor et al. (2016) attempted to delineate between simple and complex tasks; which executive function tasks were considered complex. Their data suggests that complex task (executive function) performance may in fact be degraded under conditions of heat stress (Taylor et al., 2016).

A recent study conducted by Malcom et al. (2018) aimed to investigate the type of cognitive task on passive heat exposure (Malcolm et al., 2018). 41 males (21.3 ± 1.6 years) underwent a control (21.2 ±1.8C / 41.9 ± 11.4 % RH) and 'hot' trial (39.6 ± .4C / 50.8 ± 2.3% RH) in which they completed a battery of cognitive tests after 1-hour of passive rest within each bout. These tests included the Stroop Task, Visual Search Task, Corsi Block Task and Rapid Visual Information Processing Task. These tasks were completed in that specific order and the Stroop and Corsi Block tasks represented tasks that have been previously identified in capturing executive function performance (Malcolm et al., 2018). The Visual Search task revealed a slower reaction time for the simple condition but not the complex condition in the 'hot' exposure ($p < 0.01$, $p > 0.05$) while simple accuracy was significantly higher in the hot condition ($p > 0.05$) while complex accuracy was not significantly different in either exposure. The Stroop task exhibited slower reaction times in the hot exposure for both simple and complex trials ($p < 0.01$), however, accuracy was not significantly different between exposures in either simple or complex trials ($p = 0.23$). The Corsi Block test was not significant under either exposure for simple or complex trials ($p = 0.22$) and did not significantly alter across the time of the task ($p = 0.71$).

These findings alter potential confounding outcomes as it relates to executive function. The Stroop task has been globally seen as an executive function task while the Corsi Block test is used

to assess working memory, a domain that comprises executive function. In their discussion, Malcom et al. mention that potentially these findings could be alluded to increased brain resources needed to maintain or improve working memory, while decreased prefrontal activation may be the cause for the decrement in executive function via the Stroop Task (Malcolm et al., 2018).

As such, we postulate that in conjunction with cerebral oxygenation, core and skin temperature may be a potentially modulating factor as it relates to executive function during exercise. Specifically, a limitation in the allocation of resources may divert to regions to maintain physical exercise, which includes the hypothalamus and thus may degrade prefrontal energy and thus executive function.

Summary

The ability for athletic, military and safety sensitive occupations often relies on concurrent exercise and cognitive function. While much of the above literature points to alterations of cognitive function during exercise, much is still unknown. Exercise intensity has widely been conducted in diverse manners, making it hard to objectively determine when cognitive function may be improved or impaired. The types of tasks used has been broken down by previous reviews which have aimed to delineate between general cognition and executive function (Chang et al., 2012a; Flouris & Schlader, 2015; Lambourne & Tomporowski, 2010; Taylor et al., 2016). It is now generally accepted that executive function specifically is considered 'complex' in nature and requires critical thinking skills which lie outside of normal cognition tasks. However, as we've previously seen, much of the literature has used tests aimed globally at executive function, or tasks which may or may not exhibit differences within the sub-types of executive function. This also presents a challenge in interpreting the data. Lastly, there are a myriad of potential physiological measures which have been attributed to alterations in executive function during exercise, including

the catecholamines, lactate, BDNF, Cortisol in addition to overall brain metabolism (via cerebral oxygenation) and alterations in human temperature regulation.

Chapter III: Methodology

The purpose of this investigation was to examine the relationship between executive function and exercise in ROTC members between the ages of 18-30 years.

Experimental Design

This study utilized a within-subjects study design examining the cognitive performance of ROTC cadets while they simultaneously engaged in increasing intensity exercise. The study consisted of four visits with at least 24 hours intervening. All visits were completed approximately the same time of day (± 1 hour) to control for possible effects of circadian rhythm. The visits occurred in the following manner: Visit 1- Participants were consented, screened for eligibility, enrolled, and familiarized with the CEDAR OWAT; Visit 2 – Consisted of Baseline OWAT assessment followed by a maximal graded exercise test (GXT) in Personal Protective Equipment. Visits 3 and 4- Participants completed an Executive Function-Exercise Test (EFET) consisting of the same GXT format while simultaneously completing the OWAT task. Physiological measures were collected via an Equivital sensory belt, while a wireless Near Infrared Spectroscopy (NIRS, Artinis, Netherlands, UK) was utilized to assess cerebral oxygenation. Procedures were identical for Visits 3 and 4. Each visit required approximately 90 minutes each.

Dietary Restrictions

While no dietary restrictions were implemented, cadets were instructed to maintain normal dietary intake. Participants were permitted to consume water *ad libitum*. Because there may be side effects related to the physical performance testing including acute and delayed muscle soreness, musculoskeletal injury, discomfort during exercise, feeling tired, lightheaded or faint,

researchers ensured that participants had eaten food and were hydrated prior to testing. Furthermore, all participants were asked to maintain their normal dietary regimen.

Participants

Participants (N = 15) between the ages of 18-30 years were undergraduates recruited from a Reserved Officers Training Corps at a midwestern university. A total of 23 participants qualified during the initial recruiting, consent, and familiarization processes. A total of 16 participants completed all 3 visits and were considered for analyses. Of the 7 participants that did not complete all visits, 1 participant unenrolled from the University, two others failed to complete their last visit due to scheduling conflicts and the remainder were lost to follow-up visits. 1 participant was excluded due to physiological data which indicated measurement error. Demographic data are presented in Table 2. Participants completed a series of questionnaires including a health status questionnaire to ensure safety during participation. No participant reported a history of significant cardiac pathology or recent musculoskeletal or other injury that would affect testing. The study was approved by the University of Oklahoma Health Science Institutional Review Board and was in compliance with Helsinki declaration. All protocols and procedures were strictly adhered to in an effort to reflect expectations inherent in their policies. Participants provided written informed consent in accordance with University Institutional Review Board policies.

Inclusion

1. Current enrollment in OU ROTC program between the ages of 18-30 years.
2. Healthy, without any cardiovascular or metabolic comorbidities. This includes no orthopedic, pulmonary, cardiovascular or metabolic disorders that prevent participation.

Exclusion

1. Any cardio-metabolic condition including:

a. Hypertension
b. Diabetes
c. Coronary artery disease
d. Stroke
e. Heart attack
f. Neurodegenerative diseases
g. Psychiatric disorders
h. Sleep apnea
i. Kidney disease
j. Insulin use
k. Cardiovascular medications

2. Pregnancy

3. Non-English speaking.

Questionnaires and Documentation

The research design contains questionnaires regarding health status, menstrual history (for female participants), and physical activity levels. The following documentation and questionnaires completion were required by each participant prior to their participation:

Health Insurance Portability and Accountability Act: This health insurance portability and accountability act (HIPAA) informs the participants of the potential use of protected health information during this project.

Health Status Questionnaire: The health status questionnaire consisted of a series of health, wellness, and previous medical history which assisted in the determination of participant inclusion. This document gathers information related to age, demographics, medical history, previous and current exercise habits, family medical history, medications and whether the participant is currently or has been a habitual smoker.

Menstrual History Questionnaire: The menstrual history questionnaire asks a series of questions related to the menstrual cycle. These questions are broken down into two sections: A) current

menstrual status and B) previous menstrual status. Furthermore, the questionnaire collects information regarding the use of contraceptives.

Physical Activity Readiness Questionnaire: The physical activity readiness questionnaire (PAR-Q) acted as the initial screening tool before engaging in physical activity. The questionnaire includes a series of questions determining the participant's capacity to engage in physical activity.

Anthropometric Measurements: Body weight and height was measured using a wall stadiometer (PAT #290237, Novel Products, Rockton, IL, USA) and a Tanita BWB- 800 digital scale (Tanita Corporation of America, Inc., Arlington Heights, IL).

Study Measurements

Executive Function**:** The Cedar Operator Workload Assessment Tool (OWAT; elmTEK, Australia) was used to assess executive function (Figure 1). The Cedar OWAT is the tablet implementation of the NASA Multi-Attribute Task Battery-II (MATB-II) which was designed to evaluate human performance during a multitasking paradigm. The dorsolateral prefrontal cortex is activated during multitasking tests which is associated with sustained attention, working memory and decision making, all components of executive function. The OWAT includes tasks analogous to activities that aircraft crewmembers perform in flight yet is appropriate for populations with no aviation experience. It consists of a resource management task, a communications task, and a systems-monitoring task (Table 1). These three tasks were performed simultaneously without any instruction as to task importance or how to divide attention between the tasks. Specifically, two composite scores (precision and hit rate) provide a global index of executive function performance. Precision average indicates the rate at which participants correctly respond to the stimuli being presented across all three OWAT tasks. Precision is calculated as Hit / (Hits + False Positives). A 'hit' is defined as a correct response to target stimuli. In sum, precision serves as a composite score

for executive function performance. Hit rate is a measure of accuracy that quantifies the number of 'correct' responses to target stimuli while accounting for the number of false positive and missed responses. Hit Rate is calculated as Hit / (Hit + False Positive + Miss), with miss being defined as an incorrect, or non-response to a given stimuli.

Participants were administered the OWAT while resting during Visit 1 for task familiarization followed by a session to establish baseline performance in the absence of exercise. The OWAT test were then administered on Visits 3 and 4 throughout the entirety of the graded exercise test. The OWAT test was chosen due to its extensive use in human performance studies associated with multitasking and its high level of cognitive resource sharing requirements associated with executive function (Comstock Jr & Arnegard, 1992).

***Heart and Respiration Rate, Skin Temperature*:** Heart rate (HR, via ECG trace), respiratory rate (RR), and skin temperature (T_{SK}) were simultaneously measured by *Equivital Lifemonitor system* (ADInstruments, Colorado Springs, CO) on a beat-by-beat and breath-by-breath basis.

Cerebral Oxygenation: Cerebral oxygenation (brain O_2) in the prefrontal cortex was measured by Near-Infrared Spectroscopy (NIRS; Cortech Solutions, Wilmington, NC). This NIRS system allows portable assessment of brain O_2 and offers the most practical *non-invasive* measurements of brain O_2 during exercise with superior temporal resolution (50Hz) to other measurement techniques (e.g. MRI, PET). The NIRS specifically assesses the dorsolateral prefrontal cortex which has been denoted as the main region of executive function.

***Blood Oxygen Saturation**:* (SpO_2) was continuously monitored via *Equivital* wireless pulse oximetry.

Study Timeline

Each visit was conducted with a minimum of 24-hours between each visit and were conducted at the same time of day for each participant.

Visit One: Visit One consisted of participant consent, participant screening, and OWAT familiarization assessment.

Visit Two: Visit Two consisted of a maximal graded exercise (GXT) test conducted in personal protective equipment (PPE) (including footwear of choice, pants, and 16kg of combat armor). The GXT was conducted as follows: Participants began at a speed of 3.5 mph on a level treadmill. Every 2-min the treadmill grade was increased by 2%. If the participant reached a grade of 16% the speed was increased by 0.5 mph every 2 min until exhaustion (Nelson, et al., 2009). The average HR, and RR measured during the last minute of the incremental test was used as HR_{max} and RR_{max}.

Visits Three and Four: Visits 3 and 4 were identical in nature. Combined Executive Function and Exercise Test (EFET) was conducted on each visit, with a minimum of 24 hours between visits. The EFET was conducted with the same parameters for the GXT. The only difference was that participants simultaneously completed the OWAT test while walking. Participants spent ~60min participating in each trial (exercise ~30min; instrumentation ~30min). Instrumentation will involve, 1) fitting of specialized headband with NIRS probes for measuring brain O_2, 2) fitting of Equivital sensory belt for Heart Rate (HR), Respiratory Rate (RR), and Skin Temperature (TSK) measurements, 4) dressing in PPE.

Data and Statistical Analysis

Results of the OWAT test were utilized to quantify executive function performance. The following was extracted from each task for analysis: 1.) Overall precision of responses averaged across each task representing response time and the number of false positives identified by each

task's stimuli presented. 2.) Reaction time and time-out errors averaged across each task identified as misses 3.) Overall hit rate percentage that was averaged across each task, representing an overall 'score' of task performance. These data were segmented into intervals matching stages of the EFET for each test. Scores were averaged across Visits 3 and 4.

Both HR and RR were used to describe exercise intensity by participant per stage of the GXT and EFET. Absolute exercise intensity was calculated as:

Power Output (W) = (wt. in kg + wt. of vest in kg) x (speed of treadmill in meters per minute (incline/100) x time of stage)/time of stage x 0.16344)) and relative work intensity (Heart Rate Reserve, %HRR = $(HR_{exercise} – HR_{rest}) \div (HR_{max} – HR_{rest})$) was established for GXT and EFET. HRR has a predictable linear relationship with exercise intensity and allows the ability to scale the data relative to the individual. Data was examined in both absolute and relative terms as stated above and changes from these from rest to exercise was examined. Furthermore, all OWAT measures were analyzed in both absolute and relative exercise intensities from the onset of the Stage (i.e. Stage 1) whereas

Data was analyzed using SAS, Version 9.4 (Systat, Chicago, IL). Descriptive statistics were reported as mean ± SD (Table 2) unless otherwise mentioned. A repeated measures linear mixed model (LMM) analysis was conducted to analyze cognitive performance as a function of exercise intensity. Heart rate reserve (HRR) and treadmill grade served as relative and absolute exercise intensities, respectively. Based on the present hypothesis, it was anticipated that executive function would be affected by exercise intensity. Specifically, at a lower intensity level EF performance would increase and that at a given high exercise intensity it would begin to decline. This plateau in executive function performance served as thresholds and were recorded individually per participant and utilized in subsequent analyses.

To summarize the relationships between EF and physiological measures, bivariate correlations were computed. Spearman's rank order correlation coefficient (rho) was utilized to account for the non-parametric nature of exercise intensity.

Chapter IV: Results and Discussion

Sample Size: A sample size of 12 was calculated to achieve ≥ 90% power to detect a small effect size (f = .15) for repeated measures hypothesis tests examining the acute effect of exercise on executive function performance with an alpha level = 0.05. A total of 23 participants enrolled in the study however, only 15 participants were included for data analysis (see *Participants* section above). Therefore, sufficient statistical power was achieved.

Participant Characteristics: All participants were healthy, young, physically active ROTC cadets. Baseline demographics for the 15 participants included for analyses are presented in Table 2.

Absolute Exercise Intensity Analyses

Executive Function – Precision Average: The LMM exhibited significant main effects ($F = 5.26$, $p < 0.0001$, Table 3), with significant differences in average precision scores observed at Stage 1 to Stage 10 ($p < 0.0001$). Specifically, from Stage 1, precision remained in the 90% range from Stages 1-9 with a significant decrement occurring at Stage 10. Mean and standard deviations for executive function variables are presented in Table 3, and the Tukey's multiple comparison test statistics for dependent variables at each stage according to exercise intensity are reported in Table 4.

Executive Function – Hit Rate Average: Absolute exercise intensity was significantly predictive of hit rate scores ($F = 20.11$, $p < 0.0001$, Table 3), with hit rate decreasing as absolute exercise intensity increased. While initial hit rates averaged close to 99%, gradual declines were observed

as exercise intensity increased, with significant decrements observed beginning in stage 7 and continuing until the cessation of the test (Table 4).

Cerebral Oxygenation – Hemoglobin Responses: Absolute Exercise Intensity exhibited significant main effects for deoxyhemoglobin (HHb, $F = 63.02$, $p < 0.0001$), total hemoglobin (tHb, $F = 1800.00$, $p < .0001$), and oxygenated hemoglobin (O_2Hb, $F = 50.83$, $p < 0.0001$, Table 5). Each measure significantly increased across the duration of EFET. Multiple comparison analysis indicated significant mean differences between baseline and stages 6-10 for HHb, between stages 5-11 for tHb, and from stages 5-10 for O_2Hb (Table 4). Descriptive statistics for cerebral oxygenation variables are presented in Table 5.

Relative Exercise Intensity Analyses

Executive Function – Precision Average: LMM exhibited significant main effects for relative exercise intensity expressed as HRR ($F = 4.89$, $p < 0.0001$). Pairwise comparisons revealed significant differences between 20 and 60% ($p = .004$) as well as 20 and 100% of HRR ($p < 0.0001$) (Table 6).

Executive Function – Hit Rate Average: There was a main effect of relative exercise intensity on hit rate average ($F = 18.88$, $p < 0.0001$). Hit rate remained stable up to 80% HRR, where it decreased significantly compared to 20% ($p = 0.0001$). Hit rate continued to decline at 90 and 100% HRR ($p < 0.0001$, Table 6).

Cerebral Oxygenation – Hemoglobin Responses: There were main effects of relative exercise intensity on HHb ($F = 72.59$, $p < 0.0001$), tHb ($F = 121.53$, $p < 0.0001$), O_2Hb ($F = 62.79$, $p < 0.0001$). Multiple comparison analyses indicated significant changes in cerebral oxygenation as exercise intensity increased (Table 6). Mean HHb increased significantly from 70 to 100% of HRR ($p < 0.0001$; Table 5). tHb increased from baseline at 40% HRR ($p = 0.02$) and continued to

significantly increase from 50 to 100% of HRR ($p < 0.0001$). O_2Hb remained significantly elevated above baseline from 40 % HRR to 100% HRR ($p < 0.05$).

Relationships Between Executive Function and Exercise Intensity

Executive Function – Precision and Hit Rate: Precision average and absolute exercise intensity exhibited a negatively significant relationship (rho = -0.67, p = 0.02). Correlational analysis indicated a significant negative relationship between Precision and HRR (rho = -0.7091, p = 0.0146). Hit rate average exhibited a negatively significant relationship with absolute exercise intensity (rho = -0.918, $p < 0.0001$) and relative exercise intensity (rho = -0.91325, p = 0.02).

Cerebral Oxygenation Measures: HHb (rho = 0.97, $p < 0.0001$), tHb (rho = 0.99, $p < 0.0001$) and O_2Hb (rho = 0.92, $p < 0.0001$) all exhibited a significantly positive relationship across absolute exercise intensity. Pertaining to relative exercise intensity, HHb (rho = 0.79, p = 0.001), tHb (rho = 0.92, $p < 0.0001$) and O_2Hb (rho = 0.95, $p < 0.0001$) exhibited positive relationships whereas tissue saturation index (TSI) revealed a significant negative relationship (rho = -0.94, $p < 0.001$) declining across exercise intensity (Figure 3).

Physiological Measures: Respiration rate and skin temperature both exhibited significant positive relationships across absolute (respiration rate, rho = 0.95, $p < 0.0001$; skin temperature, rho = 0.99, $p < 0.0001$) and relative exercise intensities (respiration rate, rho = 0.71, p = 0.009; skin temperature, rho = 0.94, $p < 0.0001$; Table 7). Skin temperature exhibited significant effects across the EFET ($F = 261.61$, $p < 0.0001$) with significant changes occurring from baseline (31.73 ± 0.83 °C) throughout subsequent stages ($p < 0.006$) elevating to 36.43 ± 0.39 °C.

Heart Rate Reserve and Exercise Intensity revealed a nearly perfect linear relationship. A total of 8 participants completed stage 12 in the GXT condition, while none completed stage 12 in the EFET condition. The GXT condition consisted of a greater number of participants

completing stage 11 (n = 8), Stage 10 (n = 12) and Stage 9 (n = 15). In the EFET condition, only a total of 11 participants completed Stage 9 and a total 9 participants completed Stage 10. All participants completed Stages 1-9 in both EFET and GXT conditions.

Discussion

Individuals in safety-sensitive occupations are often called to engage in tasks requiring both high cognitive and physical demand. Given the potentially deleterious consequences for performance impairment in high-risk jobs it is imperative to investigate the dynamics involved when engaging in critical thinking while simultaneously performing physically challenging tasks. The purpose of the present study was to systematically examine the relationship between cognitive function and high-intensity exercise. Specifically, by examining executive function across increases in exercise intensity, a secondary question aimed to examine relationships between physiological changes via cerebral oxygenation and executive function performance changes

Executive Function and Exercise Intensity

Both global measures of executive function exhibited significant relationships with both relative and absolute exercise intensity. Specifically, initial measures of both hit rate and precision averaged around 99% before significantly declining as exercise intensity neared maximal levels.

When assessed in terms of absolute exercise intensity, significant decrements in precision average were observed at Stages 8 and 10 while significant decrements in hit rate were observed across stages 7-10. When assessed in terms of relative exercise intensity (HRR), hit rate percentage declined significantly from 70 to 100% of HRR while hit rate significantly decreased at 90% and 100% of HRR (Figure 3).

To our knowledge this is the first study to simultaneously examine executive function and exercise intensity. Findings are consistent with previous research. Specifically, Labelle et al. saw

a significant decrement of executive function (inhibitory control) at both 60% and 80% of peak power output (Labelle et al., 2013). These findings are also in line with those of Smith et al., who found decrements of executive function at both 70 and 90% of HRR (M. Smith et al., 2016). In the present study, decrements in precision average were seen at stages 5 and 11 (e.g. 60% and 100% HRR) while hit rate average saw decrements at Stages 8-10 (70 to 100% of HRR), in line with the findings from Smith et al. and furthermore are in agreement with those from Lo Bue-Estes et al., who also found executive function when assessed post exercise (Lo Bue-Estes et al., 2008).

However, the present findings are not in agreement with the combined findings from the Chang et al. meta-analyses, who revealed an overall small positive effect for executive function during exercise. The discrepancy between findings may be due to differences in terms of a) definition of executive function and exercise intensity b) time of assessment and c) duration. Prior studies were conducted post-exercise or during exercise in a resting state (Chang, Labban, Gapin, & Etnier, 2012b; Wang, Chu, Chu, Chan, & Chang, 2013). The average duration during the present study was between 20-24 minutes, while prior studies indicate potential positive changes in executive function at submaximal exercise intensities (e.g. sustained work rate) for more than 20 minutes.

Cerebral Oxygenation

The present analyses exhibited significant alterations in all four cerebral oxygenation variables for both absolute ($p < 0.0001$) and relative ($p < 0.0001$) exercise intensities. Specifically, (tHb, HHb and O_2Hb significantly rose across exercise intensity (17.60 ± 4.5 to 16.84 ± 3.86), while TSI significantly decreased across exercise intensity (Absolute: $F = 77.4$, $p < 0.001$; Relative: $F = 86.37$, $p < 0.001$).

The present findings are inconsistent with those of prior investigations. Specifically, a 2015 study conducted by Oussaidene et al. demonstrated a significant decline in oxygenation values (tHb, HHb, O_2Hb) at the cerebral oxygenation threshold, occurring at ~85% of workload max (K. Oussaidene et al., 2015). Others have seen significant increases in cerebral oxygenation measures (tHb, HHb, and O_2Hb) up to the respiratory compensation point with no further increases from this point to maximal exercise intensity (González-Alonso et al., 2004; Tempest et al., 2014). Furthermore, additional investigations have shown decline in these measures at high to maximal exercise intensities (E. E. Smith & Jonides, 1999; A. W. Subudhi et al., 2009a; Andrew W. Subudhi et al., 2011).

In the study by Gonzalez-Alonso et al. 2004, the authors discuss the linear increase in metabolism across exercise intensity. This, in turn, increases extraction and uptake of oxygen to sustain exercise. Thus, the present findings are in line with this hypothesis, such that both tHb and HHb significantly increased across exercise intensity, indicating an increase of both metabolism in addition to oxygen uptake and extraction at high to maximal exercise intensities. Furthermore, the study conducted by Oussaidene et al. compared trained versus untrained individuals. Their findings indicate that trained individuals increased changes in cerebral oxygenation values at high exercise intensities. A study conducted by Tempest et al. 2014 examined regional differences in cerebral oxygenation across exercise intensity. Their findings revealed that at maximal exercise intensities, the ventral region of the prefrontal cortex exhibited significant increases. These findings are all in line with the present findings. Furthermore, the groundbreaking study by Subudhi et al. also indicated that throughout exercise intensities, O_2Hb and tHb increased for the right prefrontal cortex, the same region examined in the present study. In this study, the authors also demonstrated significant decrements in TSI at exercise intensity over 75% of maximal power

output. The present study also exhibited significant decline in TSI across exercise intensities. TSI (TSI = O_2Hb/tHb) as an overall index of saturation, a surrogate for cerebral activation of a given brain region. In other words, as intensity increases, saturation increases by means of vasodilation and thus improving oxygen delivery to active brain tissue. Conversely, when saturation decreases, it represents an amount of deactivation of that same region. Thus, the present findings suggest that a decline in TSI is related to a decline in executive function performance and are strengthened by a contaminant increase in HHb. These findings are in alignment with those of Subudhi et al (2009), who also found decrements in TSI with significant elevations in HHb, owing to these changes in order to sustain motor performance (A. W. Subudhi, Miramon, Granger, & Roach, 2009b)

Physiological Responses between GXT and EFET

The present findings are of particular interest as they are the first to exhibit changes in overall work completed during matched exercise conditions with and without an executive function task. The present findings suggest that physical exercise while simultaneously undergoing cognitively challenging tasks targeting executive function, that less overall work is achieved than when solely completing physical exercise. In other words, individuals may have fewer resources available to complete the same amount of work when simultaneously engaged in physical and cognitive demands.

RAH and EFET

The RAH model posits that there are three fundamental energetic principles: 1. The brain receives constant, but limited, resources to supply the energetic demands required by the brain 2. That movement in and of itself requires a large amount of metabolic resources that are allocated systemically in addition to the brain, specifically the motor, sensory and autonomic regions and 3. Neural processing is competitive in nature. Within the context of simultaneous demands, such as

cognitive and physical demands, the competitive nature of neural processing and limitation of increased resources would lead to a downregulation of activity within hierarchical brain regions in order to sustain the ever-increasing demand that exists (e.g. physical exercise). However, limitations to this model point to neurovascular coupling, a principle that elicits an increase of cerebral blood flow to specific 'activated regions' which exist during cognitive and physical tasks executed by the brain.

While the present study did not examine the entire brain, only the right PFC as that area is specifically known to house executive function, the findings exhibit that when analyzed in relative and absolute measures of exercise intensity, tHb, HHB and O_2Hb significantly rose across stages followed by a significant decline during the last stages, nearing maximal exercise. These findings support prior literature examining cerebral oxygenation, but also offer insight into the relationship between cerebral oxygenation, executive function and the theories posited to explain this relationship. These findings support the following tenants of the RAH theory: 1. Dual tasks increase neural demand associated by increases in measures of cerebral oxygenation 2. These increases are indicative of neurovascular coupling, exhibiting increases of activity within the PFC in order to sustain executive function and 3. That at near-maximal to maximal exercise intensity, both executive function and cerebral oxygenation measures significantly declined. Furthermore, when compared with the GXT condition only, participants on average finished 1 stage sooner during the combined EFET condition. This suggests that the increase in neural demand not only potentially shifts to reallocate limited resources in order to sustain exercise but may facilitate the onset of cessation of exercise sooner.

One component to consider when evaluating the present findings may be the understanding of how cerebral delivery of blood is regulated. In essence, the metabolic delivery of substrates into

the brain is tightly regulated in order to prevent both hyper and hypoperfusion of the brain. That is, the brain is much more sensitive to changes in blood flow, and this regulation maintains this order. This means that at higher exercise intensities, as more blood is 'pumped' to active tissues, potentially less blood is delivered to the brain in order to prevent an increase in brain pressure. To offset this, the brain attempts to autoregulate in order to optimize the efficiency of delivery and removal. However, it is not a perfect system and thus, the limitation of available resources then coincides with the competing demands that the brain undergoes with simultaneous exercise and critical thinking. Thus, it may seem that while there may be a limitation to the availability of these resources, future investigations should aim to uncover potential 'resiliency' to these changes.

Understanding the etiology of psychological and physiological stress resilience is imperative to human performance optimization. While psychological stress resilience has been the focus of numerous studies in recent years, less is understood about physiological stress resilience. Additionally, studies examining how physiological systems interact with cognitive performance remain quite limited. The present study was designed to systematically examine the relationship between cognitive performance and exercise intensity in 15 ROTC cadets. Results indicate that both absolute and relative levels of exercise intensity are highly predictive of both executive function scores and degree of cerebral oxygenation. In sum, this study is an important first step in identifying key factors and associated mechanisms indicative of positive adaptation to physiological stress (i.e., exercise), and enables the identification of potential targets for training or the modification of protocols to optimize performance in high risk occupations.

CHAPTER V: CONCLUSIONS

The purpose of the present investigation was to examine the understanding of individual factors that may be associated with physiological stress resilience. Specifically, our objectives were to determine, what, if any, changes in executive function occurred at a given exercise intensity and furthermore, what physiological measures may be correlated with these changes. The main research question was addressed: Does exercise intensity modulate executive function. The present findings support the answer, that yes, exercise intensity does affect executive function, such that executive function is significantly impaired above 200 watts and 70% HRR.

Secondarily, we addressed the following sub-questions: 1. Is there a threshold of exercise intensity in which executive function begins to decline? 2. Are there physiological measures associated with executive function performance changes? 3. Are there individual differences indicative of resilience to executive function decline?

The present findings suggest the following answers to the above sub-questions. 1. There seems to be a threshold of exercise intensity which significantly impairs executive function, occurring between 70-80% of HRR. 2. Cerebral oxygenation values correspond with potential measures of executive function decline, specifically decrements in TSI and non-significant changes of O_2Hb while significant changes in HHb occurred at same relative level of exercise intensities denoted as decrements in EF performance 3. The present study did not elucidate potential resilience to the decline in EF. However future investigations may examine components of physical fitness and level of explicit and implicit tasks which may alter concurrent physical exercise and cognitive challenges. Future investigations should aim to uncover these potential differences.

Significance of the Study

The significance of this study begins to uncover the etiology of the exercise-cognition relationship during exercise, specifically within military populations. It is vital to understand the integrated mechanisms that underlie military and athletic demands. Thus, the interaction between systemic and cerebral function to sustain both higher order complex and critical thinking while simultaneously exercising serves as the backbone of their operational success. The present study indicates that at intensities higher than 80% of HRR, impairments of executive function begin to occur, which may impair mission and athletic endeavors, if these are conducted at intensities higher than this. Furthermore, the present study also displays that undergoing simultaneous exercise and critical thinking tasks may elicit exercise to cease at an earlier absolute time point. These findings allow future studies to further investigate potential measures and mechanisms for these findings (e.g. blood biomarkers) which in turn may be indicative of these changes. Lastly, these findings also allow for targeted training protocols to optimize human operational performance.

Future Research

Based on the present findings, future research may aim to further investigate the physiological responses to changes in executive function during exercise. The present findings suggest that executive function is impaired at higher exercise intensities (. e.g. > 70-80% of HRR) which exhibits a significant relationship with decrements in measures of cerebral oxygenation at maximal exercise. Furthermore, the present findings suggest that concurrently undergoing physically demanding exercise in conjunction with cognitively demanding tasks may limit the overall work completed. This present investigation furthers future research aims by alluding to potential biomarkers which may be indicative of these changes. Furthermore, the implications of these findings should be extended into active duty personnel, allowing not only potential transfer

of findings, but also extend those findings in order to investigate future interventions aimed to optimize executive function changes and 'push the curve to the right' so to speak. While not an aim of the present investigation, military personnel are twice as likely to experience neurological disorders after their service. This alarming health risk can be examined by uncovering potential modulators for exacerbated differences in executive function before, during and after exercise, as previous findings suggest that decrements of cognition in aging adults may be associated with neurological disorders (e.g. dementia, stroke, Alzheimer's).

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Table 1. CEDAR OWAT test descriptions in order of administration

CEDAR OWAT Test	Description
Communications SC3	Measures processing speed by presenting an auditory target stimulus to which the user responds as quickly as possible.
System Monitoring	Measures visual scanning, reaction time, attention, and learning by presenting user with a set of scales and lights that must be attended and responded to as quickly as possible.
Resource Management	Measures visuomotor skills, cognitive flexibility, and attention by presenting complex stimuli to which the examinee must respond as quickly as possible based on a set of rules.

Table 2. Demographic characteristics of study sample

Age (yrs.), mean ± SD	19.6 ± 2
Height (cm), mean ± SD	175.9 ± 6.9
Weight (kg), mean ± SD	79.2 ±15.6
Exercise (days/week), mean ± SD	4.8 ± 1.3
Sex, n (%)	
Male	11 (68.8)
Female	5 (31.3)
Race, n (%)	
White	12 (75)
Black	1 (6.3)
American Indian or Native Alaskan	1 (6.3)
Multiracial	1 (6.3)
Unknown or Not Reported	1 (6.3)
Ethnicity, n (%)	
Non-Hispanic	15 (93.8)
Hispanic	1 (6.3)

*Values rounded to the nearest whole number

Table 3. Means and standard deviations for executive function variables at each stage

Stage	AEI	REI	Precision	Hit Rate
Stage 1	15.3	20	0.99 ± 0.01	0.88 ± 0.07
Stage 2	29.1	30	0.95 ± 0.02	0.85 ± 0.07
Stage 3	58.1	40	0.94 ± 0.03	0.82 ± 0.06
Stage 4	87.2	50	0.98 ± 0.02	0.87 ± 0.09
Stage 5	116.2	60	0.93 ± 0.04	0.83 ± 0.06
Stage 6	145.3	70	0.98 ± 0.02	0.85 ± 0.06
Stage 7	174.3	80	0.96 ± 0.02	0.81 ± 0.07
Stage 8	203.4	90	0.94 ± 0.06	0.73 ± 0.11
Stage 9	248.5	90	0.93 ± 0.02	0.70 ± 0.08
Stage 10	288.5	100	0.92 ± 0.12	0.65 ± 0.13
Stage 11	307.8	100	0.83 ± 0.21	0.51 ± 0.20

**Note*. Values are mean ± SD. Precision = Hit / (Hits + False Positives);
Hit Rate = Hit / (Hit + False Positive + Miss).

Table 4. Significant multiple comparisons for absolute exercise intensity at each stage

Comparison	t-Value	Pr > |t|	Tukey Adjusted P
Precision			
Stage 1 v 5	3.29	0.0013	0.0492
Stage 1 v 11	5.71	< 0.0001	< 0.0001
Hit Rate			
Stage 1 v 8	5.60	< 0.0001	< 0.0001
Stage 1 v 9	5.95	< 0.0001	< 0.0001
Stage 1 v 10	7.76	< 0.0001	< 0.0001
Stage 1 v 11	10.17	< 0.0001	< 0.0001
HHb			
Baseline v 6	-8.85	< 0.0001	< 0.0001
Baseline v 7	-6.74	< 0.0001	< 0.0001
Baseline v 8	-10.31	< 0.0001	< 0.0001
Baseline v 9	-12.26	< 0.0001	< 0.0001
Baseline v 10	-14.56	< 0.0001	< 0.0001
Baseline v 11	-14.45	< 0.0001	< 0.0001
tHb			
Baseline v 4	-6.52	< 0.0001	< 0.0001
Baseline v 5	-10.80	< 0.0001	< 0.0001
Baseline v 6	-14.43	< 0.0001	< 0.0001
Baseline v 7	-15.66	< 0.0001	< 0.0001
Baseline v 8	-15.93	< 0.0001	< 0.0001
Baseline v 9	-16.03	< 0.0001	< 0.0001
Baseline v 10	-16.54	< 0.0001	< 0.0001
Baseline v 11	-15.01	< 0.0001	< 0.0001
O_2Hb			
Baseline v 3	-3.83	< 0.0001	0.0104
Baseline v 4	-6.52	< 0.0001	< 0.0001
Baseline v 5	-9.96	< 0.0001	< 0.0001
Baseline v 6	-12.23	< 0.0001	< 0.0001
Baseline v 7	-12.41	< 0.0001	< 0.0001
Baseline v 8	-11.76	< 0.0001	< 0.0001
Baseline v 9	-10.69	< 0.0001	< 0.0001
Baseline v 10	-9.84	< 0.0001	< 0.0001
Baseline v 11	-8.10	< 0.0001	< 0.0001

Note: Collection of OWAT data (i.e. precision, hit rate) began in Stage 1.

Table 5. Means and standard deviations for cerebral oxygenation variables at each stage

Stage	AEI	REI	O2Hb	HHb	tHb	TSI%
Baseline	0.0	10	7.25 ± 2.51	5.40 ± 1.77	12.14 ± 3.95	66.32 ± 2.89
Stage 1	15.3	20	6.49 ± 3.21	5.76 ± 1.98	11.73 ± 4.65	66.25 ± 2.47
Stage 2	29.1	30	8.09 ± 2.87	5.54 ± 2.07	13.12 ± 4.42	66.55 ± 2.38
Stage 3	58.1	40	10.23 ± 3.19	5.56 ± 2.09	15.28 ± 4.78	66.36 ± 2.29
Stage 4	87.2	50	12.32 ± 3.14	6.19 ± 2.06	18.00 ± 4.68	65.69 ± 4.68
Stage 5	116.2	60	15.00 ± 4.50	7.35 ± 2.40	21.84 ± 6.18	64.56 ± 2.40
Stage 6	145.3	70	16.77 ± 4.58	8.76 ± 2.63	25.02 ± 6.41	63.36 ± 2.51
Stage 7	174.3	80	16.91 ± 4.76	9.80 ± 2.81	26.20± 6.40	61.97 ± 2.99
Stage 8	203.4	90	16.41 ± 5.40	10.53 ± 3.29	26.45 ± 7.30	60.43 ± 3.01
Stage 9	248.5	90	17.85 ± 4.11	13.01 + 4.12	30.28 ± 5.67	60.29 ± 3.32
Stage 10	288.5	100	17.60 ± 4.51	14.81 ± 4.90	31.84 + 6.16	59.77 ± 2.77
Stage 11	307.8	100	16.84 ± 3.86	16.13 ± 5.94	32.47 ± 6.16	59.45 + 4.42

*Note. Values are mean ± SD. AEI = absolute exercise intensity; REI = relative exercise intensityO2Hb = oxygenated hemoglobin; HHb = deoxygenated hemoglobin; tHb = total blood volume; TSI% = percent total saturation index.

Table 6. Significant multiple comparisons for HRR (relative exercise intensity)

Comparison	t-Value	Pr > |t|	Tukey Adjusted P
Precision			
20% v 100%	4.88	< 0.0001	< 0.0001
Hit Rate			
20% v 80%	4.31	< 0.0001	0.0012
20% v 90%	5.48	< 0.0001	< 0.0001
20% v 100%	9.79	< 0.0001	< 0.0001
HHb			
10% v 60%	-3.83	0.0002	0.0078
10% v 70%	-6.59	< 0.0001	< 0.0001
10% v 80%	-10.80	< 0.0001	< 0.0001
10% v 90%	-11.98	< 0.0001	< 0.0001
10% v 100%	-17.30	< 0.0001	< 0.0001
tHb			
10% v 40%	-3.49	0.0007	0.0232
10% v 50%	-6.51	< 0.0001	< 0.0001
10% v 60%	-10.78	< 0.0001	< 0.0001
10% v 70%	-14.31	< 0.0001	< 0.0001
10% v 80%	-18.20	< 0.0001	< 0.0001
10% v 90%	-16.00	< 0.0001	< 0.0001
10% v 100%	-19.40	< 0.0001	< 0.0001
O_2Hb			
10% v 40%	-3.85	0.0002	0.0072
10% v 50%	-6.55	< 0.0001	< 0.0001
10% v 60%	-10.02	< 0.0001	< 0.0001
10% v 70%	-12.30	< 0.0001	< 0.0001
10% v 80%	-14.40	< 0.0001	< 0.0001
10% v 90%	-10.75	< 0.0001	< 0.0001
10% v 100%	-11.22	< 0.0001	< 0.0001

Table 7. Means and standard deviations for physiological variables at each stage

Stage	AEI	REI	RR	SKT
Baseline	0	10	29.30	31.74
Stage 1	15.3	20	44.47	32.22
Stage 2	29.1	30	40.70	32.59
Stage 3	58.1	40	43.11	32.97
Stage 4	87.2	50	43.66	33.39
Stage 5	116.2	60	48.46	33.90
Stage 6	145.3	70	48.28	34.54
Stage 7	174.3	80	52.43	35.03
Stage 8	203.4	90	59.18	35.40
Stage 9	248.5	90	63.78	35.99
Stage 10	288.5	100	66.75	36.27
Stage 11	307.8	100	100.11	36.44

*Note. Values are mean ± SD. AEI = absolute exercise intensity; REI = relative exercise RR = Respiratory Rate. SKT = Skin Temperature.

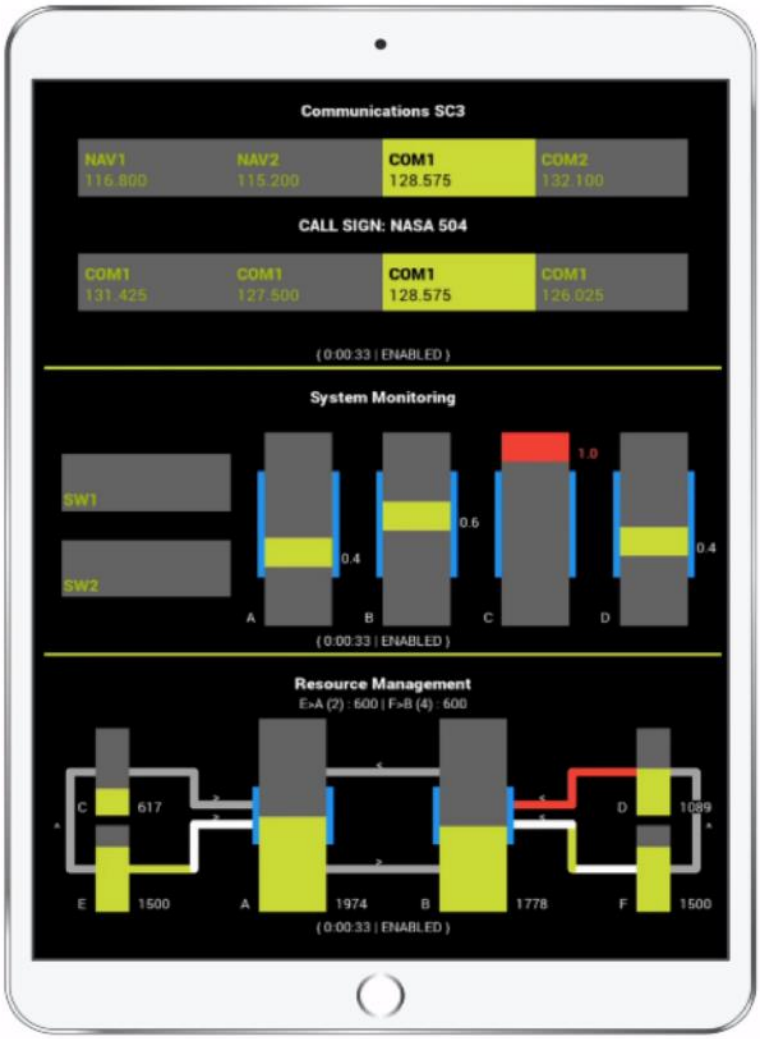

Figure 1. CEDAR OWAT task battery screen.

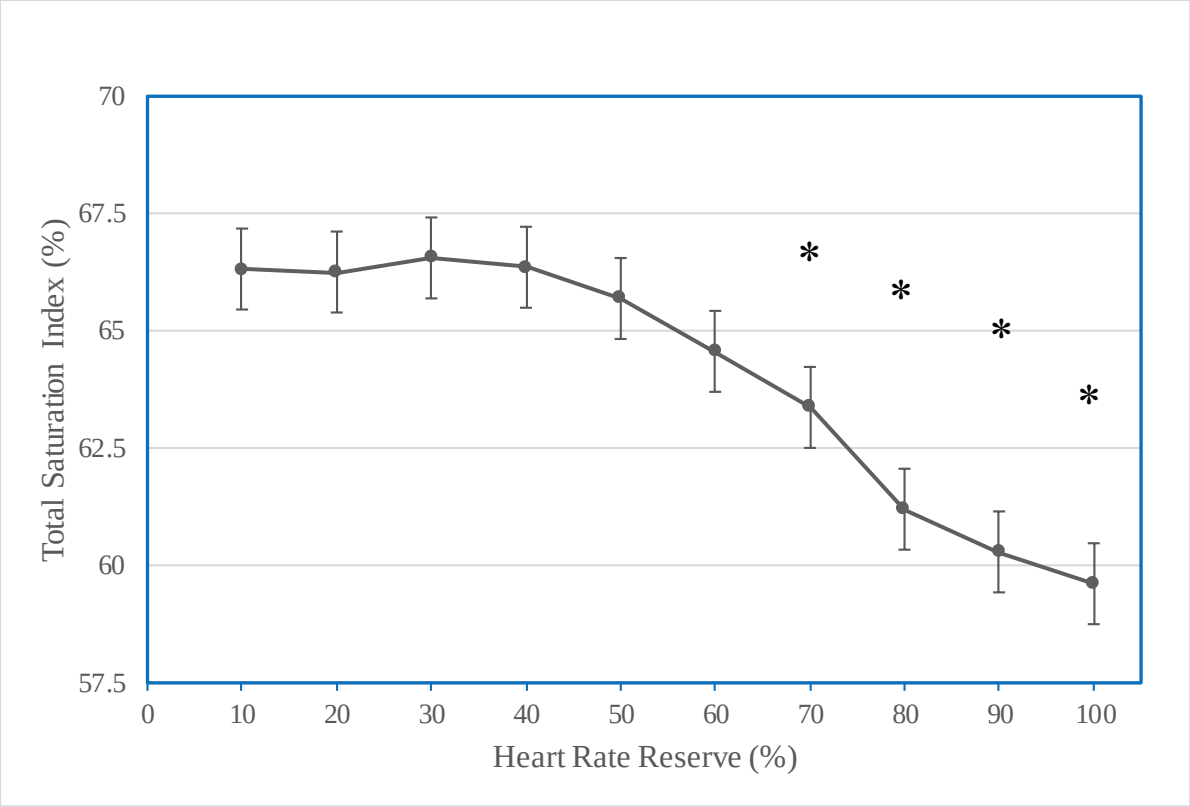

Figure 2. Total Saturation Index and Heart Rate Reserve. * indicates significance from baseline at the p = 0.05 level. A significant negative relationship was exhibited as well (rho = -0.940, p < 0.0001).

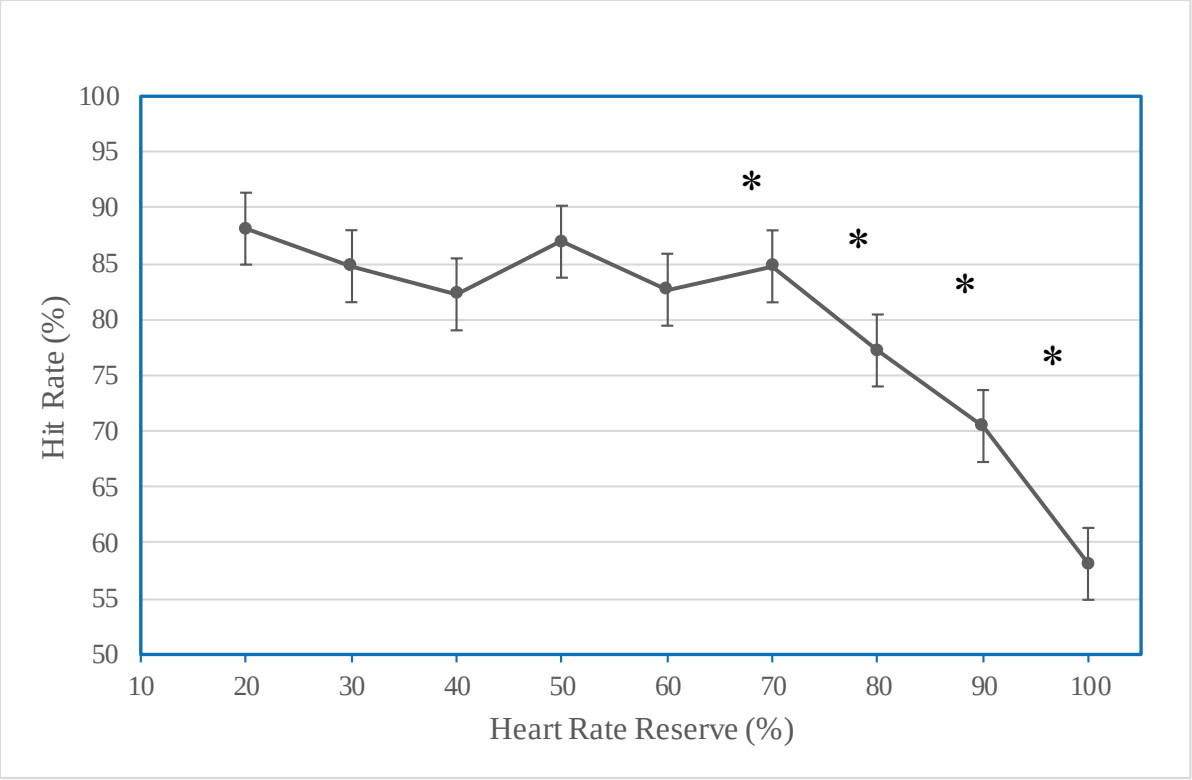

Figure 3. Hit Rate percentage and Heart Rate Reserve. * indicates significance from baseline at the $p = 0.05$ level. A significant negative relationship was exhibited as well (rho = -0.918, $p < 0.0001$).

Appendix A

AUTHORIZATION TO USE or SHARE

HEALTH INFORMATION[1] THAT IDENTIFIES YOU FOR RESEARCH

An Informed Consent Document for Research Participation may also be required. Form 2 must be used for research involving psychotherapy notes.

Title of Research Project: **Physiological Stress Resilience-Optimizing Cognitive Performance in**

Reserved Officers Training Corps During Exercise

Leader of Research Team: **J. Mikhail Kellawan, Ph.D.**

Address: **1401 Asp Avenue, Huston Huffman Center, Room 108, Norman, OK 73019**

Phone Number**: (405) 325-9028**

If you decide to sign this document, University of Oklahoma Health Sciences Center (OUHSC) researchers may use or share information that identifies you (protected health information) for their research. Protected health information will be called PHI in this document.

PHI to Be Used or Shared. Federal law requires that researchers get your permission (authorization) to use or share your PHI. If you give permission, the researchers may use or share with the people identified in this Authorization any PHI related to this research from your medical records and from any test results. Information used or shared may include all information relating to any tests, procedures, surveys, or interviews as outlined in the consent form; medical records and charts; name, address, telephone number, date of birth, race, government-issued identification numbers, and information regarding results from executive function test, maximal exercise test and the combined executive function and exercise test. This includes heart rate, blood results (epinephrine, norepinephrine, brain derived neurotropic factor, cortisol, etc.), skin temperature, core temperature, cerebral blood flow and overall work rates associated with the exercise test.

Purposes for Using or Sharing PHI. If you give permission, the researchers may use your PHI to describe the physiological responses to a combined cognitive and exercise test, including the potential relationship between variables and both the exercise level and cognitive test.

Other Use and Sharing of PHI. If you give permission, the researchers may also use your PHI to develop new procedures or commercial products. They may share your PHI with other

[1] Protected Health Information includes all identifiable information relating to any aspect of an individual's health whether past, present or future, created or maintained by a Covered Entity.

researchers, the research sponsor and its agents, the OUHSC Institutional Review Board, auditors and inspectors who check the research, and government agencies such as the Food and Drug Administration (FDA) and the Department of Health and Human Services (HHS), and when required by law. The researchers may also share your PHI with all members of the research team listed in the approved IRB.

Confidentiality. Although the researchers may report their findings in scientific journals or meetings, they will not identify you in their reports. The researchers will try to keep your information confidential, but confidentiality is not guaranteed. The law does not require everyone receiving the information covered by this document to keep it confidential, so they could release it to others, and federal law may no longer protect it.

YOU UNDERSTAND THAT YOUR PROTECTED HEALTH INFORMATION MAY INCLUDE INFORMATION REGARDING A COMMUNICABLE OR NONCOMMUNICABLE DISEASE.

Voluntary Choice. The choice to give OUHSC researchers permission to use or share your PHI for their research is voluntary. It is completely up to you. No one can force you to give permission. However, you must give permission for OUHSC researchers to use or share your PHI if you want to participate in the research and, if you cancel your authorization, you can no longer participate in this study.

Refusing to give permission will not affect your ability to get routine treatment or health care unrelated to this study from OUHSC.

Canceling Permission. If you give the OUHSC researchers permission to use or share your PHI, you have a right to cancel your permission whenever you want. However, canceling your permission will not apply to information that the researchers have already used, relied on, or shared or to information necessary to maintain the reliability or integrity of this research.

End of Permission. Unless you cancel it, permission for OUHSC researchers to use or share your PHI for their research will never end.

Contacting OUHSC: You may find out if your PHI has been shared, get a copy of your PHI, or cancel your permission at any time by writing to:

Privacy Official
University of Oklahoma Health Sciences Center
PO Box 26901
Oklahoma City, OK 73190

or Privacy Board
University of Oklahoma Health Sciences Center
PO Box 26901
Oklahoma City, OK 73190

If you have questions, call: (405) 271-2511 or (405) 271-2045.

Access to Information. You have the right to access the medical information that has been collected about you as a part of this research study. However, you may not have access to this

medical information until the entire research study is completely finished. You consent to this temporary restriction.

Giving Permission. By signing this form, you give OUHSC and OUHSC's researchers led by the Research Team Leader permission to share your PHI for the research project listed at the top of this form.

Patient/Participant Name (Print): _____

_____ _____

Signature of Patient-Participant or Parent if Participant is a minor — Date

Or

_____ _____

Signature of Legal Representative** — Date

**If signed by a Legal Representative of the Patient-Participant, provide a description of the relationship to the Patient-Participant and the authority to act as Legal Representative:

_____

OUHSC may ask you to produce evidence of your relationship.

A signed copy of this form must be given to the Patient-Participant or the Legal Representative at the time this signed form is provided to the researcher or his representative.

Appendix B

OU Human Circulation Research Laboratory

Health Screen Questionnaire

Instructions: Complete each question accurately. All information provided is confidential.

1. Date: _____
2. Name: _____
3. Mailing Address: _____
4. Phone: (cell): _____ (phone): _____
5. Sex: _____
6. Year of Birth: _____
7. Age: _____
8. Number of hours worked per week:

Less than20 20-40 41-60 60+

9. What is more than 25% of time spent doing at job (or school):

Sitting Lifting/Carrying Standing Walking

10.Date of last physical exam: _____

11.Date of last physical fitness test: _____

12.Have you had any previous surgical operations? _____

If so, where: (circle all that apply)

Back Heart Kidney Eyes Joint Neck

Ears Hernia Lung Other: _____

13.Circle any of the following for which you've been diagnosed or treated by a physician or health professional:

Alcoholism	Concussion	High blood pressure
Anemia (Sickle cell)	Congenital defect	Hypoglycemia
Anemia, other	Diabetes	Hyperlipidemia
Asthma	Emphysema	Infectious mononucleosis
Back Strain	Epilepsy	Kidney problem
Bleeding trait	Eye problems	Mental Illness
Bronchitis, chronic	Gout	Neck Strain
Cancer	Hearing loss	Obesity
Cirrhosis, liver	Heart problems	Osteoporosis
Phlebitis	Stroke	Other:
Rheumatoid	Thyroid Problem	_____
Arthritis	Ulcer	

14.Do you possess any digestive or swallowing problems? _____

15.Have you taken any medicine in the past 6 months?

16.If so, what kind?

17.Do you have any medical appointments upcoming that involve an MRI within 24 hours of your visits?

18.Do you smoke?

19.If you smoke, how many times a day?

20.Weight currently: _____

21.Weight one year ago: _____

22.How would you rate yourself as to the amount of physical activity you get as compared with others your age and sex?

1. Much more active
2. Somewhat more active
3. About the same
4. Somewhat less active
5. Much less active

6. N/A

23.Do you regularly engage in strenuous exercise or hard physical labor? _____

24.How many times a week do you exercise? _____

25.Have you had any other prior health issues that may prevent you from completing a maximal exercise assessment?

Appendix C

Department of Health and Exercise Science

University of Oklahoma

Menstrual History Questionnaire

Subject ID: _____ Date: _____

We are asking you give as complete of a menstrual history as possible. All information you provide will be strictly confidential.

(Circle your response below) Are you pregnant?

Yes – Do not complete the rest of this form

No – Complete the rest of this form

1. Approximately how many menstrual periods have you had during the past 12 months?

2. Circle the months in which your period occurred (from this time last year to the present month)
   JAN FEB MAR APR MAY JUN JUL AUG SEP OCT NOV DEC

3. What is the usual length of your menstrual cycle?
   _____ Days; Today is day _____ of your present menstrual cycle.

4. What was the date of your last period?

5. When do you expect your next menstrual period?

6. What is the length (# of days) of your menstrual flow on average?

7. How many of these days would you term “heavy”?

8. Do you experience cramps during menstruation? If yes, how many days does this usually last?

9. Do you experience symptoms of premenstrual syndrome (i.e. weight gain, increased eating, depression, headaches, anxiety, breast tenderness) If yes, please list these symptoms?

10. Do you take hormonal contraceptives or any other medication that includes estrogen and/or progesterone?

    If yes, how long have you been taking hormonal contraceptives?

    What is the brand name and dosage of the hormonal contraceptive you’re taking?

Has the contraceptive affected your menstrual cycle (regularity, length and amount of flow, length of cycle)? If yes, indicate changes.

11. Have you taken hormonal contraceptives in the past?

    If yes, what is the brand name and dosage?

    When did you start taking the hormonal contraceptive, for how long, and when did you stop taking it?

12. If you answered yes to questions 8 or 9, did you experience a weight gain and/or change in appetite because of oral contraceptive use? If so, indicate amount of weight gain.

13. If you're premenopausal, are you experiencing menopausal symptoms? Please list these (i.e. hot flashes, mood swings, headaches, etc.)

Appendix D

PAR-Q & YOU

Questionnaire - PAR-Q

Physical Activity Readiness
(revised 2002)

(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.

YES	NO		
☐	☐	1.	**Has your doctor ever said that you have a heart condition <u>and</u> 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 (for example, back, knee or hip) that could be made worse by a change in your physical activity?**
☐	☐	6.	**Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?**
☐	☐	7.	**Do you know of <u>any other reason</u> 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 not 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.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

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 _____ WITNESS _____
or GUARDIAN (for participants under the age of majority)