Cognitive Function Across a Competitive Season in Student-Athletes

By

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TABLE OF CONTENTS

ACKNOWLEDGMENTS……. i

TABLE OF CONTENTS…….. ii

ABSTRACT…….. iii

CHAPTER 1: Introduction…… 1
Study aims and hypotheses…… 5

CHAPTER 2: Review of Literature……... 7

CHAPTER 3: Methods…….. 63

CHAPTER 4: Manuscripts

4.1 Characterizing Cognitive Function in Collegiate Rowers …… 76

4.2 Adaptation across a Competitive Season in Collegiate Rowers…… 99

4.3 Fluctuations in Well-Being across a Semester in Physically Active College Students and Student-Athletes……. 124

REFERENCES…… 145

APPENDICIES

I. Cognitive Function…….. 158

II. Adaptations Across a Competitive Season……. 172

III. Gender Differences…… 176

Cognitive Function Across a Competitive Season in Student-Athletes

Student-athletes face the challenge of academic and athletic demands across their competitive season. Research has demonstrated that while athletes have positive mental health before and after the season, negative moods increase during peak training in response to repeated high-intensity exercise. A more complete picture of the psychobiological adaptation across a competitive season is lacking, specifically, little is known concerning how cognitive function is impacted. The purpose of this study was to characterize cognitive function (perceptions, performance and neural activity) in Division I collegiate rowers and to compare cognition, mood, stress and sleep with healthy student controls across a season. Participants included 43 rowers (22 female) and 23 (15 females) controls. Data were collected at baseline, typical training, peak training and recovery. Measurements included the Perceived Deficits (cognitive) Questionnaire (PDQ), Profile of Mood States (POMS), Perceived Stress Scale (PSS), Epworth Sleepiness Scale (ESS), salivary cortisol, performance (response accuracy and reaction time) and electroencephalography (event-related potentials, ERPs) during the Stroop color-naming task. Data were analyzed using 2 (group) x 4 (time) repeated-measures ANOVAs. Significant interactions were assessed using simple effects. Significant group by time interactions were observed for the PDQ ($F = 3.27$), total mood disturbance on the POMS ($F = 4.32$) and PSS ($F = 3.19$). No interactions were observed for performance on the Stroop task, resting cortisol or sleepiness. The pattern of the interaction was such that controls changed little over time while student-athletes had higher scores (indicating more impairment) at peak training compared to baseline. A significant interaction was also observed in N2 amplitude (negative going peak occurring 200ms after the event) during both congruent and incongruent trials of the Stroop task. Student-athletes did not change across testing times, but the controls had significantly larger (more negative) amplitudes at peak training and recovery compared to baseline suggesting that exercise and fatigue may be interacting to impact early stages of object and conflict recognition (N2) in a unique way in athletes. Fluctuations in cognitive function across a season have implications for academic and athletic performance, injury risk and baseline concussion testing.

Chapter One- Introduction

The student-athlete population includes over 460,000 participants (www.ncaa.org) that endure a variety of dynamic academic and athletic challenges across their competitive season. While athletes show similar academic progress as non-athlete student counterparts (Aries, 2004), little is known about the impact of the student-athlete life on brain health, namely cognition. On account of the conflicting positive relationship between exercise and cognitive function but negative relationships between fatigue and cognition, it is plausible that the repeated high-intensity exercise training occurring across an athletic season could have a variety of effects on the brain health of student-athletes. Research addressing the impact of exercise on cognition and mood will have considerable implications for academics, sports, injury susceptibility and the mental well-being of student-athletes. Whether the changes in cognitive function across the competitive season are best characterized by positive adaptation and resiliency, subtle or overt impairment or no change; the relationships between athlete's cognition, mood and exercise can help to explain how vulnerability in this population might develop over time. If healthy adaptations are observed, variables related to psychological health can be acknowledged. Alternatively, if cognitive impairment is observed over time, cognitive systems and the environmental variables associated with impairment can be identified.

College athletes are a unique population and well-suited to examine the complex relationships between exercise, stress and mental well-being. Student-athletes have similar or lower rates of depression and anxiety than non-athlete college students (Yang et al., 2007; Armstrong 2009). Further, student-athletes exhibit positive mood state profiles at the beginning and end of the athletic season characterized by low anger, depression, tension, confusion and

fatigue and high levels of vigor (Morgan et al., 1987). Interestingly, research addressing the impact of increases in training volume across a season on mental health has identified a paradoxical relationship between exercise volume and mood states. During peak training, the mood state profile of the athlete is characterized by elevated levels of anger, depression, confusion, fatigue and decreased levels of vigor. This pattern of disturbed mood has been consistently shown across the season in endurance athletes. Though athletes consistently experience an increase in mood disturbance during peak training, most will recover by the end of the season (Morgan et al., 197, Raglin et al., 1991; O'Connor et al., 1989).

In some athletes the exercise-related mood disturbances could become more severe and accompanied by other symptoms such as decreases in performance and illness. This process has generally been referred to as athlete overtraining or the overtraining syndrome and affects approximately 20-60% of athletes at some point during their career (Lehmann et al., 1993; Meeusen et al., 2013). The overtraining process has been conceptualized as the result of an imbalance between exercise demands and recovery occurring across a continuum of time and symptom severity. In the literature, athlete overtraining is not well defined or understood in terms of pathophysiology but the most common signs include decreases in objective performance, disturbed mood, elevated feelings of fatigue and changes in sleep and appetite (Meeusen et al., 2013; Purvis, Gonsalves, & Deuster, 2010, Hausswirth et al., 2013). Further, overtraining is considered a function of central nervous system dysregulation and in order to identify this dysregulation, many peripheral biomarkers, including hormones, have been examined over the course of increases in training volume with few consistent results. Significant relationships between neuroendocrine responses and mood disturbance during peak training and during the taper of the season in athletes showing signs of overtraining have been reported but

not replicated (O'Connor et al., 1989; Hooper et al., 1993). Interestingly, no research has addressed how cognition, a measure of central nervous function, is influenced throughout a competitive season in collegiate athletes.

A recent area of athlete overtraining research has examined psychomotor speed, or time of decision and response-making, in overtrained athletes. As overtrained athlete symptomology is similar in presentation to major depressive disorder and chronic fatigue syndrome, it has been hypothesized that they may also share some of the cognitive complaints (Nederhoff et al., 2007). In general, these data suggest that during more demanding cognitive tasks, athletes showing signs of overtraining (i.e. decreased performance and mood disturbance) have slowed responses and more errors in their performance (Reitjens et al., 2005; Nederhoff et al., 2007; Hynynen et al., 2008; Dupuy et al., 2010). This small body of research is limited by small sample sizes, variability in the exercise stimuli used to induce athlete overtraining, and a questionable selection of cognitive tasks to test psychomotor speed. However, the results do suggest that behavioral aspects of cognitive performance may be involved in the overtraining process, which has not been adequately addressed in the research.

Cognitive function is crucial component when considering well-being as brain activity drives the way that individuals process information and interact in their environment. Cognition is positively related to social integration, functional independence and indices of every-day functioning in a number of populations (Hanks, 1999; McSweeny, 1985). A number of systems including selective attention, central executive function and various components of memory (Duncan, 2000) fall under the heading of cognitive function. Interestingly, some aspects of cognition are impacted by both acute and chronic exercise and susceptible to fatigue. Specifically, the central executive functions, which include higher-order cognitive processes

inherent to critical thinking such as planning, inhibition, set-switching and decision making (Baddeley, 1996) are at the center of this research. The extant literature reports that the executive functions are related to fitness with higher fit individuals showing better performance during cognitive tasks (Hillman, 2006, Kamijo, 2010, Stroth, 2009, Themanson, 2008) and that improvements in executive function occur following exercise interventions in older adults (Colcombe, 2004; Predovan, 2012; Fallah, 2013). Alternatively, high levels of acute fatigue have been shown to impair executive functions in healthy young adults (Barwick, 2012, Boksem, 2005). Examining the way that repeated high-intensity exercise impacts executive function is important as they 1) impact academic and sport performance 2) have never been examined in a student-athlete population and, 3) can greatly inform our knowledge of normal central nervous system adaptation to increases in training volume.

The primary objective of this study was to examine cognitive function over the course of a competitive season in collegiate rowers. Specifically, we aimed to quantify cognition using perceptions of cognitive function and behavioral performance and brain activity using electroencephalography (EEG) during the Stroop color-naming task, a test of central executive function. Participants were tested four times during the competitive season: pre-season (baseline), mid-season (i.e. easier training), peak training, and post-season (recovery). During each testing time, participants provided a saliva sample to analyze fluctuations in resting cortisol levels. Participants also completed self-report measures of mood and perceptions of cognitive function, stress and college-related demands at each testing time. The results of the study provide insight into the dose-response relationship of behavioral and central nervous system adaptations to changes in exercise training volumes in student-athletes. The objectives of this research were accomplished with the following specific aims:

Specific Aims

Aim 1: To characterize cognitive function (performance and brain responses) over the course of an athletic season in collegiate male and female rowers. Cognitive performance will be assessed using the behavioral performance (reaction time and response accuracy) and brain responses (P1, N1, P2, N2, P3, N4, and P6) during congruent and incongruent trials of the Stroop color-naming task.

Hypothesis 1: It was expected that improvement in reaction times and response accuracy will be recorded between pre-season and mid-season. During peak training, it was hypothesized that slower reaction time and greater latencies for EEG components N2 and P3 for incongruent trials would be observed. Further, N2 and P3 amplitude would be larger during peak training for incongruent trials. Gender differences in cognition were not expected.

Aim 2: To determine the relationship between mood, stress, cortisol and cognitive function in collegiate athletes during peak training.

Hypothesis 2: It was hypothesized that perceptions of mood and cortisol would be related during peak training. Additionally, it was hypothesized that EEG components showing changes during peak training, P3 and N2 latency and amplitude, would be related to indices of fatigue and stress. Gender differences are not expected.

Aim 3: To test differences in mood, stress and Stroop performance across an athletic season between collegiate athletes and healthy college students.

Hypothesis 3: No differences between groups in mood, stress or Stroop performance were hypothesized to be observed at pre-season, early-season or post-season but cortisol that would be higher in athletes during preseason. During peak training, collegiate athletes were hypothesized to have slower reaction times than students on incongruent Stroop trials,

significantly higher cortisol and significantly higher ratings of perceived stress and mood disturbance. Female athletes are expected to have significantly higher tension than male athletes at all testing points.

Aim 4: To identify variables that predict total mood disturbance during peak training in athletes.

Hypothesis 4*:* It was hypothesized that perception of stress and levels of cortisol would significantly predict total mood disturbance during peak training.

Chapter Two- Review of Literature

Academic Performance and Emotional Health of Student-Athletes

Research on the student-athlete population shows both positive and negative consequences of participating in collegiate athletics while highlighting many health similarities to nonathlete college students. However, as data on student athletes is typically collected at one time point, it is unclear how the psychobiological response to the demands of being a college athlete is affected across the competitive season. Thus, as the demands of the student-athlete environment change over time, it is plausible that this change is reflected in mental health outcomes such as mood and cognition.

Participating in college athletics is a significant time commitment. Division II and Division III athletes report spending nearly 16 hours a week on practice and games (Richards, 1999; Vetter, 2010). The majority of Division I student-athletes report spending more than 30 hours per week on their sport and only 2% report spending less than 20 hours a week (Brown, 2000). Because of their responsibilities, student-athletes report that the causes of stress stem from both academic and athletic loads, time constraints, relationships and finances (Humphrey, 2000). Interestingly, when asked to complete the sentence 'stress is___' college athletes respond with words such as pressure, anxiety, overwhelming, frustration, conflict, worry and tension (Humphrey, 2000). Wilson (2005) found that when compared to nonathlete freshman, a sample of freshman student-athletes at a small private Division I school reported significantly greater stress from the number of responsibilities, not enough sleep, heavy demands of extracurricular activities and boyfriend/girlfriend's family with no differences in academic problems. Thus, unique time constraints and stressors of student-athletes may have important health consequences over the course of their season that might impact academic and athletic performance and well-being.

The NCAA emphasizes the importance of their commitment to student-athlete academics in their mission statement and reports a graduation rate of 82% for Division I freshman scholarship athletes. Overall rates of males and females, white and African American athletes' graduation have been steadily increasing suggesting that college athletes are competent and successful students (www.NCAA.org). When controlling for demographic variables, athletes have similar academic progress as nonathlete college students (Hood, 1992; Aries, 2004) even though as a group they enter college with lower grades and standardized test scores than nonathlete students (Aries, 2004). Further, student-athletes report their sport group involvement as a barrier to academic achievement and experiencing the negative stigma associated with being a student-athlete from professors, teaching assistants and their fellow students (Simons, 2007; Hood, 1992). Even though student-athletes show resiliency in academic accomplishments, these measurements do not reflect the dynamic impact of various school and sport-related demands on their cognitive health.

In addition to of academic performance, other aspects of the student-athlete University experience have been examined. One large study found that four-year University or college student-athletes in their first year reported that their institution provided more academic support compared to nonathlete students and that the overall engagement and participation in educational experiences/opportunities is similar between student-athletes and nonathletes (Umbach, 2007). Further, athletes self-reported more gains in personal development and practical competence compared to nonathlete students (Umbach, 2007). A nationally representative study showed that participating in intercollegiate athletics was positively and significantly predictive of motivation to earn a bachelor's degree, improvements in interpersonal skills and leadership abilities over four years and overall satisfaction with the college experience (Ryan, 1989). Finally, athletes

reported that while they encountered negative experiences with feelings of a lack of control, competence and social support, their overall experience was enjoyable and satisfying (Kimball, 2003). Therefore, the vast majority of the literature highlights positive college engagement experiences and similar development as nonathlete students.

Another area of research focusing on student-athlete academics has highlighted gender differences. Female student-athletes might benefit from participating in college athletics to a greater degree than male athletes. Division I female freshman student-athletes report more interactions with non-teammates and higher self-concepts compared to male student-athletes (Gaston-Gayles, 2009). Freshman female student-athletes are more satisfied with their experience, interact with faculty more frequently and participate in active and collaborative learning experiences more than nonathlete females with similar levels grades (Umbach, 2007). Freshman and sophomore female student-athletes at Division I and IAA Universities scored higher on academic adjustment, social adjustment and emotional adjustment than female nonathletes (Melendez, 2006) emphasizing the positive impact of athletics on their college experience.

On the contrary, it has been suggested that the female athlete may experience gender role conflict given that the stereotypical definitions of femininity are violated by the assertive and competitive nature of athletics (Allison, 1991). Although female High School and college athletes report significantly higher perceptions and experiences of gender role conflict than nonathletes, the overall ratings are low (Allison, 1991; Miller, 1996; Desertrain, 1988). Further, the physical nature of athletics may help facilitate positive feelings of competence. In one study of Division I female student-athletes in various sports and nonathlete female college students, athletes reported significantly higher ratings of physical and athletic competence self-concept,

higher body image self-concept and higher ratings of masculinity (Miller, 1996). Importantly, there was no difference in self-reported levels of gender role conflict. Ross (2008) reported that in a small group of Division I female athletes, themes such as being comfortable with being more muscular, appreciation of being powerful and confidence emerged throughout an interview. Finally, in a similar interview setting, Blinde (1993) reported that female student-athletes endorsed themes surrounding body competence, a competent sense of self and learning a proactive approach toward life from participating in athletics. The authors suggest that while athletics is an androcentric institution, female student-athletes may experience empowerment at a personal level (Blinde, 1993). This literature proposes that participating in collegiate athletics might benefit the overall experience of women and participation in collegiate sports may have differential effects on the well-being of female and male collegiate athletes.

A related area of research has surveyed aspects of student-athlete mental health, specifically depression. Overall college prevalence rates of depression are typically around 25% and depressed students report significantly higher amounts of stress (Lindsey, 2009). In athletes, psychiatric symptoms appear to be similar to slightly less than those of the greater college population. One study surveyed NCAA athletes and recreational athlete students using the Symptom Checklist-90 revised (Donohue, 2004). Athlete groups (NCAA and recreational athletes) did not differ in psychiatric symptoms and the groups had significantly fewer psychological distress symptoms when compared to college student normative data (Donohue, 2004). In athletes, psychiatric symptoms are often measured at the beginning of the season. One study reported that 31% of NCAA football players endorsed elevated depression scores prior to season commencement (Bailey, 2010) and female student-athletes report more cognitive, emotional and sleep symptoms than male athletes at baseline (Covassin, 2012). Yang (2007)

collected psychological data on 257 NCAA athletes and reported that 4% of athletes indicated a history of major depressive disorder with 21% of the total sample reporting elevated symptoms of depression. The largest predictors for depressive symptoms were being female, being a freshman and reporting elevated pain during the preceding week (Yang et al., 2007). Overall, student-athletes reported their general well-being as significantly higher than nonathlete college students (Aries, 2004) suggesting that student-athletes are generally a healthy population of young adults. However, increases in negative mood states across a competitive season (Morgan et al., 1987) have been consistently reported and it is unclear if other measured of well-being are being affected in a similar manner.

Research addressing academic performance and mental health of student-athletes is useful to inform coaches and supporting athletic staff of important athlete-specific considerations. Unfortunately this literature is limited by data that are typically collected at one time point (prior to the season), are self-report, and data from all sports and different institutions are often combined. This method of data collection and analysis may preclude important findings in terms of health variables that fluctuate over the course of a competitive season, which may differ across sport type (aerobic and anaerobic) and genders. Further, although academic performance can be measured using grades or standardized testing, it is not an indication of cognitive function, a major component of central nervous system function. Objective psychobiological data are strikingly absent from this population and longitudinal data are needed to better characterize objective changes in cognitive function and mental well-being in student-athletes across a competitive season.

On the whole, the student-athlete population succeeds in academics and has a mostly positive mental health profile. However, no objective data has been collected to characterize the impact

of college athletics on cognitive health over the course of a season. This is important to examine because changes (facilitation and/or impairment) in cognition could have large implications for sport and academic performance, injury susceptibility and daily functioning. In the sections that follow, the relationship between exercise and brain health will be examined by first presenting research on athlete overtraining. This will be followed by a discussion of the definition, importance and measurement of cognitive function. Next, the literature on cognition, exercise and fatigue will be reviewed. Finally, research addressing athlete overtraining and cognition will be covered.

Athlete Overtraining

A small body of literature has addressed aspects of psychological and biological adaptation across an athletic season in an attempt to better understand why some athletes fail to respond positively to increases in training volume across their season. The process of developing training-related symptoms is generally referred to as athlete overtraining. The contributing components of athlete overtraining have been described in the literature as a function of central nervous system (CNS) dysregulation. This dysregulation is often discussed as a result of an imbalance between the demands of exercise training and adequate recovery occurring across a continuum of time and symptom severity. The most common symptoms observed in these athletes include decreases in objective performance, disturbed mood, elevated feelings of fatigue and changes in appetite, sleep, heart rate and immune function (Meeusen et al., 2013; Purvis, Gonsalves, & Deuster, 2010) and these symptoms remain for anywhere from 2-weeks to years before athletes are considered fully recovered (Nederhoff, Zwerver, Brink, Meeusen, & Lemmink, 2008). In order to provide evidence in support for CNS dysregulation, researchers have sought to identify valid and reliable peripheral biomarkers to use as early signs of

overtraining. As an entire body of literature, there is insufficient empirical data to support a unified and comprehensive theory of CNS dysregulation in athlete overtraining. In order to present a review of this research, the challenges associated with overtraining definitions and diagnosis will be discussed. This will be followed by a review of the outcomes that have most commonly been examined in the literature in both short duration exercise intervention studies and naturalistic longitudinal studies occurring across a competitive season.

Defining and Diagnosing Overtraining

The array of terms used to describe the overtraining process (and stages of severity) and the constellation of associated symptoms in the overtrained athlete is a primary factor limiting this body of research. Increases in volume and intensity of the exercise stimulus (also termed overtraining) have been described as a normal and useful way to improve athletic performance across a season of periodized training. However, not all athletes respond to exercise training in the same way and several terms have been used to describe maladaptation associated with prolonged high-intensity exercise. Authors have employed the term stale to describe athletes showing reduced exercise performance in concert with an inability to train at customary levels (Morgan, 1987; O'Connor, 2007), while other authors have chosen the terms underperformance syndrome (Budgett, 2000), overtrained (OT) or the overtraining syndrome (OTS) to label the athlete who manifests signs of aberrant recovery and health (Urhausen, 1997; Halson, 2004; Lehmann, 1993). Recently, the use of the terms functional overreaching (FOR) and non-functional overreaching (NFOR) have been proposed to separate stages of the overtraining continuum. FOR describes responses to increases in training volume that require a short amount of recovery time and ultimately result in improved performance. NFOR describes when an athlete responds to training with more symptoms and requires a longer amount of time to fully

recover (weeks to months) (Nederhof, Lemmink, Visscher, Meeusen, & Mulder, 2006). In a recent consensus statement, a lack of clear definitions across the overtraining continuum was acknowledged as a primary limitation. Without consistent terminology the ability to identify the subtle difference between an athlete with FOR, NFOR and an athlete with overtraining syndrome (OTS) is limited (Meeusen et al., 2013). This consensus statement concluded that NFOR is the first stage on the overtraining continuum during which athletes begin to show mood and hormonal disturbances while the final stage of OTS can be thought of as 'prolonged maladaptation' including more severe symptoms requiring longer recovery time (Meeusen et al., 2013). The lack of clear and concise definitions and distinction of athlete overtraining stages reflects the complexity of the psychobiological process and prevents accurate detection of athletes in research.

A similarly restrictive aspect of studying athlete overtraining is that diagnosis is based on exclusionary criteria. Although myriad symptoms have been declared as part of athlete overtraining, no exhaustive list exists. Studies have used varying objective criteria to identify whether or not athletes are showing signs on the overtraining continuum. These criteria differ across studies and include performance-based outcomes such as maximal exercise tests and time trials, hormonal values and mood data. For example, in a 6-week study, researchers labeled athletes as overtrained if they showed a reduction in athletic performance completed in the laboratory, or had increased responses to questionnaires assessing impaired general health, negative mood or feelings of fatigue (Halson, & Jeukendrop, 2004). A 21-day intervention diagnosed the athlete as overtrained if they exhibited at least two of the following four criteria: 5% reduction in time trial performance, increased resting heart rate by 10%, increase submaximal exercise heart rate by 8 beats/minute, or a 30% decrease in the testosterone:cortisol

ratio (Slivka, Hailes, Cuddy, & Ruby, 2010). A different study concluded that maximal lactate and heart rate following incremental exercise, duration of a maximal exercise test and the mean score of a self-conditions inventory were the best predictors of overtraining (Urhausen, Gabriel, Weiler, Kindermann, 1998). Because the criteria differ across studies, the athletes labeled as overtrained in one study may be quite different than those identified in another. This introduces heterogeneity to the groups defined as overtrained and limits any conclusions that can be drawn concerning the mechanisms, prevention and treatment of athlete overtraining.

In spite of the difficulty of identification and diagnosis of overtraining, the prevalence has been estimated as approximately 60% of elite runners, both male and female at some point in their career (Morgan et al., 1987). The lifetime prevalence rate in endurance athletes is estimated to range from 20-60% (Lehmann et al., 1993). Several studies have reported approximately 20% of their sample as overtrained at one point during the competitive season (O'Connor et al., 1989; Hooper et al., 1993; Uusitalo, 1998). Identifying the etiology and contributing factors in athlete overtraining is essential for promoting well-being in athletes and may also be useful for understanding the pathophysiology of other fatiguing and stress-related disorders such as chronic fatigue syndrome and fibromyalgia. Certainly more research is needed to better understand contributing factors and consistent biomarkers involved in athlete overtraining.

Pathophysiology of Overtraining Syndrome: Neuroendocrine and Psychological Outcomes

Numerous attempts have been made to define the timeline of events in athlete overtraining including peripheral hormone levels and mood disturbance. High intensity exercise training elicits a number of hormones that could potentially be implicated in the development of athlete overtraining. Exercise also impacts mood acutely and chronically and mood states are sensitive to changes in training volumes. Athletes' moods are typically monitored using the

Profile of Mood States (POMS), which addresses changes in depression, anger, tension, confusion, vigor and fatigue (Morgan et al., 1987). Additionally, research on athlete overtraining has consisted of two primary study designs: high-intensity exercise interventions and longitudinal observations. In exercise interventions, training volume can reach as high as 200% greater than the previous normal training volume (Kirwan, Costill, Flynn, Mitchell, Fink, & Neufer, 1988) while longitudinal studies observe natural adaptations over the course of a competitive season. Both types of study design usually monitor increases in training volume over time, measure physical performance using an exercise test, collect biological samples for hormone analyses and measure mood.

The three primary groups of hormones that are discussed in the overtraining literature are those in the hypothalamic-pituitary-adrenal (HPA) axis, testosterone and the catecholamines. The HPA activation begins in the paraventricular nucleus of the hypothalamus resulting in the release of corticotropin-releasing hormone (CRH), which will bind at the pituitary and trigger the release of adrenocorticotropic hormone (ACTH). Next ACTH binds at the adrenal cortex signaling the release of the glucocorticoid cortisol into the blood stream (Rivier, 1991). Cortisol is involved in a number of important homeostatic processes (Mastorakos, 2005). For exercise, the acute release of cortisol contributes to mobilizing free fatty acids, amino acids and maintaining plasma glucose so that the metabolic demands of prolonged exertion can be met (Fragala, Kraemer, Denegar, Maresh, Mastro, & Volek, 2011). Cortisol is important for tissue repair during muscle damaging exercise (Fragala et al., 2011). Increases in cortisol have been found to occur during exercise beginning at ~70% of maximal capacity (Luger, Deuster, Kyle, Gallucci, Montgomery, & Gold, 1987). The HPA axis also has a negative feedback loop that may be disrupted during prolonged exposure to stress and can result in hyper or hypocortisolism. Chronic cortisol release has

different effects on the body than acute such as inhibiting the formation of proteins and testosterone during periods of prolonged stress (Rivier & Rivest, 1991). Hypercortisolism is associated with impaired microbial killing capacity, muscle catabolism, chronic fatigue, bone demineralization, anti-reproductive effects, depression and anxiety (Dulcos, 2007). Hypocortisolism is associated with rheumatoid arthritis (Hakkinen, 2013), post-traumatic stress disorder and chronic fatigue syndrome (Heim, 2000). It is plausible that endurance athletes are at risk for developing disturbances in HPA activity, particularly with prolonged training when failures along the negative feedback loop occur.

Another hormone that is discussed in terms of its role in exercise and overtraining is testosterone – a hormone critical for anabolic processes of muscle adaptation and recovery (Fragala, et al., 2011). Both free testosterone and total testosterone have been shown to increase during low, moderate and high-intensity, long-duration exercise (Tremblay, Copeland, & Van Helder, 2005; kutassalmi et al., 1980). Further, the testosterone:cortisol ratio has been studied in athlete overtraining as an indication of the ratio of anabolic to catabolic activity in athletes (Urhausen, Gabriel, & Kindermann, 1995). Early research hypothesized that a 30% decrease in this ratio was indicative as insufficient regeneration during training and labeled as an early marker of athlete overtraining (Vervoorn, Quist, Vermulst, Erich, de Vries, & Thijssen, 1991) but this has not been supported in subsequent research. Finally, the catecholamines, epinephrine (E) and norepinephrine (NE) have been studied in athlete overtraining. These hormones are regarded as a reflection of sympathetic nervous system (SNS) activity and are released from the adrenal medulla. During exercise, they play a role in increasing cardiac output to supply more blood to the working muscles, bronchodialation in the lungs for improved respiration, vasoconstriction to return larger amounts of blood to the heart and vasodilation to increase the

local blood flow to the exercising muscles (Zouhal, Jacob, Delamarche, & Gratas-Delamarche, 2008). These catecholamines also facilitate the contractile properties of skeletal muscle to support prolonged exercise (Fragala et al., 2011). They also contribute to the sparing of blood glucose by stimulating muscular and hepatic glycogenolysis (Zouhal et al., 2008). As cortisol, testosterone and the catecholamines are involved in both the acute and chronic response to exercise, negative responses to training might be apparent in these hormones.

In addition to the neuroendocrine response, mood states have been studied extensively in athlete overtraining. Athletes show positive mental health profiles at the beginning of the athletic season on the POMS. This mentally healthy status has been referred to as the iceberg profile and shows that athletes have lower levels of anger, depression, tension, confusion and fatigue with higher levels of vigor than population average values (Morgan et al., 1987). Previously published data has shown a consistent and significant inversion of the iceberg profile during peak training in endurance collegiate athletes in a dose-dependent manner (Morgan et al., 1987, O'Connor, 1989, Raglin, 1990). Although all athletes demonstrate a similar pattern of mood disturbance during peak training, most athletes return to healthy baseline levels after the taper period of training. Monitoring mood states over the competitive season and titrating the exercise volume accordingly has been used to prevent staleness in collegiate swimmers (Morgan, 1987). In this body of research, mood disturbance is usually significantly impacted as training volume increases, but unfortunately changes in mood are not consistently shown to be related to other central nervous system markers involved in overtraining.

One way that athlete overtraining has been studied is by increasing exercise volume for a short time and monitoring neuroendocrine and mood responses. One of the shorter exercise intervention studies employed a two-fold training volume increase for 10 consecutive days in 12

collegiate male swimmers (Kirwan et al., 1988, Costill et al., 1988, Morgan et al., 1988). Testing took place at baseline (day zero), day five and 11. Muscle biopsies and blood samples were collected, swim tests and mood questionnaires were completed, participants were asked to rate their daily perception of training effort (very easy to very difficult) and to keep a nutrition log (Costill, 1988). Resting cortisol and post-exercise test cortisol levels were elevated on days five and 11 compared to baseline. Pre-exercise testing creatine kinase levels were increased on days five and 11 suggesting higher levels of muscle damage during the second half of the training period. From day two to five, participants indicated elevated perceptions of training difficulty and increases in depression, anger, fatigue and global mood disturbance (Costill, 1988; Morgan et al., 1988). This short increase in training volume damaged muscle, led to increased cortisol levels both at rest and in response to exercise and impaired mood, all suggesting that an sufficient exercise stimulus was employed and that the athletes started to developed training-related symptoms. However, because of the short duration of training it is difficult to generalize this response to that seen during the course of a full season of training.

At the end of this 10-day study, the authors identified four athletes based on the coaches' observation and those showing the highest ratings of training difficulty and labeled them 'responders' (Costill, 1988). As a group, these athletes consumed less total calories and carbohydrates, had lower muscle glycogen prior to and after the training intervention and had lower distance per stroke at the end of the intervention (Costill, 1988). The responder's hormonal profile, performance on the maximal exercise tests and swim power did not differ from the non-responder group. The mood profiles of all of the swimmers were analyzed in a separate lab to attempt to identify whether changes in mood states could accurately predict the group of responders (Morgan et al., 1988). The researchers correctly identified three of the four

responders based on their mood data and further analyses showed that while no differences between the groups existed at the beginning of the study, the responder group had significantly greater total mood disturbance across the last five days of training (Morgan et al., 1988). The authors concluded that overtraining could be related to inadequate glycogen consumption and monitored by measuring changes in mood states but not by using the physiological data alone (Kirwan et al., 1988, Costill et al., 1988, Morgan et al., 1988). This study is interesting because of the many variables that were collected. The finding that all athletes had similar hormonal responses but the responders had elevated mood disturbance would suggest that mood is the most sensitive measurement of central nervous system function to short-term increases in training volume.

The next group of studies employed longer exercise interventions and focused on the catecholamine response to exercise and mood. Lehmann et al., 1993, examined the changes in hormones and mood over the course of a four-week high volume training period in elite male endurance athletes. By day eight, a significant increase in complaints (‘1’ meaning no complaints to ‘4’ meaning severe complaints) in muscular stiffness, fatigue, and exhaustion were observed. A decreased E response following exercise on days 21 and 28 was observed while resting cortisol, NE and E did not change over the course of the intervention. Decreased NE and E excretion recorded in morning urine samples positively correlated with the reported complaints at week four (Lehmann et al., 1991). Decreased release of E in response to a stressor (i.e., exercise test) and a decrease excretion of E and NE in spite of similar resting levels suggest a blunted catecholamine response to prolonged exercise training.

A similar study observed comparable decreases in the catecholamine response to exercise across their four-week intense training intervention in 15 female endurance athletes (Uusitalo et

al., 1998). Testing occurred at baseline training, following four-weeks of normal training (control group, CG) or of high-intensity experimental training (ET group) and following a four-week recovery period. If athletes showed a decreased maximal exercise performance or an unwillingness to train in combination with mood disturbance, they were identified as overtrained (OA). Five athletes met these criteria. No changes in urine catecholamine concentrations or cortisol were observed, but there were decreases in plasma NE at submaximal, E and cortisol at maximal work in the ET group. In the OA group, plasma Eat maximal work rate decreased during the four-weeks of intensified training. Mood scores from self-reported scales increased for the average of negative feelings while the average of positive feelings decreased in both the ET group and OA group from baseline to the end of the study. Further, there was no relationship between menstrual phase and recorded hormone levels. This study contradicts the previous study by observing similar levels of E and NE in overnight excretion. However, during exercise, a blunted catecholamine response was reported again. The authors concluded that the physiology of overtraining is complicated and due to the large individual variability, a consistent pattern of physiological response to overtraining was not detected (Uusitalo et al., 1998).

In contrast to the previous studies, other research has reported mood changes but failed to find any change in the hormonal response during intense training. Halson et al., 2002, examined catecholamines and mood throughout two-weeks of moderate training, two-weeks of intensified training and following two-weeks of recovery training in male endurance athletes. All athletes met the criteria for overreaching according to the authors, which was a reduction in performance and increases in mood disturbance. Total mood disturbance, tension, fatigue and confusion and vigor (decreased) changed significantly after the intense two-week training period, but returned to baseline following the two-weeks of recovery. No differences in resting or exercise levels of

plasma E or NE were observed across the study (Halson, et al., 2002). A further well-designed intervention assessed the changes in hormones over a 21-day intensified training period (Slivka et al., 2010). Although both salivary cortisol and testosterone decreased from wakening to the afternoon sample, no changes over the course of the high-intensity training period were reported. The testosterone:cortisol ratio increased from pre-daily exercise to post-daily exercise but no significant changes occurred over time. Vigor scores from the POMS decreased significantly from day one to four and remained lower for the duration of the study. The authors concluded that these particular hormonal biomarkers were not useful to identify athletes showing signs of overtraining (Slivka et al., 2010). As mood but not endocrine markers were disturbed in these studies, it may be the case that the exercise stimulus was not intense or prolonged enough to induce physiological signs of negative responses to increases in training volume.

Other research highlights the importance of performance markers in identifying athlete overtraining. A four-week overload period followed by a two-week taper was used to study the influence of high-intensity training on 16 well-trained male triathletes (Coutts, 2007). Athletes were assigned to either the intensified training (IT) or a normal training (NT) group. The IT group showed significant decreases in performance during the overload but performance then improved following the taper. No changes in hormones were observed following the overload in either group but cortisol decreased and the free testosterone-cortisol ratio (TCR) increased significantly in the IT group after the taper. A significant increase in total stress and reduction in total recovery measured by the RESTQ-76 was present in the IT group compared to the NT group during the overload and these values recovered during the taper. The authors argued that performance and psychological, not biochemical or physiological measures, are the most useful indicators of overreaching (Coutts, 2007). A final exercise intervention took place over a 10-

week training camp with collegiate cyclists (Martin, 2000). Three-weeks of baseline training were followed by six-weeks of increased training and a final week of recovery. Performance improved compared to baseline values for all weeks of training and again after recovery. Cortisol increased at weeks one, two, three and four from baseline and peaked at week four. No significant changes in global mood disturbance, fatigue or vigor were observed over the intervention. The authors concluded that the POMS was not able to differentiate or predict those athletes showing signs of overtraining and stated that performance measurements should be the primary dependent variable in this research (Martin et al., 2000).

The exercise intervention research has not produced consistent results in terms of a hormonal or mood state profile for the athletes experiencing symptoms of overtraining. It could be argued that with increases in exercise training, an initial increase in cortisol occurs followed by a blunted HPA and SNS response to exercise. It is not clear how long these stages occur or whether they are indicative of any deviation from normal adaptation. Numerous methodological limitations exist for this body of research. For example the biological sample (plasma, saliva and urine) and time of day that the sample was collected vary across studies. Small sample sizes and large variability have limited statistical power and the ability to identify definitive response patterns. Variations in exercise intensity and volume make it possible that there are individuals in each study for whom the exercise stimulus was insufficient to induce any stage of overreaching. This body of work requires consistent procedures and larger sample sizes in order to define the psychological and biological correlates of athlete overtraining.

Longitudinal studies have been used in the overtraining research to characterize normal neuroendocrine and mood responses over the course of an athletic season. These studies provide stronger ecological validity and a longer duration in which to study responses to changes in

training volume. One well-designed study assessed mood and cortisol over the course of a six-month season in collegiate female swimmers (O'Connor et al., 1989). Testing occurred at baseline, during high-intensity training and after a taper period. Athletes completed a swim performance test, the POMS and provided a salivary cortisol sample in the afternoon. Swimmers had elevated cortisol at baseline and during high-intensity training compared to a group of healthy control college students. Athletes had elevated tension, depression, anger, fatigue, and decreased vigor on the POMS during high-intensity training compared to baseline. Additionally during this testing point, cortisol was significantly related to the POMS depression scores. Three athletes were identified as stale during the season (decreased training ability and decreased performance by 5-10% or larger). During high-intensity training, this group demonstrated greater total mood disturbance, greater depression and higher cortisol levels compared to the healthy athletes (O'Connor et al., 1989). Thus, both mood and a decreased sensitivity to cortisol in the HPA axis may reflect multiple systems responding to the increases in training volume.

A similar study examined catecholamine and cortisol responses across a 6-month competitive swim season in both male and female swimmers (Hooper et al., 1993). Athletes were asked to record training volume, intensity and fatigue in a daily journal. During each of the testing days, athletes completed the POMS and a resting blood sample was collected before the athletes performed a maximal exercise test. A significant decrease in E during the recovery testing time compared to the baseline sample was observed, and no changes in resting cortisol or NE were found. Both E and NE levels were positively correlated to training volume. Interestingly, three athletes (all female) in the sample met the criteria for overtraining: lack of improvement during a maximal exercise test and high consecutive ratings of fatigue. When these athletes were separated from the rest of the sample, significantly elevated levels of NE during the

taper period (4th testing time) compared to any other time of the season were observed. The authors suggest that overtraining could be prevented by monitoring NE (Hooper et al., 1993). This study partly contradicts the previous study in that resting cortisol was not elevated or related to mood, but rather NE had the strongest relationship with training volume and symptoms of overtraining during the taper period. This response may be the result of athletes who increase their training during the taper when they should be resting to compensate for decreased performance.

An interesting study examined elite endurance athletes over a 10-month training season to describe the rates of transforming free cortisol into an inactive form by measuring the overnight excretion of both cortisol and cortisone (Gouarné et al., 2005). It was hypothesized that the HPA axis becomes adapted (less sensitive or more efficient at elimination) to the repeated exercise-related increase in cortisol. Nine untrained healthy males (UT) and 10 endurance-trained men (T) were tested at three different time points (1= baseline in November, 2 = intensified training in March, 3 = competition season in June). For each testing time point, a fatigue inventory was completed, saliva was collected at wakening and 30-minutes later, a urine sample was provided and a maximal exercise test completed. In both groups, overnight cortisone excretion was significantly higher at time points 1 and 2 compared to 3 with no group differences. Further, when cortisol excretion was similar between groups, the T group had significantly higher cortisone excretion suggesting that a higher inactivation of cortisol into cortisone in highly trained men compared with the untrained group occurred. Two of the 10 T men showed signs of overtraining (high fatigue, high training load and decreased performance) and their urinary cortisone was lowest when the urinary cortisol levels were greatest, suggesting a low rate of cortisol to cortisone conversion, which may be contributing to their overtrained

status. The authors concluded that the pattern of increased overnight urinary cortisone in the well-trained athlete was a protective adaptation to counter the prolonged exposure to cortisol following repetitive strenuous exercise cortisone (Gouarné et al., 2005).

Testosterone and cortisol responses across a season have also been studied primarily in national team rowers. Urhausen et al., 1987, examined the hormonal and performance changes over a 7-week season in elite level male and female rowers. Blood samples were collected once a week in the morning under fasting conditions and analyzed for cortisol and testosterone. Testosterone for both male and female athletes tended to decrease over the course of the training period but only the males showed a significant decrease. Furthermore, though no change in cortisol was identified, the TCR fell below baseline in both males and females at weeks five and seven, but it was only significantly lower in males at weeks six and seven. The authors concluded that the anabolic processes of testosterone are likely important for successful performance and it is unclear what amount of decrease is detrimental to performance and mental-health (Urhausen, et al., 1987). Elite male rowers (n = 6) were observed over a 9-month season at 10 testing points for changes in free TCR (Vervoon et al., 1991). A decrease in the free TCR was generally found during week three to five in which the athletes participated in a training camp, but due to the large individual variability, a consistent pattern of response was not observed. The FTCR did not correlate with any performance marker. Other research during a rowing season has reported no changes in resting morning or afternoon salivary cortisol (Iellamo et al., 2002). These two studies failed to measure mood and did not support the claim that a decrease of 30% of the TCR is an indication of overtraining in endurance athletes.

Other studies have failed to find significant changes in mood or hormones in endurance athletes. Collegiate male runners and swimmers were studied across a training season (Flynn,

Pizza, Boone, Andres, Michaud, & Rodriguez-Zayas, 1994). Athletes completed the POMS, a maximal and a submaximal exercise test and provided blood samples at four times across their season. Runners showed no change in either mood or hormones over the course of the season. Swimmers demonstrated significantly higher total mood disturbance after their winter training camp at time points two compared to all the other times and lower total testosterone at time points two, three, and four. No changes in cortisol or the cortisol:testosterone ratio during the season were reported (Flynn et al., 1994). It is not clear why the runners did not show any changes in these the POMS but it is one of the few endurance groups that did not show the inverted iceberg profile during their season. This study utilized multiple time points across a season in collegiate endurance athletes and a clear pattern of responses was not detected.

A final group of studies in the athlete overtraining literature has examined the hormonal response to repeated maximal exercise tests within a few hours of each other to examine the central nervous system response to repeated potent physical stressors (Meeusen et al., 2004; Meeusen et al., 2010). In the first study, seven cyclists completed a 10-day training camp. Athletes completed the two maximal exercise tests before and after the training camp. In the tests before the training camp a 3% decrease in performance was observed from the first to the second test with similar increases in ACTH, prolactin, cortisol and growth hormone during the exercise tests. After the training camp, performance decreased by 6% with nonsignificant increases in ACTH or cortisol during in the first and second test. One athlete in this study had received a clinical diagnosis of OTS and demonstrated a large increase in ACTH and a diminished growth hormone response to the first test followed by a large decrease in ACTH and increase in growth hormone during the second maximal exercise test. This response suggests a hypersensitivity of the HPA axis at the pituitary but a down-regulation at the adrenal cortex in this athlete with OTS

(Meeusen et al., 2004). This same study protocol was repeated at one testing time in ten athletes (two females) with a diagnosis of NFO or OTS according to a physician and a retrospective examination of symptoms and performance decrements (Meeusen et al., 2010). A one-year total duration of symptoms differentiated NFO (n = 5) from athletes with OTS (n = 5). Resting cortisol, ACTH and prolactin were higher for OTS athletes compared with NFO. The only exercise difference between the groups was higher ACTH and prolactin response in the NFO group during the second test where OTS athletes showed very small or no increases. Prolonged maladaptation to exercise training may then be characterized by elevated ACTH at rest, but blunted responses to a physical stressor.

The overall pattern of psychological and hormonal changes in response to increases in training volume is difficult to ascertain from these studies, but some consistencies have emerged. From the separate analyses for athletes with OTS, it appears that these athletes show higher cortisol during intense training, decreased responses (cortisol and catecholamine) at maximal exercise and higher NE, cortisol and mood disturbance levels during the taper. Another trend that may be useful in understanding the etiology of overtraining are the data demonstrating that testosterone levels at rest may be decreasing over the course of the season. The equivocal findings in the athlete overtraining literature are a result of the complex nature of the psychophysiological process associated with endurance training and the inconsistent methodology employed to collect data. Future research would benefit from adherence to consistent methods for hormone collection, inclusion of larger sample sizes, use appropriate control groups to identify the difference between what constitutes normal adaptations and how overtrained athletes deviate from this pattern. Further, a stronger theoretical background linking central nervous system dysfunction and athlete overtraining should be identified. For example,

does brain function of these athletes differ and might this contribute to or drive the changes in peripheral circulating hormones? Few studies in the athlete overtraining literature have assessed how repeated high-intensity exercise impacts cognitive function. This question is important to consider because any changes observed in cognition can provide useful insight to the nature of central nervous system adaptation (reflecting healthy adaptation or negative responses to training) occurring over a season. Before reviewing that literature, it is necessary to provide a thorough description of research defining and measuring cognitive function.

Cognitive Function

Cognitive function is comprised of the mental processes underlying thoughts and emotion, which guide behavior, (Tomporowski, 2005). Cognition includes perceptions, behaviors and the neural systems related to selective attention, central executive functions and aspects of memory. Although the exact mechanism(s) guiding communication across brain regions is unknown, the literature on cognition generally concludes that substantial interactions across brain regions exist during cognitive processing (Banich et al., 2009; Carpenter et al., 2000; Smith et al., 1999). The study of cognitive function is important because cognition is related to central nervous system function, behavior and overall quality of life. Further, cognition is affected in a number of physical and psychiatric illnesses including Alzheimer's disease, major depressive disorder, bipolar disorder, schizophrenia, multiple sclerosis, fibromyalgia and chronic fatigue syndrome (Lee et al., 2012; Michiels & Cluydts, 2001; Jongen, 2010, Chadhuri, 2004; Lewandowski, 2013,). Aspects of cognition have been shown to be predictive in measures of social integration and functional independence during rehabilitation in a sample of traumatic brain injury inpatients (Hanks, 1999). Additionally, neuropsychological test performance predicts indices of every-day life functioning such as self-care and social roles in a sample of

patients with chronic obstructive pulmonary disease (McSweeny et al., 1985) and community functioning in a sample of bipolar and schizophrenic patients (Lewendowski, 2013). Thus, as cognition is related to many aspects of daily functioning and mental health, there is a great need to better understand how cognitive function becomes impaired and how it can be improved.

Central Executive Functions and Electroencephalography

One aspect of cognition that has been extensively studied is the central executive functions. The (central) executive functions were originally proposed by Baddeley (1974) as a part of working memory; a system responsible for the temporary storage and manipulation of information. The executive functions are suggested to have a limited capacity and include an array of higher-order processes inherent to critical thinking such as planning, inhibition, set switching, decision-making and handling novel information (Baddeley, 1996). These functions are essential for monitoring the external world for relevant information, integrating knowledge, inhibiting irrelevant actions and therefore driving appropriate behavior (Funahashi, 2001). Further, executive functions are a complicated coordinated set of processes that can be influenced by disease states and enhanced with exercise. However, because changes in executive functions are often subtle, they are difficult to identify in a research setting and are typically only present during challenging cognitive tasks. In healthy populations, it is currently unknown whether elevated levels of fatigue experienced at peak training influence executive function.

One cognitive task used to examine executive functions is the Stroop color-naming task (Stroop, 1935; McLeod, 1992). This task measures responses to concurrently presented stimuli across sensory modalities (auditory, visual and semantic domains) that compete for cognitive resources. When two types of stimuli are similar, for example reading a color-word and viewing a color stimulus, the information interference is greater (McLeod, 1992). One common Stroop

testing paradigm uses word and color stimuli that are congruent (a color word presentation in the same colored text) such as the word ‘red’ in red text. An example of an incongruent trial (a color word in a different colored text) might be the word ‘red’ in green text. Some studies also use a neutral condition in which a color word appears in grey letters. The relatively automatic processing of the meaning/reading of the word and identifying the color competes for cognitive resources and therefore conflict arises during incongruent trials (MacLeaod, 1992; Zurron, 2009). Although there are various versions of the task, typically, participants can be instructed to respond according to the text color, read the word, or respond whether the trial is congruent or incongruent. The results of the test consistently show that response time is slower and response accuracy is lower in trials where interference exists (incongruent) as one of the processes that occur relatively automatically without conscious effort must be inhibited. Theories on the Stroop effect surmise that interference could occur at the attention demand stage, the perceptual encoding stage, be a function of the strength of each processing pathway, or occur at the response-selection stage (McLeod, 1992).

The standardized color-naming Stroop task has been studied in the cognitive literature for reliability and for the influence of important variables. The standardized Stroop (as opposed to an emotional Stroop) has been shown to have test-retest reliability across 3 months of testing (Kindt, 1996). Additionally, over the course of an 11-month examination of cognitive inhibition tasks (Eriksen flanker task, Go No/Go and Stroop), stability in inhibition-related performance and good internal consistencies for most variables were reported (Wostmann, 2013). Response times for a paper pencil version of the Stroop task did improve from baseline to the 11-month follow-up test (Wostman, 2013). The influences of gender and age in Stroop performance have also been examined. While some research has concluded that women perform significantly faster

during the Stroop task (Mekarski, 1996), other research has concluded that no gender difference exists (MacLeaod, 1992). A meta-analysis on the influence of age on Stroop performance concluded that response time latency during incongruent trials was significantly different from congruent latency for both young (30 years or younger) and older adults (greater than 60 years), supporting a Stroop effect that is observable across ages (Verhaeghen & Meersman, 1998). Another review suggested that performance on the Stroop shows the greatest interference in young children and will improve during adulthood and then decline with advanced age (McLeod, 1992).

Performance on the Stroop task can be analyzed by comparing the behavioral and neural responses during congruent and incongruent trials. The aim of utilizing this task in research is to locate where and when in the brain irrelevant information is being inhibited, conflict is being resolved and correct responses are determined. Generally, the executive functions involve neural networks that reside in frontal brain regions and deep subcortical regions involved in response selection and conflict detection (Cannon, 2009). Research using functional brain imaging methods have identified greater brain responses during incongruent compared to congruent Stroop trials in orbital frontal, superior and inferior frontal, middle frontal, insular, anterior cingulate and cerebellar regions (Harrison, 2005; Leung, 2000; Pardo, 1990). Further, responses in the prefrontal cortex (PFC) and the anterior cingulate cortex (ACC) have been consistently shown when using brain-imaging techniques to examine the Stroop effect (Duncan, 2000; Silton, 2010; Markela-Lerenc, 2004; Banich, 2009; Garavan, 2002).

Brain imaging measurements allowing for a more precise analysis of the temporal sequence of neural activity during executive processes are ideal for research aimed at detecting nuanced changes in information processing. Subtle changes in cognition may be well

characterized by changes in the speed of information processing or network recruitment during challenging cognitive tasks. One method used to examine brain responses during cognition is electroencephalography (EEG), which has been shown to be related to the functional magnetic resonance imaging (fMRI) blood oxygen dependent-level signal but with superior temporal resolution (Sammer, Blecker, Gebhardt, Bischoff, Stark, Morgen, & Vaitl, 2007). EEG is an indirect measure of neural activity that is quantified as voltage differences in electrical currents between an electrode on the surface of the scalp and a reference electrode located in a neutral area such as the ear lobe, forehead or mastoid (Tonner & Bein, 2006, Tzovara, 2012). Pyramidal neurons that comprise the gray matter of the neocortex are responsible for producing ionic currents across synapses in order to process information. When large groups of functionally related neurons are simultaneously activated, excitatory postsynaptic potentials propagate throughout the brain and can be recorded at the scalp (Schaul, 1998, Hallez et al., 2007). Components of the potentials are impacted by the distance from the current generator (source), the duration of the potential, the number of synchronized potentials and the orientation of the pyramidal cells to the scalp (Schaul, 1998). EEG allows for a precise temporal examination of information processing, but is limited in its spatial resolution and in determining where the processing originated (generators) referred to the inverse and forward problem respectively (Hallez, 2007; Pasqual-Marqui, 2002).

EEG data collected during a cognitive task can be analyzed in a variety of ways to characterize cognition including assessing event-related potentials (ERPs) (Thakor & Tong, 2004). ERPs are transient changes in the electrical brain activity in response to a stimulus and are quantified by latency (ms) and amplitude (μV) (Kok et al., 1997; Crowely, 2004). The waveform collected from the array of electrodes can be segmented as either positive (P) or negative (N)

going peaks or troughs over an epoch before or after the stimulus onset. One overarching goal of using dependent variables that have a temporal component is to differentiate separate cognitive processes or stages. For example, the early (<250ms) components of the neural response to a stimulus are often referred to as exogenous or stimulus-driven and are indicative of selective attention, perception and sensory processes while the later components (>250ms) have been argued to be endogenous and reflect higher-order cognition such as decision-making and/or response selection (Hillyard et al., 1998; Kok, 1997). Research using EEG to assess executive function has found that parallel, as opposed to serial, information processing occurs with a high degree of coordinated and overlapping neural involvement (Carpenter, 2000; Markela-Lerenc, 2004). Therefore, understanding the temporal sequences of information processing and integration during executive function tasks can allow for an examination of how cognition is impacted by a variety of variables including elevated fatigue associated with repeated high intensity exercise.

ERPs

Early ERP components have been labeled P100 (P1) and N100 (N1) and they appear approximately 20-150ms after the stimulus being generated from the temporal and occipital regions (Hillyard, 1998; Crowley, 2004). They have been interpreted as indices of automatic attention and the distribution of perceptual resources to achieve a sensory gain for the particular stimulus type (auditory, visual etc.) (Kok,1997; Ibanez, 2012). N1 occurs after P1 and is related to initial perceptual processing and object categorization (Hillyard, 1998). P200 (P2) occurs 150-250ms after the stimulus and has often been analyzed in combination with the N1 peak. However, research supports a functionally independent significance of P2 during cognition in terms of withdrawing attention from irrelevant stimuli (Crowley, 2004). N200 (N2) occurs 200-

380ms and is related to perceptual feature detection, novelty and response preparation. This peak has been associated with response conflict monitoring during a go/no go task and localized in the anterior cingulate cortex (Nieuwenhuis et al., 2003), a region responsible for the integration of physiological and internal information (Vogt, 2005). As these components occur early in the processing stream, they reflect automatic orienting attention toward relevant information and basic sensory perception and are generally not considered to reflect higher-order executive function processing.

A large positive going peak occurring 250-500ms after the stimulus has been labeled P300 (P3) and is associated with increases in attention allocation and task demands that require engaging higher-order cognitive processing, such as executive functions (Ibanez, 2012). The largest peaks are observed over the midline of the brain and have at least two generators at medial parts of the inferior temporal lobe for novelty (365ms) and in the frontal lobes (peak at 300ms) thought to be involved in target detection (Mecklinger, 1995). This wave component has been suggested to be part of stimulus evaluation in terms of updating working memory and detecting conflict/interference (classification speed) (Polich, 2007). Interestingly, as the task demand increases, P3 amplitude decreases suggesting possible information loss, greater resource allocation or inhibition of extraneous activity to focus on the relevant information (Kok, 1997; Polich, 2007). Arousal levels, personality and psychological illness have been shown to affect P3 latency and amplitude (Polich, 2007). P3 has been argued to have multiple intra-component peaks including a frontal/central region P3a response related to novelty/focal attention and a parietal region P3b response related to context-maintenance in working memory (Polich, 2007).

N400 (N4) is a negative going peak that occurs between 200-600ms after the stimulus and is largest over centro-parietal regions. This component is related to complicated aspects of

processing that integrate basic and higher-order cognition related to word expectancy, assessing meaningful similarities across modalities as well as semantic memory (Kutas & Ferermeier, 2011). Data from intracranial electrode recordings have identified N4s in regions such as temporal (medial, inferior and superior) and prefrontal (dorsolateral) cortices supporting its role in the processing of semantic memory and storage (Kutas & Federmeir, 2011; Ibanez, 2012).While the latency remains stable, the N4 amplitude can be manipulated by a variety of stimuli including complex language, math and auditory information. Due to the inconsistency in the research, some authors argue that the N4 reflects the retrieval of semantic meaning from long-term language or early lexical structure building and that information integration occurs later in the controlled processing stream (Hahne, 1999; Brouwer, 2012). However, because many cognitive processes are engaged across different regions during this time, the topography of the N4 is not well defined and may be a general index of coordinated distributed system communication (Kutas & Federmeier, 2011).

A final later component assessed in executive function tasks is the positive going wave occurring between 300-700ms, the P600 (P6). This wave has been argued to be functionally different from the earlier occurring P3 wave (Frische, 2002) and involved in emotion, language comprehension and argument violations (Kutas, 2011; Ibanez, 2012; Schupp, 2006). The P6 may reveal a second pass parsing of language processing that occurs when conflict or violation has been detected. In a recent review on the P6, Brouwer et al., 2012, concluded that this wave is better conceptualized as an assessment of whether the presented information matches that of the information retrieved from short or long-term memory for the specific task (N4) (retrieval integration). Thus, the N6 amplitude can be influenced by difficulty when the two sets of information do not match.

In executive function tasks, both early and late ERP components have been used to study interference and inhibition. Boenke et al., 2009, presented participants with stimuli that were geometrical shapes with a global feature G and a spatially more confined inner local feature L. The shapes of these contours could be either triangular or rectangular and thus congruent or incongruent. Participants were instructed to respond to the local feature by pressing one of two keys for either shape. Neither P1 (peak at 114ms) nor P3 (peak at 414ms) differed across trial types. During incongruent trials both N1 (peak at 174ms) at parieto-occipital areas and N2 (peak at 281ms) at fronto-central regions were significantly more negative compared to congruent trials. The authors suggest that the Stroop conflict may best be described as an early component that operates at the within-presentation level (170ms) and by a later component representing expectancy and memory. However, this task did not involve a reading/semantic conflict, which may occur later in processing and involve greater interference. A greater N2 and N4 during incongruent Stroop trials using words and color has been reported in one other study (Silton, 2010) but differences in N1 and P1 are not common findings suggesting that interference and conflict are detected and resolved at a later time.

Semantic and color conflict may be occurring during further down the line in processing and represented by decreased P3 amplitudes. Participants were instructed to respond whether the trial was congruent or incongruent in the color-naming version of the Stroop task and differences in P3 and P3b amplitude (but not latency) were detected across trials (Zurron, 2009). Further, P3 (peak at 290-330) and P3b (peak at 360-430ms) were significantly smaller for incongruent trials, suggesting that the incongruent trials were more difficult (Zurron, 2009) and that semantic conflict begins to occur during this time. Other research has identified greater grand average positive amplitude at 370-480ms in incongruent compared to congruent trials at frontal midline

electrodes but a more negative peak at central and parietal regions during the color-naming Stroop task (Badzakova-Trajkov et al., 2009) suggesting that different processes occurring at this time might be responsible the conflicting results. It was suggested that these regions were responsible for conflict monitoring and updating task demands. Further, this later time period may be capturing the P3b aspect of the response rather than a greater negativity in the earlier occurring P3a. These conflicting results may represent a general network for detecting, resolving and responding to conflict that involves different responses across separate regions.

Some studies have identified differences in the N4 between Stroop trial types as a primary outcome while other studies have identified this time as the first component of conflict detection followed by a later response-related component. Rebai et al., 1996, reported a significantly larger N4 for incongruent trials in both a list of only incongruent words as well as during a mixed list of congruent and incongruent trials with instructions to mentally name the color of the word. They also reported that differences were greatest at centro-parietal regions, which may be related to violations of target expectancy (Rebai, 1996). Liotti et al., 2000, tested the influence of the response modality (overt speaking, covert speaking or pressing a button) in the EEG responses during the Stroop. An early effect (350ms-500ms) (likely N4) had a smaller negative peak at medial anterior regions during incongruent trials in covert and overt responses while the manual response had a more posterior and broad distribution. The authors suggest that the early negativity originates in the dorsal ACC and relates to word meaning suppression. A similar pattern was described by Hanslmayr and colleagues (2008) who examined EEG differences across neutral, congruent, incongruent and negative priming Stroop trials between 400-500ms post-stimulus. The more difficult trial types (incongruent and negative priming) showed greater negativity in fronto-central regions but greater positivity at fronto-polar regions

supporting the involvement of several processes at this time involved in task complexity and conflict identification.

Finally, differences in positivity during later processing have been described. A late effect (500-800ms) was indicated by a greater later positive complex in incongruent compared to congruent trials at the left posterior superior scalp region (Liotti, 2000). This late effect was interpreted as representing reactivation of regions involved in word meaning retrieval. An additional study identified a later component (600-800ms) characterized by greater negativity in fronto-central regions and greater positivity in parieto-occipital areas (Hanslmayr, 2008). Source localization identified the ACC as driving the observed increased negative waveforms during incongruent trials (Hanslmayr, 2008). Finally, a prolonged positivity was identified during incongruent trials (600-1000ms) over parietal regions (Markela-Larenc, 2004). This later processing is likely related to successful irrelevant information inhibition and correct response selection and activation related to motor planning.

Research on ERPs is a useful tool to assess the temporal cascade of neural responses to a stimulus. For the proposed study, ERP data collected during the Stroop task will be the primary dependent variable. Analyzing these outcomes will allow for the characterization of how both early and late components change during a challenging cognitive task are impacted by increases in training volume across an athletic season. Further, this data will be used to examine how changes in reported mood and stress are related to objective changes in cognition.

Impacting Cognition: Fatigue and Exercise

Cognition is affected by a number of internal states such as fatigue and alertness. Due to the relatively small amount of literature examining these relationships, an additional EEG outcome will be introduced because it, along with ERPs, has been minimally measured. The

EEG signal can be deconstructed into its component spectra, or frequencies, by using specific filter boundaries and Fast Fourier transformation (Tonner & Bein, 2006). The synchrony of neural oscillations can be used to enhance or inhibit neural communication at rest or during tasks and has been interpreted as specific and global indexes of neural communication (Basar, 2008). Although frequencies are not analyzed to examine the fast event-related neural response to an event, both slow and high wave frequency bands appear to have functions during cognitive processes. Alpha (8-12Hz) activity is involved in active cortical-cortical network inhibition of irrelevant information, is related to mental effort but has not been shown to be directly related to executive function (Fruenberger, 2011). Beta frequency (12-20Hz) is involved in levels of arousal, motor responses, large-scale communication and is often coupled with higher frequency activity during exposure to novel events (Haenschel et al., 2000; Neuper & Pfurtscheller, 2001). Finally, the theta band (4-8Hz) is present during information encoding, retrieval aspects of memory and task difficulty (Sammer et al., 2007). Component spectra and ERPs have been analyzed to measure how illness can impact cognition (Neu, 2011; Sherlin, 2007; Flor-Henry, 2010), how cognitive function changes with the induction of mental fatigue (Barwick, 2012; Boksem, 2005) and how cognition can be affected by exercise (Kamijo, 2011; Hilty, 2011).

Fatigue

Due to the nonspecific, multifaceted and difficulty in measurement (self-report or objective decreases in performance), the definition of fatigue has been an object of debate in the literature (DeLuca, 2005). Some authors suggest that central fatigue should be defined as reduced motor output resulting in a decreased ability to generate force at the muscle as measured by increases in motor-evoked potentials and silent periods (Chen, 1999; Benwell, 2006). Other authors propose that central fatigue should be separate from peripheral markers and focus on

aspects of central nervous system functions such as sensation, cognition and neural activity (St. Clair Gibson, 2006; Meeusen, 2007; Leavitt, 2010). While the location of central fatigue in the brain has not been definitively identified, results from brain imaging suggest that a distributed network is involved (Cook, 2007; Schamling et al., 2003). The neural correlates of chronic and acute fatigue have been assessed, to a degree, in disease populations with high levels of self-reported fatigue and healthy populations and using EEG.

One way to assess the relationship between fatigue and cognition is to study patient populations that report high levels of fatigue. Chronic Fatigue Syndrome/Myalgic Encephalomyelitis (CFS/ME) is a complex illness, which includes myriad unexplained symptoms. Interestingly, between 50-95% of CFS patients report cognitive problems including impaired concentration, memory and language (Michiels & Cluydts, 2001). A plethora of studies have addressed a range of cognitive complaints in CFS and how behavioral performance on these cognitive tasks differs from normative data with mixed results (Michiels, & Cluydts, 2001; Tiersky, 2001; Jongen et al., 2012; Bertolucci et al., 2013). CFS patients describe mental work as aversive (Wearden & Appleby, 1996) and they tend to perform to a lesser degree than controls on most neuropsychological tasks but this difference often fails to reach significance. One conclusion from this literature is that CFS patients are impaired at integrating information at a quick pace, such as during challenging executive function tasks. The mechanisms driving these deficits have been debated. Defining and measuring cognitive function as it relates to real-life situations can be difficult and when combined with the general heterogeneous make-up of the CFS population, a clear mechanism of cognitive impairment has been difficult to define. On one hand, it has been proposed that because performance is usually within the normal range, CFS patients have impairments in their perception of fatigue or effort and mental abilities (Knoop,

Prins, Moss-Morris, & Bleijenberg, 2010). However, some research that has identified different neural correlates of fatigue at rest and during cognition in this population compared to healthy controls.

At rest, CFS patients exhibit significantly more theta activity across the scalp compared to healthy controls and another matched sleep-disordered population with cognitive complaints (sleep apnea-hypopnea syndrome) (Neu, Kajosch, Peigneux, Verbanck, Linkowski, & Le Bon, 2011). Resting theta activity did not correlate with ratings of fatigue (Neu et al., 2011). The authors concluded that the high prevalence of theta activity at rest may contribute to the report of global cognitive impairment in CFS patients. In addition, EEG was recorded during rest in a study of monozygotic twins discordant for CFS, (Sherlin, Budzynski, Budzynski, Congedo, Fischer, Buchwald, 2007). EEG frequencies and 3-dimensional spatial assessment using LORETA were performed. The twin with CFS showed significantly greater delta (2-3.5Hz) in the left uncus and parahippocampal gyrus and significantly higher theta in the cingulate gyrus and right precentral gyrus at rest (Sherlin et al., 2007). Thus fatigue, as an internal state can be quantified as differences in resting brain activity using EEG.

Neural activity during executive function tasks in patients with CFS using different modalities of brain imaging has been examined. Using fMRI, one study reported an extended recruitment of neural regions when compared to healthy controls during a mentally fatiguing Paced Auditory Serial Addition Test (PASAT) (Cook, O'Connor, Lange, & Steffener, 2007). Interestingly, ratings of mental fatigue during the PASAT predicted neural responses in parietal regions (negatively), temporal, cerebellum, cingulate, hippocampal and frontal regions (positively). Although this finding does not provide information on the temporal processing of

information in CFS, it provides, at least in part, a viable explanation of how cognitive function differs in this population.

EEG data were collected at rest as well as during two spatial cognitive tasks in patients with CFS using word finding and dot localization and analyzed for differences in frequencies and sources (Flor-Henry, Lind, & Koles, 2010). Compared to controls, CFS patients showed decreased alpha and beta bands in the right hemisphere during the word finding task. Further, during word finding, CFS patients demonstrated an increase in the left frontal-temporal-parietal regions of the beta band. During the dot finding task, CFS patients showed greater source current density at nearly all leads supporting the idea of additional neural recruitment to perform a task. Unfortunately, this study did not assess whether any of the regions or frequencies correlated with reports of fatigue and included tasks that did not specifically assess executive functions.

Finally, ERPs have been assessed during cognition in CFS with slightly less conclusive results. Scheffers et al., 1992, reported that no group differences between CFS patients and matched controls were found during an attention or visual oddball task in N1, P2, N2, frontal P3 or behavioral measurements. The authors concluded that CFS patients were capable of appropriate attention allocation and inhibition of irrelevant information. No group differences between healthy controls and patients with CFS for N1, P1, N2 or P3 during an auditory discrimination task have also been reported (Polich, 1995). Finally, the results of one ERP analysis during an auditory discrimination task (frequency and duration of a target tone) showed significantly longer N2, P3 latencies and response time in a group of ME patients (Prasher, 1990). Additionally, P3 latency was more related to response time in the healthy controls compared to the ME group suggesting that while the stimulus categorization may be unaffected in ME, the evaluation stage is impaired. The inconsistent findings regarding ERP amplitude in

this research are likely because of the greater variability in patient responses and the low difficulty of the employed tasks. Consistently longer ERP latencies during executive function tasks have been reported in the CFS population.

Another illness with high amounts of fatigue and cognitive impairment is multiple sclerosis (MS), an inflammatory demyelinating disease of the central nervous system resulting in lesions in the brain (Lori, 2011). EEG data during an extensive neuropsychological battery were collected over a 2-year period in a sample of healthy controls and pediatric patients with MS. ERPs to an auditory odd-ball paradigm task (the target was the rare tone) were analyzed for latency and amplitude. At the baseline measurement, MS participants had significantly increased N1, N2, P3 and P3b latencies and smaller P3b amplitudes. Of the six MS participants, four were categorized as cognitively impaired. For these patients, P3a and P3b latencies were more than two standard deviations greater than the healthy controls. Both P3 latency and amplitude correlated with the number of failed cognitive tests. At the 2-year follow-up testing, P3 latency was significantly increased in MS patients (Lori, 2011). In addition, increased N2 and P3 latency during visual and auditory oddball tasks accompanied by reduced P3 amplitudes has also been reported in adults with MS compared to healthy matched controls (Piras, 2003). Further, visual N2 latency and P3 latency and amplitude correlated with a cognitive deterioration index calculated from the Wechsler's formula. P3 latency correlated with frontal horn and brain stem lesions. The authors concluded that in MS patients, both attention orientation and stimulus discrimination are impaired (Piras, 2003). While consistent longer ERP latencies were again reported and decreased P3 amplitude in the MS patient population, it is impossible to separate the complications of the specific illness from the measured cognitive impairment and fatigue.

Another way to study the influence of fatigue on cognition is to induce mental fatigue in healthy participants using long and challenging cognitive tasks. Faber et al., 2012, examined the effects of mental fatigue on selective attention for targets and flankers of various sizes in a modified Eriksen flanker task, an executive function task that requires inhibiting responses during incongruent trials, in a group of healthy volunteers. Participants completed a testing session consisting of six blocks of 20-minutes of mixed incongruent and congruent flanker trials with small, medium or large letters for targets or flankers (H and O). Subjective fatigue was measured between each block of trials by asking participants to rate how much resistance towards performing the upcoming task they felt from 0 (none at all) to 9 (extremely much). With time on task, subjective fatigue and errors increased while reaction time for only incongruent trials increased significantly. The grand average ERP data showed that N1 amplitude decreased with no change in P1, N2, or P3. However, from visual inspection, the positive deflection at 300-400ms (likely P3) decreased with time on task only for large target letters (time by relevance interaction). The authors concluded that the 300-400ms epoch is when relevant information is being selected and irrelevant information is being actively suppressed. Mental fatigue was concluded to impact both early regulation of neural activity (N1) and later processes involved in the selection of relevant and irrelevant information (Faber et al., 2012).

Kato et al., 2009, examined the effect of mental fatigue on resource allocation and error monitoring using an executive function task (Go/NoGO) requiring response inhibition. During this task, participants were asked to respond to a target (triangle) but to inhibit their response to another target (circle). During the experimental session, participants completed 12 blocks of 120 trials, which lasted for approximately 1 hour. Performance across the blocks was summarized into four portions. Participants rated their mental fatigue using the Fatigue Scale before and after

the session. Ratings of mental symptoms and physical symptoms of fatigue, response times and errors all increased significantly over time. NoGo N2 latencies were significantly longer during the third portion of trials compared to the first two with no change in amplitude. Go trial P3 amplitude did not change over time, but latency increased significantly during the third portion of trials. NoGo P3 amplitude decreased and the latency increased significantly during the third portion of trials (Kato et al., 2009). Thus, during the more challenging trials, healthy adults demonstrated quantifiable changes in early and later information processing speed.

The physiological changes related to fatigue and attention during a 3-hour neurocognitive battery has also been examined in healthy adults (Boksem, 2005). For the task, participants completed 50 blocks of 160 memory trials to induce mental fatigue. The task was a memory set of two letters and target locations with relevant and nonrelevant cued locations. At the beginning of each block of trials, participants were given a memory set and instructed to attend to the relevant display positions as indicated by a cue frame and, in case of the occurrence of a target on one of these positions, to release their finger from the response button as quickly as possible. Participants were asked to rate their level of resistance to performing the task from 0 (no aversion) to 10 (maximum aversion) after every 10^{th} block. The behavioral results showed a significant slowing of response times and increased missed targets, false alarms and self-reported task aversion with time on task. P1 amplitude did not change with time, N1 amplitude decreased for all trials and N2 amplitude increased with time on task only for stimuli presented on the irrelevant diagonal. Additionally, the authors analyzed the EEG data by spectral power (frequency) and found that oscillations in the range of theta (3.5-7.5) increased with time, lower alpha (7.5-10Hz) increased with time, and beta (12.5-30Hz) power increased with time. Finally, the increase in lower alpha power was positively related to the increase in self-reported task

aversion (Boksem, Meijman, & Lorist, 2005). This frequency band has been interpreted to be involved in local cortical inhibition during cognitive processing and efforts to stay alert (Klimesch et al., 1999). The authors concluded that their task did induce mental fatigue and that this fatigue can be characterized by a reduced amplification of the responsivity to both relevant and irrelevant stimuli, which then leads to a reduced discrimination process.

Using the Stroop task in healthy young adults, Barwick et al., 2012, assessed fatigue-related changes in brain activity throughout a 90-minute neurocognitive battery (Barwick et al., 2012). The Stroop was administered at the beginning, middle and end of the battery. EEG was recorded using a 64-electrode cap and fatigue was measured using the Beatty 16-item inventory at the beginning and end of the neurocognitive battery. Total fatigue was significantly higher at the end of testing compared to baseline and more errors and longer reaction times were found during the incongruent trials. Theta activity increased over time during the incongruent Stroop task trials at central and frontal sites while Beta peak frequency decreased at central and parietal regions. Alpha power increased at frontal and occipital regions during incongruent trials and a shift of alpha power from posterior to anterior regions with time was reported (Barwick et al., 2012). The results of this study strongly suggest that acute fatigue is results in significant changes in neural responses during executive function tasks.

Based on the previous literature on central executive functions and EEG, it is reasonable to suggest that any changes occurring in cognitive function in response to high amounts of exercise training can be characterized using this measure. Unfortunately there are no studies of fatigue and ERPs during the Stroop task. Another challenge when interpreting this research is that the chronic fatigue observed in the patient populations might be inherently different due to their specific illness. Further, the acute fatigue induced in healthy adults might be different from

what occurs in a somewhat more chronic state. From the literature on fatigue and executive function, behavior is impacted (slower reaction times and more errors) and ERP latency of both early and later components is increased. Further, from other brain imaging techniques, it can be surmised that additional regions of the brain are recruited during the more difficult incongruent trials in a fatigued state. Finally, although there are few correlations between ratings of fatigue and cognitive function, it could be hypothesized that increases in fatigue are related to brain responses during executive function tasks.

Exercise

Examining the relationship between exercise and cognitive function is a burgeoning area of research. A number of hypotheses have been proposed to explain the why exercise might impact cognitive function including exercise-related increases in neuroendocrine activity, elevated arousal, enhanced prefrontal activation along with increases in angiogenesis and neurogenesis (Dishman, 2006; Tomporowski & Hatfield, 2005). In order to better understand the impact of exercise on cognition, research has tested cognition during and following an acute bout of exercise, across interventions in older adults, and across fitness levels.

In the acute exercise setting, cognitive performance has been assessed before, during and after a variety of exercise modalities and intensities. Research has demonstrated that similar improvements in cognition are observed after moderate intensity aerobic and resistance training. Chang and colleagues (2013) assessed the influence of an acute bout of resistance training (70% of 10-repetition maximum for seven exercises) and a control condition on aspects of a modified Stroop task in older adults. Greater improvement after the resistance exercise training session was observed in all Stroop conditions (Chang et al., 2013). Alves and colleagues, (2013), asked middle aged women to complete 30-minutes of aerobic exercise session (at 50-60% heart rate

reserve), resistance training session and a control condition in a counterbalanced order before completing the Stroop color-naming task. Participants had significantly better performance in Stroop conditions after both exercise bouts compared to the control condition were observed (Alves et al., 2013). Similar results have been reported in college-aged students. Working memory (the Sternberg task) was tested immediately before, immediately after and 30-minutes after: 1) an acute bout of aerobic exercise at 60-70% $V0_2$ max, 2) a bout of resistance training of three sets of 8-12 repetitions at 80% 1-repetition maximum for seven major muscle groups and 3) a seated-rest control condition (Pontifex, 2009). Reaction times improved immediately and 30-minutes after the bout of aerobic exercise when compared to the pretest but only for task conditions requiring increased working memory capacity suggesting that the mode of exercise and the complexity of the cognitive task are important. Thus, acute moderate exercise improves aspects of cognitive function in healthy young adults.

Cross sectional research also supports a beneficial impact of higher fitness on cognitive function. Positive relationships between regular exercise and cognition have been consistently reported across the lifespan. A meta-analysis of exercise interventions in children (6-16 years of age) reported an effect size (Cohen's *d*) of 0.28 on cognitive outcomes and academic achievement, including improvements in math, reading and English (Fedewa et al., 2011). This effect was larger for exercise interventions that combined both aerobic and strength exercises, occurred in groups of less than 10, included 36-70 total hours of activity and occurred on three days/week. A meta-analysis on the effects of exercise interventions in adults older than 18 years reported an effect size (Hedge's *g*) of 0.158 on attention and processing speed and an effect size of 0.123 on central executive function. These effects were not related to exercise duration or intensity (Smith et al., 2010). Another meta-analysis on the impact of exercise interventions on

cognitive function in older adults reported an overall effect size (Hedge's *g*) of 0.478 (Colcombe et al., 2003). The effect size for executive function was $g = 0.68$ and was significantly larger for spatial and simple reaction time /finger tapping tasks (Colcombe et al., 2003). A more recent meta-analysis in older adults reported an effect size of 0.26 for aerobic exercise on cognitive function when controlling for practice effects on the cognitive tasks (Hindin et al., 2012). Therefore, exercise in a variety of modes appears to have small to moderate positive effects on aspects of cognitive outcomes.

The impact of exercise interventions on cognitive function has been primarily tested in older adults. Changes in executive functions were examined in healthy sedentary older adults who were randomly assigned to either a three-month aerobic exercise intervention or a control group (Predovan, 2012). Participants worked up to 40-minutes of exercise, but there was no measure of intensity. Baseline and post-intervention measurements included a modified Stroop color-naming task. After the intervention, the exercise group had significantly improved Rockport mile test performance and a greater improvement score only during the inhibition/switching condition of the Stroop color-naming task during which participants were cued to either read the word or respond to the color of the word. Finally, the increase in aerobic capacity was inversely related to the reaction time at post-test during the inhibition/switching condition of the Stroop task (Predovan, 2012) suggesting beneficial impacts of chronic exercise during challenging executive function tasks.

Additional research has suggested that the influence of exercise training on cognition may be based in neural adaptations. Colcombe and colleagues (2004), reported that older adults with higher fitness, as determined by the Rockport mile walk test and adults completing a 6-month aerobic exercise intervention at approximately 60-70% heart rate reserve demonstrated

different brain activity patterns (using fMRI) during interference trials of the Flanker task. Fit adults had greater signal in attention-related brain regions when compared to unfit adults or a stretching control group. The results included increased signal in the middle and superior frontal gyrus and superior parietal lobule with decreased signal in the anterior cingulate cortex (Colcombe et al., 2004). These results support the role of increases in physical activity in older adults, aerobic or resistance training, improving higher-order executive function. Thus, several bodies of exercise research support the positive relationship between both acute and chronic physical activity and executive function (behavior and brain responses).

Additionally, assessing the influence of exercise on electro cortical activity has been collected. In a meta-analysis, it was reported that exercise increased all measured frequency bands by moderate effect sizes including delta (*SD* = 0.50), theta (*SD* = 0.53), alpha (*SD* = 0.54) and beta (*SD* = 0.38) when collected after, but within 30-minutes post-exercise (Crabbe et al., 2004). These changes were not region or hemisphere specific suggesting exercise has a global effect on brain electro cortical activity. A more recent study examined EEG in young healthy and older adults at rest before and after 20-minutes of cycling at 80% age-predicted maximal heart rate using standardized low resolution brain electromagnetic tomography (sLORETA) across 20 electrode sites to identify the activity source (Moraes et al., 2011). For young adults (average age of 25 years), increases in theta, alpha and beta and lower delta at post-exercise were observed compared to the pre-exercise measurement while no significant changes were observed for older adults. Furthermore, a significant increase from pre to post-exercise was found in alpha at the cingulate gyrus and for beta in the anterior and posterior cingulate cortex (Moraes et al., 2011). Thus, EEG changes in response to an acute bout of moderate exercise have been characterized as global increases in all bands in young adults. Notably, it is unclear whether or not the EEG

changes associated with repeated high-intensity exercise bouts would influence or translate into benefits or potential impairments in central executive functions.

Fitness and enhanced cognition might be related through a number of mechanisms including greater attention allocation, more efficient error monitoring and greater flexibility during challenging cognitive tasks. Specifically, EEG during cognition after an acute bout of exercise has been examined in high-and low-fit individuals. One cross sectional study aimed to examine the underlying brain function associated with academic achievement in high- and low-fit young adults (average age = 19) and children (average age = 9) (Hillman, 2006). Participants were asked to complete an oddball task. This task requires individuals to withhold their response to the common target (80% probability) and to respond to a less frequent target (20% probability). Fitness was determined using the *fitnessgram* assessment, which includes a measure of aerobic capacity, strength, flexibility and body composition. Individuals that fell into the top 10% and bottom 10% were selected for the high- and low-fit groups, respectively. EEG data were collected and analyzed for P3 amplitude (275-775ms post stimulus). Adults and high-fit participants had faster reaction times to the target compared to children and low-fit participants (Hillman et al., 2005). Further, high-fit children were significantly faster than low-fit children and had significantly larger P3 amplitudes than all other groups. The authors concluded that based on their findings, higher-fit individuals had greater attention allocation and faster information processing compared to low-fit individuals.

This same research group examined the influence of fitness in younger and older adults during a task-switching paradigm (Hillman, 2006). Four total groups were composed of older and younger physically active and sedentary individuals. Each participant completed the Yale Physical Activity Survey for Older Adults to estimate fitness. The cognitive task included 3

separate blocks. Two blocks included only task-homogenous trials asking participants to respond to weather the presented number is greater than five or is the number odd or even. The third block included task-heterogeneous trials, which included both a non-switch trials (repeated) and trials that required them to switch the task. Trial types were cued using a solid or dashed presentation prior to the number. EEG data were collected and P3 was defined as the largest positive-going peak between 275-750ms post stimulus presentations. Reaction times were faster for younger adults and physically active adults compared to sedentary adults. No differences in N1, N2 or P2 were observed across condition or group. P3 amplitude was larger at midline sites (i.e. Cz was greater than C3 and C4) in active compared to sedentary participants during the easier homogenous blocks. During the heterogeneous block, active participants showed significantly increased P3 amplitude at midline compared to lateral regions while the sedentary participants did not. Shorter P3 latency was observed in active participants compared to sedentary participants during the heterogeneous block of trials. The authors concluded that being physically active facilitates perceptual and central processing of central executive functions. This study is limited in the self-report estimation of fitness and the relative simplicity of the cognitive task.

Several other studies have examined the influence of fitness on cognitive performance at rest and following a bout of moderate-intensity exercise. Themanson et al., 2006, evaluated the influence of cardiovascular fitness and acute exercise on neuroelectric activity during an executive function task in healthy college students (Themanson, 2006). Participants completed the Eriksen flanker task after 30-minutes of rest and after 30-minutes of a ‘somewhat hard’ to ‘hard’ exercise bout. Based on maximal exercise testing, a high-fit group was formed of individuals scoring above the 80th percentile and a low-fit group near the 50th percentile of

normative gender and age values. Error-related negativity (ERN) amplitude, the maximum negative peak from 0-200ms post-incorrect response, at the frontal central midline electrode site (FCz) was significantly smaller for the high-fit group compared to low-fit participants during error trials. N2 amplitude showed no difference between groups. Finally, the higher-fit group had significantly longer reaction times for correct trials following an incorrect trial, suggesting they may have a different error-related behavioral response than sedentary participants. Acute exercise did not influence any ERP measurement. The authors concluded that higher-fit individuals exhibited a relative decrease in ERN, thought to occur in the ACC, indicated by the smaller ERN amplitude during errors. Thus, being physically active may improve top-down attention control surrounding an incorrect response.

A similar study assessed the relationship between fitness and cognitive flexibility during the Eriksen flanker task in healthy young adults with instructions emphasizing the importance of either speed or accuracy (Themanson 2008). The accuracy instruction group was significantly more accurate and slower than the speed instruction group. Fitness (measured by a maximal exercise test) did not impact the behavioral performance of either instruction group. ERN amplitude was greater in the accuracy instruction group. Further, in the accuracy instruction group, higher fitness was associated with larger ERN amplitude independent of gender while there was no influence of fitness in the speed instruction group. The authors concluded that fitness is associated with an increased flexibility in the implementation of cognitive control as evidenced by larger ERN amplitude during the accuracy instructions. However, in the previous study, smaller ERN amplitude during an Eriksen flanker task was concluded to indicate more efficient error-monitoring. These inconsistent results may be a reflection of complicated

relationships between fitness, exercise and cognition in addition to the difficulty of interpreting EEG components.

Stroth et al., (2009) examined the relationship between fitness, acute exercise and executive control in healthy adolescents using ERPs. In order to determine fitness, participants completed a graded maximal exercise test. High and a low fitness groups were formed based on the median fitness score for both girls and boys. Participants completed a combination of a go/nogo and an Eriksen flanker task on two separate days, once after 20-minutes of rest and once after 20-minutes of exercise at 60% maximal heart rate. During the task, participants were presented with a string of 5 letters and instructed to respond to the center letter with a key press if the letters were B or U and to withhold their response for the letters D and V. Reaction time and response accuracy scores during the task were recorded. The EEG data were analyzed for the contingent negative variation (CNV), a measure of anticipation and expectancy of a stimulus (450-350ms prior to the stimulus), N2 (240-300ms post stimulus), and P3 (340-440ms post stimulus). The higher fitness group showed enhanced cognitive control when compared to the low-fit group after the rest condition. Specifically, higher-fit participants had greater CNV amplitude and smaller N2 amplitude at the fronto-central electrodes than the low-fit group. The P3 measurement did not differ across conditions or groups. No behavioral differences across conditions or between groups were found and no group differences were observed for any cognitive outcome after the 20-minute exercise bout. The authors concluded that the higher-fit adolescents demonstrated stronger task preparation and more efficient task monitoring during the cognitive task.

Scisco et al. (2008) examined the relationship between cardiovascular fitness and different components of the P3 wave during a cognitive task in healthy young adults. Fitness was

determined using the YMCA protocol, which, in this study included two successive incremental workloads. Participants were grouped based on whether their scores fell at or above the 70th percentile (high-fit) or below the 30th percentile (low-fit) for their gender. All participants completed an executive control task that had four separate trial types. Depending on the type of symbols presented with the target digit(s), participants had to indicate if the target was odd or even, greater or less than 50, was the sum of the two digits greater than 10 or was the sum of the two digits odd or even. ERPs consisted of N1 (150-200ms post stimulus), P3a (250-300ms post stimulus), and two measurements of P3b (300-450ms and 475-525ms post stimulus). No behavioral or EEG differences across groups were observed. Although this was not a widely used executive function task, the authors concluded that cardiovascular fitness is not related to more efficient cognitive control in healthy young adults.

A recent study used EEG to assess the relationship between physical activity and functional connectivity during an executive cognitive task. Kamijo et al., 2011, asked young healthy adults to complete a spatial priming task while wearing a 19-site EEG cap. Physical activity was measured using the International Physical Activity Questionnaire. Active and sedentary groups were formed based on their physical activity scores. During the cognitive task, participants were instructed to respond to the presentation of the target letter ‘O’ and to ignore the letter ‘X’. These letters could be located in one of four squares and the location of the target and distractor letter served as cues for the subsequent trial. If the target of the previous trial was in the same position as the target in the subsequent trial, this was termed ’positive priming’ and if the distractor of the previous trial was in the same location as the target for the subsequent trial, this was termed ‘negative priming’. The EEG data were analyzed for phase-locking values, which are a reflection of the phase synchrony across two electrodes during the trials. This

measurement can be interpreted as functional connectivity between regions. Synchronization was assessed in both the beta (14-30Hz) and gamma (32-60Hz) frequencies and across epochs of time following the stimulus presentation. During the 300-400ms post-stimulus epoch, active participants had greater synchrony in the beta frequency band during both priming conditions compared to control trials. The sedentary group did not show any difference in phase-locked values between the two trial types. No differences across groups were found in the gamma frequency. Finally, based on a graph theoretical degree analysis, it was concluded that brain structure was similar across groups. Thus, the differences between groups in the beta phase-lock values were due to functional rather than structural differences. The authors concluded that being physically active is associated with greater neural connectivity and efficiency during challenging cognitive processes (Kamijo et al., 2011).

The relationship between exercise and cognition appears to be positive and ranges from small to moderate in size depending on the type of exercise used and population tested. However, the mechanisms driving these effects are not clear. Moreover, the impact of repeated high-intensity exercise on this relationship is unknown. It is plausible that a dose-response relationship between exercise intensities and cognition exists. Research that examines the effects of both “low” and “high” intensity chronic exercise on cognition would help clarify this relationship. Further, athletes show signs of central nervous system dysregulation, such as increases in fatigue and depression during peak training. It is not known if they also experience disrupted executive functions during this time. This is important to consider as collegiate student-athletes have important academic responsibilities during peak training and their performance could be impacted if cognition is negatively affected.

Athlete Overtraining and Cognitive Function

One aspect of overtraining that is highly relevant but poorly understood for the student athlete is the influence of repeated high-intensity exercise on cognitive function. If central nervous system dysregulation occurs during athlete overtraining, cognitive function would be impacted. Unfortunately there is a paucity of research on the way that cognition changes in response to increases in training volume. Based on the similarities between OTS, major depression and chronic fatigue syndrome, a number of authors have hypothesized that psychomotor speed, or the speed of information processing and response during a cognitive task will be slower in athletes showing signs of overtraining. One of the first articles addressing this hypothesis was completed with 14 healthy cyclists who did not change their training regimes and 14 cyclists who completed an intense 9.5-day cycling training camp (Nederhoff et al., 2007). Baseline values of the REST-Q, which measures general stress, general recovery and sport specific stress and recovery (Kellmann, 2000), POMS, finger-cuing task, a determination task (reaction and inhibition time) and maximal exercise capacity were collected 1-2 days prior to the commencement of the training camp, after the camp ended and 2-weeks after the camp had finished. Athletes were diagnosed as functional overreached (FO) based on two out of three objective decrements in performance and at least one subjective report of mood disturbance on the POMS or REST-Q after the camp ended. Five athletes met these inclusion criteria and seven athletes were identified as well-trained after the completion of the training camp (two participants were excluded for disturbed stress-recovery balance at baseline). No differences between groups were observed in reaction time during the determination task or the finger-cuing task at baseline. While statistical significance was not reached, the greatest group difference was seen after the exercise camp between the FO and control athletes during the most difficult trials of the finger-cuing task (uncued and neither-cued). The authors concluded that the performance

of athletes exhibiting more severe symptoms of overtraining on have impaired performance during challenging cognitive tasks and this may be a useful tool in identifying FO athletes (Nederhoff et al., 2007).

A similar intervention study aimed to describe the effects of a 2-week training volume overload on central executive function (Dupuy et al., 2010). This study collected data from 10 male athletes who were tested prior to and following 2-weeks of training. The athletes were asked to increase their training volume by 100% during this time. The testing days included the POMS, a simple reaction time test, the Stroop color-naming task, a maximal exercise test and a constant speed exercise test. During the simple reaction time task, participants were instructed to respond by releasing a home key (initiation time) to press a response key (execution time) when a target appeared on the computer screen. The Stroop task involved neutral trials consisting of colored non-color words, congruent and incongruent trials and switch trials requiring the participant to read the word out loud when presented with a specific cue. Following the completion of the study, participants were separated into a negative adaptation group (NAG) who were considered overreached and a positive adaptation group (PAG) based on their performance on the constant speed test. The NAG showed a significant decrease in performance from the first to second constant speed test while there was no significant change in the PAG. Furthermore, the NAG had significantly lower vigor scores and higher fatigue scores after the intervention on the POMS compared to baseline values (Dupuy et. al, 2010). The NAG had significantly slower initiation time on the simple reaction time test following the overload period compared to baseline values. Performance on the Stroop task improved for both groups from baseline to after the overload period on for the control trials and both congruent and incongruent trials. However, during the more challenging switching trials the NAG group performed slower

(ES = 0.44) while the PAG performed faster (ES = -0.59) after the overload. The authors of the study concluded that maladaptation to training volumes challenge attention control processes that are required for complex cognitive tasks (Dupuy, 2010).

Athletes with a diagnosis of OTS have also been compared to healthy athletes in terms of performance, mood and cognition. One study addressed differences between athletes in terms of heart rate variability (HRV) to standing and relaxing as well as performance on the Stroop color-naming task (Hynynen, 2008). Overtraining may attenuate autonomic nervous system (ANS) modulation as a result of repeated exercise training and lead to a change in sympathetic and parasympathetic balance during rest and stress. The study collected data from 12 healthy control athletes (6 women) and 12 (6 women) athletes who had been diagnosed with OTS by a medical doctor based on decreased performance, increased fatigue for 3-weeks and increased in training volume prior to symptom onset. All testing was completed 3-6 months after the diagnosis of OTS. Participants were asked to complete the perceived stress scale (PSS), the Stroop task and a maximal exercise test. During the Stroop task, each individual's performance threshold was calculated by using decreasing inter-trial intervals until the individual's performance was at 5-10% error and athletes completed a slow, moderate and fast condition. Athletes with OTS reported significantly more perceived stress on the PSS and significantly lower VO_2 max scores.. During both the moderate and fast inter-trial interval conditions of the Stroop, the overtrained athletes made significantly more errors than the healthy athletes. The authors concluded that overtrained athletes have lower cognitive performance compared to control athletes but that this finding is limited in the cross-sectional design of the study (Hynynen et al., 2008).

A similar study aimed to induce a state of overreaching by increasing training load and measuring several different potential biomarkers associated with overtraining (Rietjens et al.,

2005). The authors collected data from seven male cyclists that completed an intensified training camp and seven healthy age-matched cyclist controls. During the 2-week training intervention workload was increased by 100% and intensity was increased by 15%. Measurements of body mass, body fat %, time trial performance, workload, maximum exercise, peak lactate, blood components, an insulin tolerance test and a combined anterior pituitary test (CAPT) were collected 2-weeks before the intervention and following the completion of the training camp. Cognitive performance was measured using a finger-cuing task and mood was assessed using the POMS. Training load and RPE's significantly increased following the intervention but no changes in body mass, body fat, POMS or exercise performance tests or lactate values during testing were observed. Both summed cortisol concentrations during the CAPT and hemoglobin were significantly lower following the exercise intervention. During the finger cuing task, faster reaction times were recorded for all of the conditions in the post-test compared to the pre-test for all participants suggesting a learning or practice effect. Interestingly, the learning effect was larger for the control group compared to the intervention group but only for the most challenging conditions (uncued and neither-cued trials) (Rietjens, 2005). The authors concluded that the central nervous system fatigue precedes peripheral fatigue and that cognitive ability is an early biomarker of overtraining.

While the general impression of this research is that athletes showing signs of overtraining (decreased performance and increased fatigue) appear to perform differently on challenging tasks, this body of work is limited in a number of ways. First, some studies are selecting cognitive tasks that do not reflect a cognitive system. Neither the finger-cuing task nor the simple reaction time tasks will be sensitive to subtle changes in executive function. The Stroop task was used in a few studies with interesting results, but the sample sizes were

relatively small. Further, the interventions were likely not intense enough or long enough in duration to induce symptoms of overtraining that resemble those which occur across a season. Additionally, group inclusion is limited as the diagnostic criteria for overtraining varies across studies. Finally, none of these studies was completed in a longitudinal naturalistic design preventing any conclusion about what typically occurs to cognitive function over the course of an athletic season. Thus, it is unknown if behavioral aspects of cognition are sensitive to increases in training volume. Therefore, there are many gaps in the literature assessing the influence of prolonged high-intensity training on cognitive function.

Endurance athletes undergo increases in training volume to improve performance. Although many attempts have been made to define the psychophysiological response to increases in exercise volume, the pattern of healthy adaptation compared to the central nervous system dysregulation that occurs during athlete overtraining remains undefined. The literature suggesting a cognitive component to this process is in its infancy, currently lacks a strong theoretical foundation and has significant methodological limitations. In the interest of student-athlete health, more research is needed to characterize objective changes in cognition. Further, relationships with other variables such as cortisol, stress and mood states must be assessed to improve our understanding of central nervous system adaptation across a competitive season. The proposed study has been designed to better understand the potential impact of repeated high-intensity exercise on cognitive function.

Chapter Three- Methods

Participants

For all study aims, male and female rowers were recruited from the University of Wisconsin-Madison. Rowers were the targeted population because there are existing data on mood state and hormone changes over a competitive season in these athletes (O'Connor, 1989; Flynn, 1994). Volunteers from the University of Wisconsin-Madison varsity rowing team were recruited at the beginning of the 2014 spring semester. A control sample of healthy college students was included for group comparisons of cognitive performance, mood and perceived stress. Healthy controls will be matched with rowers for gender, age (±2 yrs), class credits (±2) for the semester and time spent on extracurricular activities (±5 hrs) such as work and club participation. Following the informed consent process, all participants were given an identification number for coding information. Only designated study personnel had access to participant information. This information was kept separate in a locked file in Dr. Dane Cook's laboratory (room 1060, 2000 Observatory Drive, Madison WI, 53706). All participants received $17 for each session they completed, which was $85 for completing the entire study.

Participant Recruitment

Study personnel were granted permission from the UW athletic administration and rowing coaches to meet with the rowers to introduce the study and ask interested volunteers to provide their contact information on a sign-up sheet. Study personnel then sent an email to interested athletes to schedule for their first session at the Exercise Psychology laboratory located in the Natatorium (2000 Observatory Drive, Madison WI 53706).

Student controls were recruited by flyers posted around campus with the exercise psychology laboratory phone number and email address during the beginning of the 2014 spring semester. According to the flyer, interested participants were instructed to call or email the

laboratory to schedule an appointment with study personnel at the natatorium to receive additional study information. Interested participants contacted the lab and scheduled a time to meet for their familiarization session.

Inclusion Criteria

Participants were included in the study if they were full-time sophomore, junior or senior college students between the ages of 18-25. Athlete participants were required to be current members of the University of Wisconsin-Madison rowing teams, not currently injured and regularly training with the team. Healthy college student controls were required to be free from injury that would interfere with their regular physical activity and have satisfactory academic status (e.g. not currently on probation). All healthy college student controls were required to report meeting the physical activity recommendations provided by the American College of Sports Medicine (Garber, 2011). This will require at least 20-minutes of moderate physical activity on five days a week on the Godin Leisure-Time Exercise Questionnaire (Godin, 1985). Control participants were also required to report at least 20 hours/week of work and or extracurricular activities. Participants were excluded if they have a history of traumatic brain injury, current thyroid or endocrine disease, attention or learning disability, current mental illness or taking analgesic, anti-convulsant or high-dose anti-depressant medications as these might impact cognition and hormonal levels.

Research Design and Procedures

Over the course of the study, participants completed a familiarization session and testing on four subsequent separate occasions. For all participants, each testing time point included: 1) cognitive testing with EEG, 2) completion of a battery of questionnaires, 3) a saliva sample. All testing occurred at the Exercise Psychology laboratory at the Natatorium. The familiarization

session took place during the beginning of the 2014 spring semester between January 21 and February 19th. This session included further description of the study and the informed consent process. Once the informed consent form was signed study personnel reviewed the inclusion criteria. Next, all participants reported their physical activity using the Godin Leisure-Time Exercise Questionnaire. Then study personnel introduced the procedures for each of the next testing sessions. This included practice trials for the cognitive task, practice opening an email link to the online survey, and introduction to the saliva collection procedures and the EEG cap. If participants did not meet study criteria or were not interested, they were thanked for their time and were not scheduled for any other study events. This session took approximately 20 minutes.

Each testing time was completed across a two-week period approximately one month apart. Due to scheduling and training conflicts, female athletes were tested during the first week and male athletes were tested the second week. When possible, testing occurred at the same time of the day and same day of the week for each participant. Testing schedules and sample sizes (n) in controls, female and male athletes are listed in Table 1. The first testing session (**time 1 = baseline**) took place in the beginning of the semester after the familiarization session. The second testing session (**time 2 = typical training**) occurred during typical training loads for both teams. The third testing session **(time 3 = peak training)** was scheduled to coincide with the highest training load as the teams approached their final competitions. The fourth testing session (**time 4 = recovery**) occurred after completion of the competitive season. For this final testing session, many participants had just graduated or were about to leave campus to start working. Although we tried to have at least 2-weeks after the season/semester, we collected data when the participants were available.

When participants came in for testing, they first completed compliance questions ensuring that they had not consumed caffeine or exercised in the last two hours. They were also asked about medications they had taken in the last 24 hours. Next, control participants completed the Godin Leisure-Time Exercise Questionnaire to quantify the amount of physical activity they completed the previous week. Then the cognitive task instructions were reviewed, the EEG cap was placed on their head, practice trials were completed followed by the test trials. After the completion of the test trials, participants were reminded about their surveys and saliva sample if they had not completed them yet. All participants were compensated $17/session, $85 total if they completed all testing sessions. Testing took approximately 30-minutes to complete.

Table 1. Testing Time Dates

	n	**1 (baseline)**	**n**	**2 (typical)**	**n**	**3 (peak)**	**n**	**4 (recovery)**
Controls	23	Feb. 2-24	22	March 25-April 3	22	April 29-May 8	19	May 29-June 27
Female Athletes	22	Feb. 3-10	21	March 24-April 4	21	April 28-May 1	19	June 2-20
Male Athletes	21	Feb. 10-19	20	March 31-April 4	20	May 5-12	17	June 11-23

Cognitive Testing and EEG

All participants completed the Stroop color-naming task for their cognitive testing. The Stroop is a sustained attention task that presents the words “red”, “green”, “yellow” and “blue” in text colors red, green, yellow and blue. The word trials are either congruent (the word “red’ appears in the color red) or incongruent (the word “red” appears in a different color). The Stroop is one of the most reliable tests of attention in cognitive science (MacLeod, 1992) and has been used extensively in cognitive and brain imaging studies of memory and attention (Belanger, 2002; Collette, 2002). The task was completed on a 19-inch Dell computer screen using E-Prime (Psychological Software Tools, Inc). During the consenting day, participants completed a

familiarization session, including instruction and 100 randomized practice trials (50 congruent and 50 incongruent). Participants were instructed to respond to the color of the text and ignore what the word said by pressing the corresponding keyboard button as quickly and accurately as possible. Colored paper was taped to the top of keyboard buttons to be used for their responses. Blue was "v", yellow was "b", green was "n" and red was "m". Participants completed 4 blocks of 25 trials and between each block was a rest period that lasted for 10-seconds. Each color-word was presented on the screen followed by a fixation cross for 1500ms. During the familiarization session, the speed of the color-word presentation got faster across blocks starting with a 2000ms presentation in the first block, a 1500ms presentation during the second block and a 1000ms presentation for the third and fourth block to acclimate them to the speed to be used for testing.

During each subsequent testing session, 60 (30 congruent and 30 incongruent) practice Stroop trials and 256 Stroop test trials (128 congruent and 128incongruent) were completed. For practice, participants completed three blocks of 20 trials with 10 -second rest periods between blocks. Color-word presentation was 1500ms for the first block and 1000ms for the second and third block of practice. For all test trials, color-words were presented for 1000ms, the fixation cross was presented for 1500ms, trials (congruent and incongruent) were presented randomly and participants finished four blocks of 64 trials with a 10-second rest period between each block. The test trials took approximately 12 minutes to complete. The EEG cap was placed on the participant's head either before the practice trials or the test trials depending on time constraints.

Participant's head circumference was measured during the first testing day and they were fitted with the appropriate 256 channel Geodesic EEG cap for measurement of brain electrocortical activity (EGI Geodesic EEG systems, Eugene, OR) for each testing day.

EEG data was sampled at 500Hz and amplified at 22.5 K. Before testing, impedance was measured and efforts were made to have all electrodes under 50 Ωs. Electrical geodesic Netstation software was used to analyze all EEG data. Raw data was bandpass filtered to include data between 0.5-30Hz and then segmented to include 200ms before the trial and 1000ms post-event for correct congruent and incongruent trials. Next Artifact detection was performed including specific algorithms to identify segments that had bad channels (maximum difference of 200μv), eye movements (maximum difference of 140 μv) or blinks (maximum difference of 55 μv). Further, inferences were performed if more than 20% of the channels in a segment were identified as bad. Segments were identified as 'bad' if it contained more than 10 bad channels, contained an eye blink or eye movement. Next, data were baseline corrected using the 200ms before the stimulus as the baseline. Then, a single average segment from the segments that were identified as 'good' was computed for congruent and incongruent trials per participant for each testing time. Grand averages were created using all the participant average data for congruent and incongruent trials to define the timing of event-related potentials (ERPs).

Previous research has provided the following parameters for expected ERP components. P100 (P1) and N100 (N1) occur within 50-180ms after the stimulus and have peaks ranging from 0.5 μv (P1) to -6 μv (N1) (Hillyard, 1996; Boenke, 2009; Liotti, 2000). P200 (P2) has an amplitude of 1 μv and occurs 150-250ms post-event (Crowley, 2004; Liotti, 2009) while N200 (N2) occurs between 200-380ms with an amplitude of -3.0 μv (Nieuwenhuis, 2003; Boenke, 2009; Badz). P300 (P3) peak amplitude ranges from 4-7.5 μv and occurs between 250-400ms after the stimuls (Kok, 1997; Boenke, 2009; Lew; Zurron). N400 (N4) occurs approximately 350-600ms after the event with peak amplitude of -1.2 μv (Kutas & Feremeier, 2011; Silton). P600 (P6) is a positive going wave occurring between 300-700ms after the stimulus with

amplitudes of 2-6 μv (Frische, 2002; Zurron, 2009). Based on previous research and after reviewing the grand averages from the student-athletes for both congruent and incongruent trials, the following parameters were set. After the stimulus onset, P1 was identified as the positive peak between 20-100ms, N1 was the negative peak between 75-165ms, P2 was the positive peak between 115-200ms, N2 was the negative peak between 175-275ms, P3 was the positive peak between 250-400ms, N4 was the negative peak between 350-475ms and P6 was the positive peak between 430-600ms. Finally, adaptive mean amplitude (μv) and latency (ms) for these epochs were identified in congruent and incongruent trials for each participant at each time point.

While efforts were made to keep impedance low and to ask the participants to stay as still as possible during the Stroop task, many participants had numerous segments labeled as 'bad'. The average amount of segments used in the computation of an individual's average for each time was 74/128 for congruent trials and 70/128 for incongruent trials. Grand averages were created using all the athlete data for congruent and incongruent trials to define the timing of ERP epochs.

Saliva Sampling

A resting state saliva sample was collected from participants at each of the four testing sessions similar to the methods used by O'Connor et al., 1989. A Salimetrics oral swab (Carlsbad, CA) was used to collect saliva. The cotton swab was handed to the participant and study personnel instructed them to place the swab under the tongue until it was saturated (approximately 60 seconds). The participant then placed the swab into a protective tube. Each tube was labeled with the participant's identification number and stored at -80º Celsius in a freezer at the Natatorium after collection. For athletes, study personnel went to the Porter Boathouse during the Wednesday of the women's and men's testing week from 2:00-4:00pm to

collect saliva samples. Controls provided their saliva sample between 2:00 and 4:00pm on the Monday of the second testing week for that time point. If participants were unable to provide a sample at their designated time, they came to the natatorium when they were able and all efforts were made to get the sample between 2:00 and 4:00pm. Samples were then sent to the University of Wisconsin Institute for Clinical and Translational Research for cortisol analysis using radio-immuno assays.

Questionnaires

A battery of questionnaires was completed online for each testing time using Qualtrics Survey Hosting Service (Qualtrics, LLC) at the University of Wisconsin-Madison. Qualtrics is online survey software that allows the researcher to email the battery of questionnaires to study participants and to receive their results online. The survey software and online delivery procedures abide by the responsible use of information technology policy at the University of Wisconsin-Madison. Participants were given the details of the online survey completion process during the familiarization session. This included a short demonstration of how to open the link to the surveys in an email they receive from study personnel. Participants were instructed not to respond to the email, but to click on the link and follow the instructions as their responses were saved to Qualtrics with their ID number and no other identifier. The battery of questionnaires was completed during the week of their cognitive testing. This online procedure was for the convenience of the participant and was intended to limit their time commitment in the laboratory.

The battery of questionnaires consisted of demographic information including questions about injury and illness that month and instruments designed to reliably and validly assess mood states, perceived stress, perceived cognitive deficits and dimensions of fatigue and subjective reports of training and academic load. The questionnaires included in the battery are the profile

of mood states (POMS), the perceived stress scale (PSS), the perceived deficits questionnaire (PDQ), multidimensional fatigue inventory (MFI), the Self-Motivation Inventory (only at testing time 1), and visual analogue scales (VAS) of perceived training and academic loads.

The POMS measures anger, depression, fatigue, vigor, depression, confusion and tension and can be summed to calculate a global mood disturbance score (McNair, 1989). The POMS has been shown to have acceptable criterion and construct validity in adults and adolescents (Terry et al., 2003). The confirmatory factor analysis for samples of adult and adolescent athletes was greater than 0.90 and factor loading in both samples for each construct in the POMS was significant. The POMS questionnaire has been used extensively in the athlete overtraining literature to monitor mood disturbance in response to increases in training load (Morgan et al., 1987).

The PSS measures the degree to which situations in the last month are appraised as stressful. The PSS is reliable and correlates with life-event scores, depressive and physical symptomology and social anxiety (Cohen et al., 1983). Higher PSS scores have been associated with failure to quit smoking and greater vulnerability to stressful life-event-elicited depressive symptoms (Cohen, 1983). The average female college student value is 25.7 and the male college student average is 21.7 and these values are not significantly correlated with age. Further, coefficient alpha reliability for the PSS was 0.85 in a student sample with a test-retest correlation of 0.85 (Cohen, 1983). A more recent review lists the average PSS score for women as 16.14 and 15.52 for men and 16.78 for adults under the age of 25 years old (Cohen, 2012).

The PDQ provides information concerning attention/concentration, retrospective memory, and prospective memory and planning/organization (Sullivan, 1990). In cognitively unimpaired individuals with multiple sclerosis the convergent validity of the PDQ was 0.74 and

the divergent validity was -0.14. The Cronbach's alpha of the PDQ was 0.93 in this same sample (Marrie et al., 2003).

Finally, participants were asked to complete visual analog scales assessing perceived academic load and training load with 0 indicating 'low' and 100 representing 'high' for the past month (Sansgiry, 2006). Participants were also asked to rate how frequently they experienced mental and physical fatigue on a scale from 0 indicating 'none of the time' to 100 indicating 'all of the time' in the past month. VASs to measure psychosocial constructs have been validated being highly correlated to Likert items measuring the same construct (Hasson, 2005).

Statistical Analysis and Power

Statistical analyses were conducted using SPSS for Windows (20.0 SPSS, Chicago, IL). Data were characterized by computing means and standard deviations for the dependent variables and frequency for categorical variables. Skewness and kurtosis values were examined for all outcomes and outliers were identified based on standard deviations from the mean, distribution of QQ plots, box plots and scatter plots. One student-athlete had a cortisol outlier at test time 2 and one student-athlete had outlier response accuracy during incongruent trials at test 3 and their data were not included in the analyses for those variables. For all aims, a sample size of 42 participants was necessary to yield a power of 0.80 using a conservative small effect size of 0.20 from the influence of exercise on executive function in healthy adults (Smith, 2010, Fedewa, 2011; Hindin, 2012), a moderate correlation of 0.20 among repeated measures and an alpha of 0.05 (G*Power 3.1.7). To control for Type I error with multiple comparisons, Holm's sequential procedure was applied to significant time by group interactions and correlations. For example for a family with 5 outcomes the per comparison the alpha for the most significant finding was 0.05/1 = 0.05, 05/2 = 0.025 for the second most significant interaction, .05/3 =

0.0166 for the third, .05/4 = .0125 for the fourth and .05/5 = .01 for the fifth. If the assumption of sphericity (significant Mauchly's test) for any repeated-measures analysis of variance (ANOVA) was violated for any of the study aims, a data-driven adjustment was made using the Huynh-Feldt epsilon correction.

Aim 1 addresses changes in ERPs and Stroop performance in student-athletes over the course of the competitive season. The performance on the Stroop test was analyzed using response accuracy in percent correct and reaction time in milliseconds for congruent and incongruent trials. Only correct trials were included in the reaction time and ERP average and the first trial in each block was deleted for reaction time, ERP average and response accuracy as there was no warning when the trials were beginning and many participants missed this trial. In order to accomplish this aim, changes over time and across groups (controls and student-athletes) were examined using a group (2- controls and student-athletes) x time (4) repeated measures (RM) ANOVA for ERP amplitude, latency, response accuracy and reaction time during congruent and incongruent Stroop trials. During baseline data collection, we missed six control participant's EEG data due to software problems. If all 4 testing times are included in the RM ANOVA, only 6 control participants have all of their EEG data. Therefore, paired samples *t*-tests were performed to see if differences existed between time 1 and 2 in healthy controls for all ERP outcomes. For amplitudes, only N4 during incongruent trials differed from time 1 (more negative) to time 2 and no significant differences were observed in latencies. For the 6 control participants that had data at the second testing but not baseline, testing time 2 were used as their baseline values. Therefore, for ERP data, 3 (times 1, 3 and 4) x 2 (group) repeated measures ANOVAs were computed but all 4 time points were used for the Stroop performance analyses. Following a significant main effect of group, Tukey's honest significant difference (HSD)

procedures were completed to assess pairwise comparisons. Following a significant interaction in the repeated-measures ANOVA, simple effects of test time in each group were performed. One-way repeated measures ANOVA addressing changes in the student-athlete population in perceived cognitive deficits were performance using Bonferroni post-hoc tests for a conservative assessment of differences across times.

For aim 2, the relationships between additional dependent measures (sleep, stress, mood and cognition) were assessed. Correlations among the variables were determined using Pearson product moment correlation coefficients (r) using a level of significance of $\alpha < 0.05$ as data were normally distributed. Additionally, after exploring descriptive scatter plots of ERP data and stress, mood and sleep variables at peak training, Pearson moment correlations were computed between ERP components and variables that appeared to be related (daytime sleepiness and performance during the Stroop task).

Aim 3 examines group differences between athletes and healthy students. Group differences in frequency were assessed using Chi Square or Kruskal Wallis H tests if there were more than 2 groups and differences in variables with continuous data were determined using independent samples *t*-tests. Differences in outcomes over time were examined using a 2 (group-controls and student-athletes) x 4 (time points) repeated-measures ANOVA for sleep, stress, mood and cognition. Additionally group differences were assessed between genders by including four groups – female controls, male controls, female athletes and male athletes and these differences were addressed using a 4 (time) x 4 (group) repeated-measures ANOVAs for sleep, stress, mood and cognition. For significant interactions, simple effects were computed and following significant group effects, post-hoc Tukey's honest significant difference (HSD) procedures were completed for the comparison across groups and genders. Finally, to assess

differences between controls and student-athletes at peak training, independent-samples *t*-tests were computed (peak training) for sleep, stress, mood and cognition (perceived deficits, Stroop performance and ERPs). Finally, in order to better assess the process of overtraining, athletes were labeled as overreached (OR) if they reported a decrease in their individual performance at time point 3 or 4 with a concomitant increase in total mood disturbance of at least 10 points between times 1 and 3. All other athletes were labeled as well-trained (WT). To describe the group differences between OR and WT athletes in this exploratory examination, effect sizes were calculated using Cohen's *d*.

In order to address aim 4, identifying variables that predict total mood disturbance at peak training, two multiple linear regression analyses were performed using variables collected at baseline (time1) and typical training (time 2) as predictor variables, respectively. Variables were added to the regression model depending on their theoretical relevance for predicting mood and based on previous literature. For both time points the order of the variable list was: Gender, Academic Load, Exercise Load, Cortisol, Epworth Sleepiness and Perceived Cognitive Deficits. Because perceived stress and perceived deficits were highly related at baseline and typical training, we chose to examine the influence PDQ.

Chapter 4. Manuscripts

4.1 Characterizing Cognitive Function in Collegiate Rowers across a Competitive Season

Student athletes train in a dynamic environment that could impact their cognitive health. Positive relationships between exercise and cognition have been consistently reported across the lifespan (Fedewa et al., 2012; Smith, 2010; Colcombe et al., 2003; Hindin, 2010) and a number of hypotheses have been proposed to explain these findings including neuroendocrine activity, elevated arousal, enhanced prefrontal activation and angiogenesis and neurogenesis (Dishman, 2006; Tomporowski & Hatfield, 2005). However, exercise training volume and intensities are considerably greater in athlete compared to non-athlete populations and during peak training, athletes experience elevated negative mood states (Morgan et al., 1987; Raglin, 1991; O'Connor, 1989). These changes in mood could conceivably affect cognition, but there is little research on how cognitive function is affected during periods of repeated high-intensity exercise in collegiate athletes. Examining this relationship is important because cognition is implicit in academic and sport performance and has never been examined in this population across a season. Moreover, a greater understanding of the central nervous system adaptations that occur across a competitive season can help improve student-athlete health considerations.

Interestingly, some aspects of cognition have been shown to be particularly sensitive to acute and chronic exercise and fatigue. The (central) executive functions, which include higher-order cognitive processes inherent to critical thinking such as planning, monitoring conflict, inhibition, set-switching and decision making (Baddeley, 1996) are improved following an acute bout of moderate intensity exercise (resistance training and aerobic) (Chang et al., 2013; Alves et al., 2013). Exercise also results in global increases of electro cortical activity as measured using electroencephalography (EEG) (Crabbe et al., 2004; Moraes, 2011) supporting the basis for

improved behavioral performance. Further, there is evidence to support a positive relationship between fitness and EEG activity when performing executive function tasks. When comparing groups of high and low fit children and adults, fit groups show better performance and neural activity reflective of enhanced perceptual processing and more efficient task monitoring during executive function tasks (Hillman, 2006; Stroth, 200; Themanson, 20089). While the data generally support a cognitive advantage in higher fit individuals, there are no data on how cognition is dynamically affected across a competitive season in response to increases in training load.

During peak training, athletes demonstrate increases in negative mood states including significant increases in fatigue (Morgan et al., 1987; Raglin, 1991; O'Connor, 1989) that could influence executive function. Both acute and chronic fatigue has been shown to negatively impact behavioral and neural aspects of cognitive function (Cook et al., 2007; Michiels & Cluydts, 2001; Faber, 2012). One method that has been utilized to study the impact of fatigue on executive function is to induce mental fatigue using a long and challenging cognitive task. During this laboratory testing, healthy adults report elevations in fatigue and task aversion with time on task, commit more errors, show slowed reaction times and EEG activity reflective of less efficient attentional orienting and diminished response selection (Faber et al., 2012, Kato, 2009; Boksem, 2005). It is unclear if the impact of acute fatigue on cognition experienced in these research settings would be similar to the impact of fatigue on cognitive function that athletes experience during peak training.

The primary objective of this study is to characterize cognitive function over the course of a competitive season in male and female collegiate rowers. Specifically, we aim to characterize cognition by measuring: 1) perceived cognitive deficits, 2) behavioral performance,

and 3) event-related potentials (ERPs) using EEG during the Stroop color-naming task, a test of executive function. It is hypothesized that during peak training, perceived cognitive deficits will increase, behavioral performance will decrease (increased reaction times and more errors) during the more challenging trials and neural activity will show differences compared to baseline.

Methods

This manuscript includes the unique cognitive testing and analyses, which will be emphasized in this methods section. All other methods (participants, testing sessions) are identical to what was presented in chapter 3 of this dissertation.

Cognitive Testing

All participants completed the Stroop color-naming task for their cognitive testing. The Stroop is a sustained attention task that presents the words "red", "green", "yellow" and "blue" in text colors red, green, yellow and blue. The word trials are either congruent (the word "red' appears in the color red) or incongruent (the word "red" appears in a different color). The Stroop is one of the most reliable tests of attention in cognitive science (MacLeod, 1992) and has been used extensively in cognitive and brain imaging studies of executive function and attention (Belanger, 2002; Collette, 2002). The task was completed on a 19-inch Dell computer screen using E-Prime (Psychological Software Tools, Inc). During the consenting day, participants completed a familiarization session, which included instruction and 100 randomized practice trials (50 congruent and 50 incongruent). Participants were instructed to respond to the color of the text and ignore what the word said by pressing the corresponding keyboard button as quickly and accurately with their right hand as possible. Colored paper was taped to the top of keyboard

buttons to be used for their responses. Blue was “v”, yellow was “b”, green was “n” and red was “m”. Participants completed 4 blocks of 25 trials and between each block there was a 10 second rest period. Each color-word was presented on the screen followed by a fixation cross. During the familiarization session, the speed of the color-word presentation got faster across blocks starting with a 2000ms presentation in the first block, a 1500ms presentation during the second block and a 1000ms presentation for the third and fourth block to acclimate them to the speed that was used for testing. For all trials the fixation cross appeared on the screen for 1500ms.

During each subsequent testing session, 60 (30 congruent and 30 incongruent) practice trials and 256 test trials (128 congruent and 128 incongruent) were completed. During practice, participants completed three blocks of 20 trials with 10-second rest periods between blocks. Color-word presentation was 1500ms for the first block and 1000ms for the second and third block of practice. For all test trials, color-words were presented for 1000ms, the fixation cross was presented for 1500ms, trials (congruent and incongruent) were presented randomly and participants finished four blocks of 64 trials with a 10-second rest period between each block. The test trials took approximately 12 minutes to complete. Response accuracy (% correct) and reaction time (ms) for only correctly identified trials was averaged per testing time for congruent and incongruent trials.

EEG

Participant head circumference was measured during the first testing day and they were fitted with the appropriate 256 channel Geodesic EEG cap for measurement of brain electro cortical activity (EGI Geodesic EEG systems, Eugene, OR). EEG data were sampled at 500Hz and amplified at 22.5 K. Before testing, impedance was measured and efforts were made to keep

all electrodes below 50 Ωs. Electrical Geodesic Netstation software was used to analyze all EEG data. Raw data were bandpass filtered for 0.5-30Hz and then segmented to include 200ms before and 1000ms post-event for all correct congruent and incongruent trials. Next, artifact detection was performed including specific algorithms to identify segments that had bad channels (maximum difference of 200μv), eye movements (maximum difference of 140μv) or blinks (maximum difference of 55μv). Further, inferences were performed if more than 20% of the channels in a segment were identified as 'bad' and a segment was 'bad' if it contained more than 10 bad channels, contained an eye blink or eye movement. Next, bad channel data were replaced by interpolating data from the remaining surrounding channels in segments labeled as good. Responses to each trial were baseline corrected using the 200ms before the stimulus. A single average segment from the segments that were identified as 'good' was computed for congruent and incongruent trials per participant for each testing time. While efforts were made to keep impedance low and participants were repeatedly reminded to remain as still as possible during the Stroop task, many participants had multiple segments labeled as 'bad'. The average amount of segments used in the computation of an individual's average for each time was 74/128 for congruent trials and 70/128 for incongruent trials. Grand averages were created using all the athlete data for congruent and incongruent trials to define the timing of ERP epochs.

Previous research has provided the following parameters for expected ERP components. P100 (P1) and N100 (N1) occur within 50-180ms after the stimulus and have peak amplitudes ranging from 0.5 to 2.3μv (P1) and -6μv (N1) during trials involving conflict (Hillyard, 1996; Boenke, 2009). P200 (P2) has an amplitude of 1μv and occurs 150-250ms post-event (Crowley, 2004) while N200 (N2) occurs between 200-380ms with an amplitude of -3.0μv (Nieuwenhuis, 2003; Boenke, 2009). P300 (P3) peak amplitude ranges from 4 to7.5μv and occurs between 250-

400ms after the stimulus (Kok, 1997; Boenke, 2009; Lew; Zurron). N400 (N4) occurs approximately 350-600ms after the event with a peak amplitude of -1.2μv (Kutas & Feremeier, 2011; Silton). P600 (P6) is a positive going wave occurring between 300-700ms after the stimulus with amplitudes of 2 to 6μv (Frische, 2002; Zurron, 2009). Based on previous research and after reviewing the grand averages from the student-athletes for both congruent and incongruent trials, the following parameters were set. After the stimulus onset, P1 was identified as the positive peak between 20-100ms, N1 was the negative peak between 75-165ms, P2 was the positive peak between 115-200ms, N2 was the negative peak between 175-275ms, P3 was the positive peak between 250-400ms, N4 was the negative peak between 350-475ms and P6 was the positive peak between 430-600ms. Finally, adaptive mean amplitude (μv) and latency (ms) for these epochs were identified in congruent and incongruent trials for each participant at each time point.

Statistical Analyses

Statistical analyses were conducted using SPSS for Windows (20.0 SPSS, Chicago, IL). Data were characterized by computing means and standard deviations for the dependent variables. The performance on the Stroop test was analyzed using response accuracy in percent correct and reaction time in milliseconds for congruent and incongruent trials. Only correct trials were included in the reaction time average and the first trial in each block was deleted for reaction time and response accuracy as there was no warning when the trials were beginning and many participants missed this trial.

To test the specific hypotheses that peak training would have the greatest impact of cognition, one-way repeated measures analysis of variance (RM ANOVAs) were performed for

all aspects of the PDQ and following a significant main effect of time, Bonferroni post-hoc tests were performed. Stroop performance (response accuracy and reaction time for congruent and incongruent trials) was examined using a 2 (group) x 4 (time points) and significant interactions were assessed using simple effects of time within each group. To assess changes in ERPs, 2 (group) x 3 (time) repeated-measures ANOVAs were performed. During baseline data collection, we missed six control participant's EEG data due to software problems. Therefore, paired samples *t*-tests were performed to see if differences existed between time 1 and 2 in healthy controls for all ERP outcomes. For amplitudes, only N4 during incongruent trials differed from time 1 (more negative) to time 2 and no significant differences were observed in latencies. Thus, data from testing time 2 were used as their baseline values. ERPs at baseline, peak training and recovery were included in the RM ANOVA. Following any significant interactions, simple effects of time were conducted.

Independent samples *t*-tests were performed for each ERP variable comparing healthy controls and student-athletes at peak training (time 3) to assess the potential differential impact of peak training on cognition. Finally, after exploring descriptive scatter plots of ERP data and stress, mood and sleep variables at peak training, select Pearson moment correlations were computed. To control for Type I error from multiple comparisons, Holm's sequential procedure was applied to the familywise alpha of .05 only for significant interactions. If the assumption of sphericity (significant Mauchly's test) for the within-subjects design was violated, Huynh-Feldt's epsilon correction was applied.

Results

Participant Demographics

A total of 67 participants (23 controls, 22 female athletes and 22 male athletes) completed the informed consent process and familiarization session. One participant opted out of the study before the first testing session. All other missing data are due to scheduling conflicts or illness. Group descriptive data for ethnicity, gender, class, age, number of credits, whether they were recruited to UW to row and whether they ever experienced symptoms of overtraining in their career (decrements in performance, increased negative mood states for more than two-weeks that were not alleviated by rest) are located in Table 1. Additional descriptive data on exams, paper/projects, illness and injury frequency at each testing time are listed in Table 1A Appendix I titled "Cognitive Function".

Table 1. Demographics

	Controls	Student-Athletes	Test Statistic	*p*
N	23	43		
Ethnicity* %			$\chi^2 = 7.96$	0.019
Caucasian	82.6	100		
Asian American	13	0		
African American	4.3	0		
Gender %			$\chi^2 = 1.20$	0.27
Female	65.2	51.2		
Class %			$\chi^2 = 7.09$	0.07
Sophomore	0	23.3		
Junior	63.6	41.9		
Senior	36.4	32.6		
Red-shirt	NA	2.3		
Recruited (% Yes)	NA	30.2		

Overtrained (% Yes)	NA	14		
Age (M ± SD)	20.83 ± 0.83	20.51 ± 1.06	t = 1.24	0.221
Credits* (M ± SD)	14.83 ± 1.88	13.70 ± 1.60	t = 2.57	0.012
BMI (M ± SD)	23.08 ± 1.74	23.84 ± 1.70	t = 1.61	0.11
Godin* (M ± SD)	74.1 ± 29.8	119.61 ± 36.99	t = -5.08	0.00

SA = Student-Athlete, Godin = Godin Leisure Time Physical Activity Questionnaire, BMI = body mass index, M = means, SD = standard deviation
* indicates significant group difference at $p < 0.05$

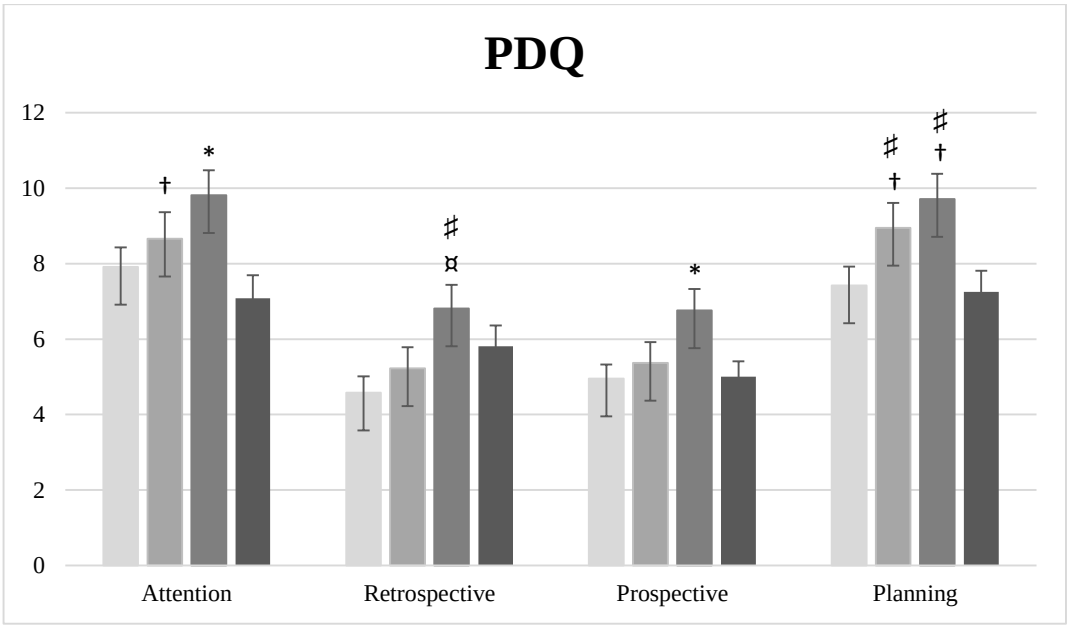
Controls differed from the student-athlete group in their Ethnicity, had more semester credits and scored significantly lower on the Godin Leisure Time Physical Activity Questionnaire. The groups were similar in age, BMI and gender. Independent samples *t*-tests revealed no significant differences ($p > 0.05$) between male and female athletes in any descriptive outcomes so their data were combined and analyzed as one group of student-athletes.

Perceived Cognitive Deficits

Averages for the PDQ at testing times across the competitive season are illustrated in Figures 1 and 2. Group means and standard deviations are presented in Table 2A of Appendix I titled "Cognitive Function". There was a significant main effect of time for attention (F = 14.275, $p = 0.000$). Post-hoc analyses indicated significant differences in attention at peak training (time 3) compared to all other times ($p = 0.001$). Typical training (time 2) also differed from recovery (time 4) ($p = 0.039$). Similar results were observed for retrospective memory (data violated sphericity and the Huynh-Feldt correction was applied, F = 7.97, $p = 0.000$) with peak training being significantly different from baseline ($p = 0.003$) and typical training ($p = 0.001$). Prospective memory differed significantly across time (F = 7.827, $p = 0.000$) with peak training differing from all other times ($p < 0.016$). Planning changed across time (F = 9.337, $p = 0.000$) and while the values during typical and peak training did not significantly differ, they were both

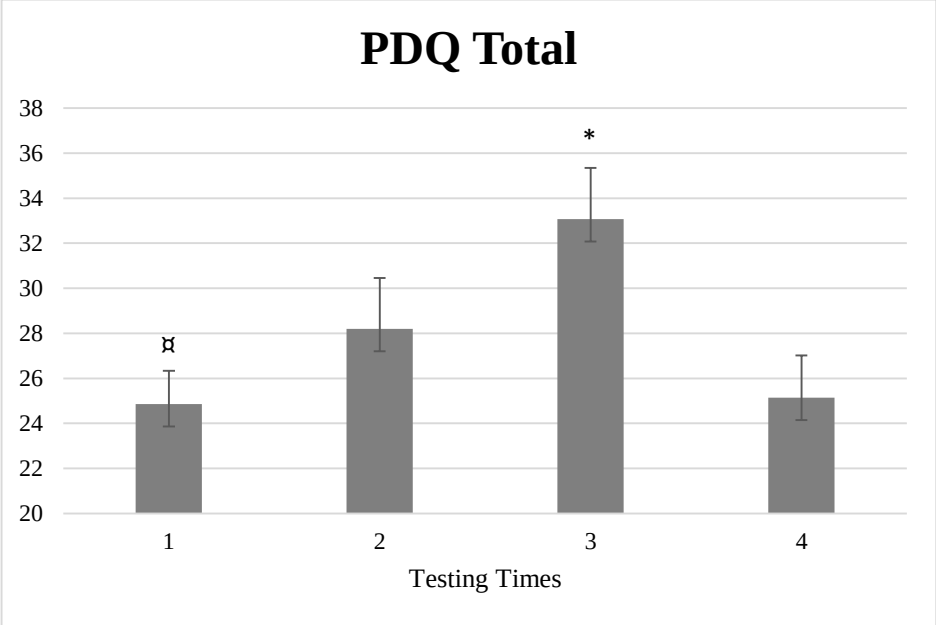
significantly higher than baseline and recovery ($p < 0.011$). Finally, PDQ total changed over time ($F = 13.369$, $p = 0.000$) with post-hoc analyses indicating that peak training was significantly different than all other times ($p < 0.001$).

Figure 1. Student-Athlete means and standard errors on subscales of the Perceived Deficits Questionnaire



*signifies group average differs significantly from all other testing times
†signifies group average differs significantly from time 4
¤ signifies group average differs significantly from time 2
♯ signifies group average differs significantly from time 1

Figure 2. Student-Athlete means and standard errors on Perceived Deficits Questionnaire Total



*signifies group average differs significantly from all other testing times
¤ signifies group average differes significantly from time 2

Stroop Performance

Response Accuracy

Stroop performance outcomes for both controls and student-athletes performance are presented in Table 2. The response accuracy for incongruent trials during peak training for one student-athlete was removed as it was identified as an outlier (> 2 standard deviations from the mean of the group). For response accuracy during congruent trials, there were nonsignificant main effects for time (F = 0.411, p = 0.746), and group (F = 0.025, p = 0.874) and a nonsignificant interaction (F = 0.357, p = 0.784). For response accuracy during incongruent trials, a nonsignificant main effect of time (F = 0.576, p = 0.621, Huynh-Feldt corrected) and a nonsignificant interaction (F = 0.510, p = 0.676, Huynh-Feldt corrected) were observed. A significant main effect for group (F = 5.823, p = 0.020) was observed with controls (92.3%

correct) having significantly higher response accuracy compared to student-athletes (87.7% correct)

Reaction Time

For reaction time to congruent trials, nonsignificant main effects for time (F = 0.878, p = 0.434, Huynh-Feldt corrected) and group (F = 1.497, p = 0.227), and a nonsignificant interaction (F = 2.23, p = 0.102, Huynh-Feldt corrected) were observed. Similarly for reaction time for incongruent trials, nonsignificant main effects of time (F = 0.523, p = 0.636, Huynh-Feldt corrected), group (F = 1.59, p = 0.213) and the interaction (F = 1.62, p = 0.195, Huynh-Feldt corrected) were identified. No group differences in any reaction times or response accuracies were observed during peak training ($p > 0.05$).

Table 2. Means (M) and standard deviations (SD) for group performance during the Stroop color-naming task

	Test	**Controls**		**Student-Athletes**	
		n	**M ± SD**	**n**	**M ± SD**
Response Accuracy Congruent (%)	1	23	96.03 ± 3.17	41	95.81 ± 3.54
	2	22	95.87 ± 2.86	42	96.20 ± 2.94
	3	23	96.05 ± 5.92	41	95.80 ± 3.63
	4	17	96.12 ± 3.47	34	96.03 ± 5.33
*Response Accuracy Incongruent (%)	1	23	91.79 ± 4.36	41	87.56 ± 8.56
	2	22	92.44 ± 4.05	42	88.24 ± 7.82
	3	23	90.57 ± 7.53	41	87.62 ± 8.22
	4	17	92.68 ± 4.36	34	87.94 ± 7.37
Reaction Time Congruent (ms)	1	23	584.54 ± 46.83	41	608.06 ± 65.34

	2	22	583.77 ± 50.31	42	607.47 ± 64.01
	3	23	583.75 ± 58.14	41	605.37 ± 68.73
	4	17	581.47 ± 67.94	34	620.73 ± 87.22
Reaction Time Incongruent (ms)	1	23	667.15 ± 65.12	41	694.89 ± 90.76
	2	22	663.31 ± 60.04	42	692.77 ± 88.67
	3	23	660.36 ± 71.16	41	688.33 ± 95.66
	4	17	643.49 ± 81.64	34	692.73 ± 101.56

*indicates group average differs significantly

ERPs

ERP group means and standard deviations for amplitude during congruent trials and incongruent trials are listed in Table 3A and Table 4A of Appendix I, respectively. ERP amplitudes and latencies for controls and student-athletes during congruent and incongruent trials are illustrated in Appendix I figures 1A-13A. Results of the RM ANOVAS for ERP amplitudes are listed in Table 3.

Table 3. Results of RM ANOVAS for ERP Amplitude for congruent and incongruent trials

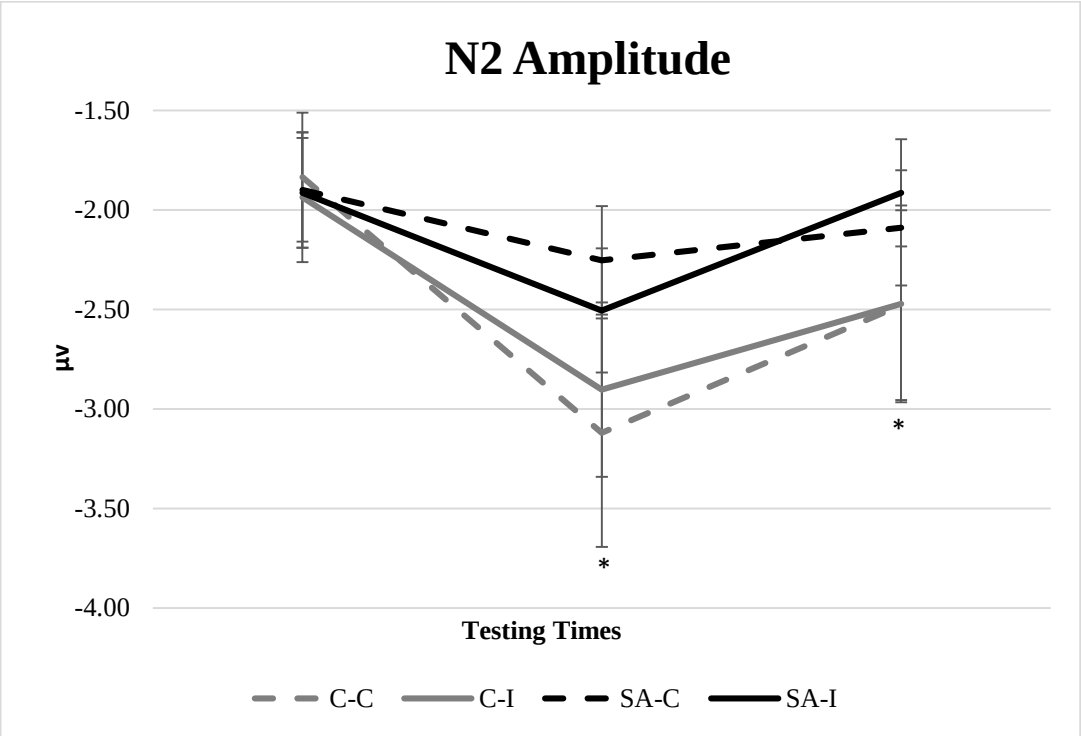
	Time		**Group**		**Interaction**	
Congruent	**F**	***p***	**F**	***p***	**F**	***p***
N1	7.778	0.001	0.458	0.503	0.387	0.681
N2	17.63	0.000	1.997	0.165	7.098	0.001
N4	1.985	0.144	2.391	0.13	2.802	0.067
P1	5.984	0.004	1.039	0.314	0.069	0.933
P2	4.833	0.011	1.359	0.251	0.039	0.962
P3*	1.23	0.29	7.243	0.01	1.507	0.229

P6*	6.203	0.004	0.199	0.658	3.314	0.046
Incongruent						
N1	10.643	0.000	0.396	0.533	0.802	0.452
N2*	17.746	0.000	1.784	0.189	7.95	0.001
N4	1.24	0.294	1.271	0.266	2.37	0.100
P1	6.556	0.002	0.642	0.428	0.193	0.825
P2*	12.979	0.000	0.536	0.469	1.246	0.291
P3	1.71	0.187	3.177	0.082	0.891	0.414
P6	4.367	0.016	1.451	0.235	1.337	0.268

* Signifies that data violated the assumptions of sphericity and the Huynh-Feldt correction was applied

Significant interactions were observed for N2 amplitude in both congruent and incongruent trials. Group means and standard error are presented in Figure 3 for N2 amplitude during congruent and incongruent trials. Examining the simple effect of time in each group revealed that for controls, N2 during congruent trials at baseline was significantly smaller (less negative) than during peak training and recovery ($p = 0.000$). For student-athletes, no significant differences across testing times were observed ($p > 0.05$). During incongruent trials, N2 amplitude in controls was significantly less negative at baseline compared to peak training and recovery ($p = 0.000$). In the student-athlete group, no significant differences across testing times were observed. No group differences at peak training were observed for any ERP amplitude ($p > 0.05$). The interaction of P6 amplitude during congruent trials did not survive the Holm's sequential procedure to control for multiple comparisons ($0.046 > 0.0166$).

Figure 3. N2 Amplitude (μv) at testing times baseline, peak training and recovery



C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials
*indicates significant difference from time 1 in controls for both congruent and incongruent trials

Results of the ERP latency RM ANOVAS are listed in Table 4. Group means and standard deviations for ERP latency during congruent and incongruent trials are listed in Tables 5A and 6A of Appendix I, respectively. The interaction of P2 latency during incongruent trials did not survive the Holm's sequential procedure (0.050 > 0.0125). Therefore, Simple Effects analyses were not performed.

Table 4. Results of RM ANOVAS for ERP latency for congruent and incongruent trials

	Time		**Group**		**Interaction**	
Congruent	**F**	***p***	**F**	***p***	**F**	***p***
N1*	0.367	0.659	0.636	0.430	0.122	0.853
N2	1.80	0.172	0.144	0.706	0.052	0.949
N4	12.67	0.000	0.186	0.669	1.36	0.263
P1	0.529	0.591	0.055	0.816	0.91	0.407
P2*	2.315	0.111	1.255	0.269	2.06	0.139
P3*	0.217	0.79	0.028	0.867	0.776	0.456
P6	0.107	0.898	0.98	0.328	1.02	0.365
Incongruent						
N1	0.818	0.445	0.018	0.893	0.409	0.666
N2*	1.39	0.255	0.003	0.956	1.483	0.234
N4	6.26	0.003	0.237	0.629	0.591	0.556
P1	1.49	0.231	0.249	0.621	0.734	0.483
P2	2.513	0.087	1.218	0.276	3.12	0.050
P3	0.399	0.672	0.315	0.578	0.471	0.626
P6	2.018	0.14	0.275	0.615	0.301	0.741

* Signifies that data violated the assumptions of sphericity and the Huynh-Feldt correction was applied

Relationships between ERP components during congruent and incongruent trials and select data during peak training were explored (see adaptations paper). Pearson's r values are reported in Table 5 for ERP amplitude and latency during congruent trials, Table 6 for ERP amplitude and latency during incongruent trials. ESS was significantly and positively related to N2 amplitude during congruent trials ($p = 0.035$) and P6 amplitude during incongruent trials ($p =$

0.029). Reaction time during congruent trials was positively related to N2 latency (p = 0.011). Finally, response accuracy during incongruent trials was negatively related to N4 amplitude (p = 0.018) and P6 amplitude (p = 0.041).

Table 5. Correlations at peak training for ERP amplitudes and latencies during Congruent Trials

	N1A	N2A	N4A	P1A	P2A	P3A	P6A	N1L	N2L	N4L	P1L	P2L	P3L	P6L
ESS	0.12	.34*	-.19	.04	.08	-.29	-.30	-.20	.02	.21	-.29	.13	-.29	-.15
RA	-0.01	-.03	-.10	.11	.03	.26	.11	.15	-.01	-.16	.22	.16	.11	.13
RT	0.021	.010	.07	-.02	-.15	.08	.01	-.09	.40*	-.03	.07	.05	.27	-.03

ESS = Epworth Sleepiness Scale, RT = Reaction time during Stroop Trials, RA=Response Accuracy during Stroop Trials, A = Amplitude, L = Latency

Table 6. Correlations at peak training for ERP Amplitudes during Incongruent Trials

	N1A	N2A	N4A	P1A	P2A	P3A	P6A	N1L	N2L	N4L	P1L	P2L	P3L	P6L
ESS	.07	.24	-.08	.06	.19	-.20	-.35*	-.13	-.04	.16	.02	.13	-.21	-.02
RA	.06	.19	-.38*	.07	.25	.15	-.33*	-.21	.11	.25	-.05	.04	-.20	-.15
RT	-.21	-.17	.05	-.27	-.03	.08	.08	-.23	.16	.05	-.03	.13	.19	.26

ESS = Epworth Sleepiness Scale, RT = Reaction time during Stroop Trials, RA = Response Accuracy during Stroop Trials, A = Amplitude, L = Latency

Discussion

The cumulative impact of repeated high-intensity exercise and elevated levels of fatigue on cognitive function during a competitive season in student-athletes has not previously been examined. In the current study, aspects of cognition were found to change across a season in collegiate rowers and physically active healthy controls. For student-athletes, perceptions of cognitive deficits total score were highest at peak training. Additionally, compared to baseline, all the subscales of cognition assessed by the PDQ (attention, retrospective and prospective

memory and planning) increased suggesting effects on multiple aspects of perceived cognition. On the other hand, performance on the Stroop color-naming task did not change across the season. This could indicate the student-athlete's ability to maintain behavioral performance in spite of the observed changes in their perception and brain electro cortical activity. Fortunately a learning effect was not observed and this allowed for an examination of the impact of training volume on executive function rather than the impact on a practice effect or of the diminished novelty of the Stroop task. A small body of literature has proposed that psychomotor speed (speed of information processing) is impacted in athletes showing signs of negative adaptation (Nederhoff, 2008; Reitjens, 2005; Dupuy, 2010). Our data do not provide information that is supportive of this hypothesis.

To our knowledge, there are no data detailing the way that brain function adapts to repeated high-intensity exercise. Thus, it is plausible that even though Stroop performance was maintained, the neural activity driving this behavior was affected. Several aspects of objective brain activity during the Stroop changed across the season and in some cases differed significantly from controls. Examining event-related potentials during an executive function task allows for a precise temporal assessment of information processing, which is an ideal outcome for examining nuanced changes in cognition that might be sensitive to demands occurring across an athletic season. While no group differences were observed in any ERP amplitude or latency at peak training, data from the current study, suggest that student-athletes had a different pattern of habituation in earlier stimuli-driven properties of conflict monitoring compared to controls.

The earliest ERP outcomes, P100 (P1) and N100 (N1), are generated from the temporal and occipital regions (Hillyard, 1996; Crowley, 2004) and represent automatic attention and perceptual resources necessary to achieve a sensory gain for the stimulus (auditory, visual etc.)

(Kok, 1997; Ibanez, 2012). Both components showed changes in amplitude over the course of the season/semester, but no differences between groups and no changes in latency were observed. P1 demonstrated smaller peaks over time while N1 amplitude became larger (more negative). These response patterns may be an indication of improved orienting to relevant stimuli or habituation - rather than a measurement of impairment. In previous research, no differences in P1 amplitude or latency were reported between congruent and incongruent trials, between fitness levels or in response to fatigue (Boenke, 2009; Faber, 2012; Boksem, 2005). The N1 peak has been shown to be larger (more negative) during incongruent Stroop trials (Boenke, 2009), smaller (less negative) with time on task during acute fatigue (Faber, 2012; Boskem, 2005) and not impacted by fitness levels (Hillman, 2006; Scicso, 2008). Thus, the gradual changes in P1 and N1 amplitudes across the season in both athletes and non-athletes likely reflect a general pattern of habituation in attentional orientation and sensory priming.

The P200 amplitude in both congruent and incongruent trials changed significantly over the course of the season by getting smaller over time. In general, P2 is involved with withdrawing attention from irrelevant stimuli (Crowley, 2004) and is not impacted by fitness or during acute fatigue (Hillman, 2006; Faber, 2010). Although our study did demonstrate a change over time, based on the cognitive performance data, these changes were similar across controls and student-athletes. N200 (N2) is related to perceptual feature detection, conflict monitoring, inhibition, novelty and response preparation (Nieuwenhaus, 2003, Ibanez, 2012). In the current study, N2 amplitude changed over time and across groups with controls showing a significantly larger (more negative) peak at testing time 3 and 4 compared to baseline during both congruent and incongruent trials with no changes latency. In contrast, N2 amplitude and latency did not change over the course of the season in student-athletes.

Increases in N2 amplitude have been reported for challenging tasks involving conflict monitoring and found to be generated by the anterior cingulate cortex, a region responsible for the integration of physiological and internal information (Vogt, 2005; Silton, 2010, Nieuwenhaus, 2003). N2 amplitude is modulated by the frequency of conflict (larger for less frequent trial type) and has been interpreted as a reflection of conflict monitoring opposed to inhibition processes (Nieuwenhaus, 2003). Thus, larger amplitudes could indicate more resources dedicated to monitoring the task for conflict and may then augment response processing. Further, research has reported smaller N2 amplitude in fit adolescents during the Eriksen flanker task and this was interpreted as more efficient information processing (Stroth, 2009). However this finding was not replicated in healthy young adults (Themanson, 2006).

In acute fatigue research, one study reported no changes in N2 amplitude during an executive function task (Faber, 2012) while another one reported significantly longer latencies for both easier (Go trials) and more challenging (noGo trials) cognitive task trials with time on task (Kato, 2010). Boksem et al., 2005, found increased N2 amplitude with time on task for trials involving relevant and irrelevant information suggestive of diminished discrimination between types of information and increased distractibility. The observed increase in N2 amplitude only for healthy controls suggests that this pattern may reflect a general habituation response to the Stroop task that is not occurring in athletes. Conversely, as increases in N2 peak are consistent with increased task demand (Silton, 2010) and conflict monitoring (Nieuwenhaus, 2003), athletes may be showing a cognitive advantage for more efficient monitoring during this stage or diminished N2 amplitude may reflect impairment from decreased resource allocation during this stage of conflict processing. N2 amplitude during congruent trials was positively related to daytime sleepiness at peak training suggesting that the more negative amplitude, the less

sleepiness. Additionally, controls were significantly more accurate during incongruent trials supporting a benefit of larger N2 amplitude for successful conflict resolution. Without significant changes in cognitive performance a more direct interpretation of the data is not possible at this time.

Early ERP components (P1, N1, P2, N2) are categorized as indexes of orienting attention toward relevant information and basic sensory perception. Amplitudes in these components were found to change across a competitive season, which might indicate more efficient (fewer resources needed to process information) processing even though improved performance was not observed on the Stroop task. Group differences and relationships with perceived sleepiness and cognitive performance for N2 at peak training, suggests that the information processing occurring during this period of time (175ms-275ms after the stimulus) is potentially sensitive to exercise training and could represent either a lack of habituation or of more efficient conflict monitoring.

Later components of cognitive processing also changed over the course of the season/semester. P300 (P3) is a highly studied positive peak associated with increases in attention allocation and task demands that require engaging higher-order cognitive processing, such as executive functions (Ibanez, 2012). In executive function research, as the task demand increases, P3 amplitude decreases suggesting possible information loss, greater resource allocation or inhibition of extraneous activity to focus on the relevant information (Kok, 1997; Polich, 2007). The present results demonstrate that P3 amplitude during both congruent and incongruent trials changes over time with no significant interaction. P3 amplitude has inconsistently been reported as influenced by fitness with some reports of larger amplitude in high fit children during an executive function task (Hillman, 2006) but others not supporting any

difference (Stroth, 2009; Scisco, 2008). Further, during fatiguing cognitive tasks, one study reported P3 amplitude decreases (Kato, 2009) and another reported no change (Faber, 2012). This time interval represents a general network for detecting, resolving and responding to conflict that involves responses from different generators (sources) across regions (Zurron, 2009; Polich, 2007).

N400 (N4) is related to complicated aspects of processing that integrate basic and higher-order cognition related to word expectancy, assessing meaningful similarities across modalities as well as semantic memory (Kutas & Ferermeier, 2011). Although no changes in N4 amplitude were found across the study, latencies increased across the season in both controls and athletes, suggesting a slowing of information processing in both groups. Further, N4 amplitude during incongruent trials was negatively related to response accuracy supporting a role in successful resolution and responses to conflict, which is consistent with previous research (Hanslmayr, 2008; Silton, 2009). Because N4 latency was not related to any Stroop performance outcome, it is unclear if a delay in N4 peak is indicative of improvement (able to spend more time monitoring before responding) or impairment (slowed conflict resolution) across the season.

A final later component assessed in executive function tasks is the P600 (P6). In a recent review on the P6, Brouwer et al., 2012, concluded that this wave is better conceptualized as an assessment of whether the presented information matches that of the information retrieved from short or long-term memory for the specific task (N4) (retrieval integration). Thus, the P6 amplitude increases with difficulty when the two sets of information do not match (Hanslmayr, 2008). In the present study, P6 amplitude changed over time in a similar manner in controls and student-athletes. Additionally, P6 amplitude during incongruent trials was significantly and negatively related to both daytime sleepiness and response accuracy at peak training. Thus

smaller amplitudes were associated with increases in sleepiness and less accurate performance on the Stroop task in student-athletes.

The findings of this study are limited in a number of ways. First, the quality of EEG data was low for a number of participants limiting the accuracy of the average amplitudes and latencies. Because the criteria to define useable segments were stringent, the averages for some participants were computed from less than half of the actual events. Further, as few ERP components and Stroop performance were related, the Stroop task may not have been sensitive enough to elicit training-induced changes in behavior that relate to brain activity changes. Future research should focus on region-specific changes in ERP components and relationships with other variables across a training season. Further, the normal process of habituation to the Stroop stimuli should be detailed so that deviations from this process can be quantified.

We observed significant increases in perceptions in cognitive deficits (attention, executive function and memory) during peak training without a concomitant change in performance on an executive function task. Additionally, interesting interactions were identified in N2 amplitude for both congruent and incongruent trials between controls and athletes with no group differences or interactions in ERP in latencies. These results suggest that the demands of student-athletes across a semester may be interacting to impact early stages of object and conflict recognition (N2) and response selection but not the timing of processing in the student-athlete population. Future research is needed to identify how these changes impact injury susceptibility and whether this stage of processing affects academic or athletic performance.

4.2 Adaptation Across a Competitive Season in Collegiate Rowers

Over 450,000 athletes participate in the National Collegiate Athletic Association representing an understudied population that thrives in the face of academic and athletic challenges (www.ncaa.org/student-athletes). Although student-athletes demonstrate overall positive mental health (Donohue, 2004), little is known about the psychological and biological adaptations, including changes in sleep, mood, stress, and cognitive function, that occur over the course of a competitive season. Due to the conflicting positive relationships between exercise and cognition and negative relationships between fatigue and cognition, it is plausible that the repeated high-intensity exercise training occurring across an athletic season could have negative, albeit temporary, effects on the brain health of student-athletes. Whether changes across the competitive season are best characterized by positive adaptation and resiliency, subtle or overt impairment or no change; the impact of repeated high-intensity exercise on the student-athlete's well-being is important to examine. If healthy adaptations are observed, variables related to psychological health can be acknowledged. Alternatively, if any sign of impairment is observed over time, associated variables can be identified and ultimately strategies to ameliorate them can be devised.

College athletes are a unique population and well-suited to examine the complex relationship between exercise and brain health. Previous research has been conducted to understand the influence of athletic and academic demands on the mental health of student-athletes. Collegiate athletes have similar or lower rates of depression and anxiety than non-athlete college students (Yang et al., 2007). Further, student-athletes exhibit positive mental health profiles at the beginning and end of the athletic season - characterized by low anger, depression, tension, confusion and fatigue and high levels of vigor (Morgan et al., 1987).

Interestingly, research has demonstrated that training volume and negative mood states increase in a dose-response relationship throughout a season. Thus, during peak training, the mental health profile of the athlete is negatively impacted – characterized by elevated levels of anger, depression, confusion and fatigue and decreased levels of vigor. This pattern of disturbed mood has been consistently shown across the season in endurance athletes though most athletes recover by the end of the season (Morgan et al., 1987, Raglin et al., 1991; O'Connor et al., 1989). Although changes in mood states across a competitive season have been rigorously characterized in college athletes, patterns of adaptation and relationships amongst other markers of central nervous system function such as sleep and cognition have not been as thoroughly examined.

In some athletes, mood disturbances that are observed during peak training become more severe and are accompanied by decreases in performance and additional physiological perturbations. This process has generally been referred to as athlete overtraining, overreaching or the overtraining syndrome and affects approximately 20-60% of endurance athletes at some point during their career (Lehmann et al., 1993; Meeusen et al., 2013). The overtraining process has been conceptualized as the result of an imbalance between exercise demands and recovery occurring across a continuum of time and symptom severity (Kellmann, 2010). In order to identify this dysregulation, many peripheral biomarkers, including hormones, have been examined following acute increases in training volume with few consistent results. The most common signs include decreases in objective performance, disturbed mood and elevated feelings of fatigue (Meeusen et al., 2013; Purvis, Gonsalves, & Deuster, 2010).

When considering the negative impact that elevated levels of fatigue have on challenging cognitive tasks in healthy adults (Barwick, 2012, Boksem, 2005), it has been proposed that the elevated fatigue and negative mood states that athletes experience at peak training would also

impact cognitive function. The psychomotor speed hypothesis suggests that slower reaction times during challenging cognitive tasks may be an early indicator of overtraining (Reitjens et al., 2005; Nederhoff et al., 2007; Hynynen et al., 2008; Dupuy et al., 2010). Unfortunately, there are no data characterizing normal changes in cognition across a season making this hypothesis difficult to assess.

The primary objective of this study was to characterize psychological and biological adaptations over the course of a competitive season in Division I collegiate rowers. An additional objective was to identify variables that are related at peak training and those occurring earlier in the season that predict mood disturbance during peak training. It was hypothesized that student-athletes would demonstrate positive mental health profiles at the beginning and the end of the competitive season. However, during peak training, student-athletes would show significant increases in negative mood states, sleepiness, cognitive impairment and measures of stress. Examining changes in these variables over the course of an athletic season was intended to provide insight to the dose-response relationship between exercise and central nervous system function with a specific focus on stress and cognition in student-athletes.

Methods

This manuscript focuses on the overall adaptation in student-athletes across a semester. For complete methods, the reader is referred to chapter 3 of this dissertation.

Statistical Analyses

Statistical analyses were conducted using SPSS for Windows (20.0 SPSS, Chicago, IL). Data were characterized by computing means and standard deviations for the dependent variables. Within and between group differences in sleep, mood, stress and cognitive function

were analyzed with repeated measures analysis of variance (ANOVAs) applying a familywise alpha of 0.05. Following significant interactions, simple effects of time were calculated for controls and student-athletes. At testing time 3, independent samples *t*-tests were performed to identify differences between controls and student-athletes. Independent samples *t*-tests were also performed to assess differences in variables between male and female athletes. Pearson correlations were used to assess relationships for the student-athlete group at peak training (test time 3).

In order to identify variables that predict total mood disturbance at peak training, two multiple linear regression analyses were performed using variables collected at baseline (time1) and typical training (time 2) as predictor variables, respectively. Variables were added to the regression model depending on their theoretical relevance for predicting mood and based on previous literature. For both time points the order of the variable list was: Gender, Academic Load, Exercise Load, Cortisol, Epworth Sleepiness and Perceived Cognitive Deficits. Because perceived stress and perceived deficits were highly related, we chose to examine the PDQ.

In order to better assess the process of overtraining, athletes were labeled as overreached (OR) if they reported a decrease in their individual performance at time point 3 or 4 with a concomitant increase in total mood disturbance of at least 10 points between times 1 and 3. All other athletes were labeled as well-trained (WT). To describe the group differences between OR and WT athletes in this exploratory examination, effect sizes were calculated using Cohen's *d*). For all measures, the level of significance was $p < 0.05$.

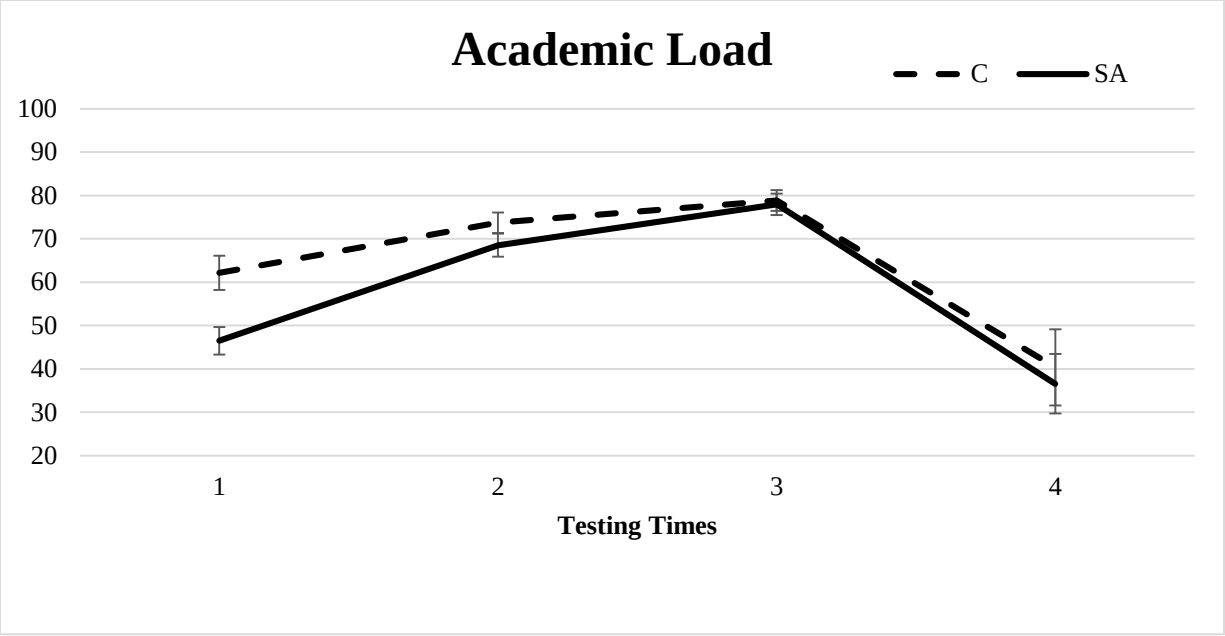
Results

Exercise and Academic Loads

Perceived academic and athletic load values are illustrated in Figures 1 and 2 and listed in Table 1A of Appendix II. For academic load, a significant main effect for time ($F = 30.78$, $p = 0.000$ Huynh-Feldt corrected), a nonsignificant main effect for group ($F = 1.52$, $p = 0.23$) and a nonsignificant interaction ($F = 1.12$, $p = 0.34$, Huynh-Feldt corrected) were observed. During peak training (time 3), perceived academic load did not differ between controls and student-athletes ($t = 0.25$, $p = 0.81$). No differences between male and female athletes in academic load values ($p > 0.05$) were detected at any time point.

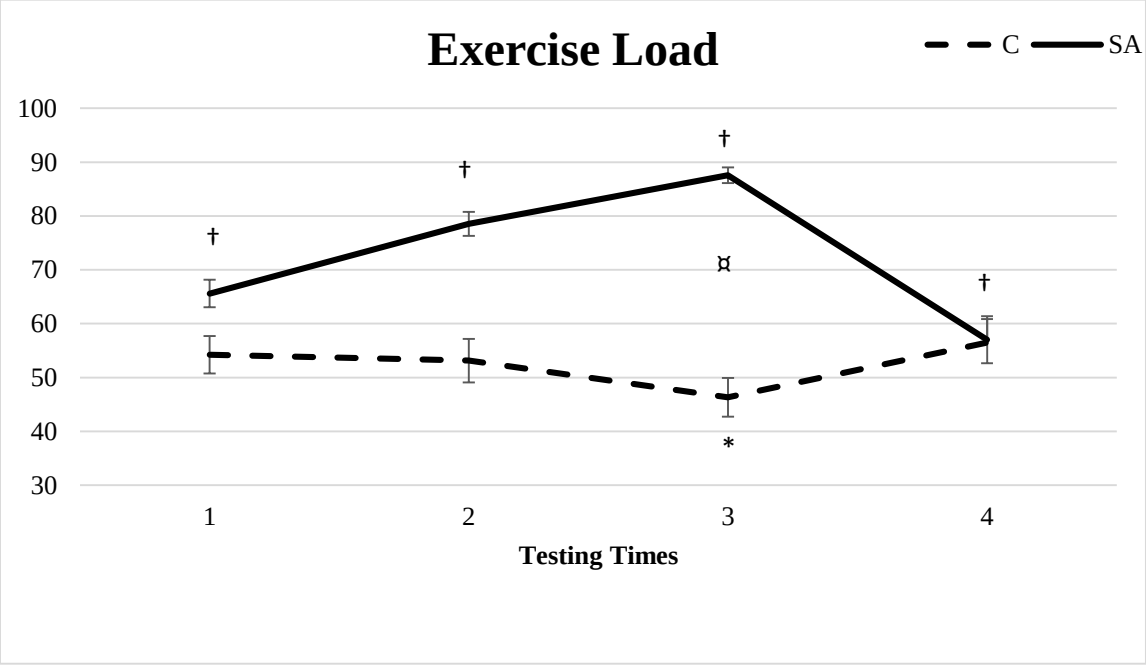
For exercise load, a significant main effect for time ($F = 6.33$, $p = 0.002$, Huynh-Feldt corrected) significant main effect for group ($F = 28.42$, $p = 0.00$) and a significant interaction ($F = 16.45$, $p = 0.00$, Huynh-Feldt corrected) were observed. Simple effects of time revealed that for student-athletes, perceived exercise load at each time was significantly different from all other testing times ($p < 0.05$). In controls, values during typical training (time 2) were significantly higher ($p = 0.018$) than during peak training. At peak training, perceived exercise load differed significantly between controls and student-athletes ($t = -12.53$, $p = 0.000$). No differences between male and female athletes in academic load values ($p > 0.05$) were detected at any time point.

Figure 1. Means and standard errors for Visual Analog Scale for academic load



C = Controls, SA = Student-Athlete group

Figure 2. Means and standard errors for Visual Analog Scale for exercise load

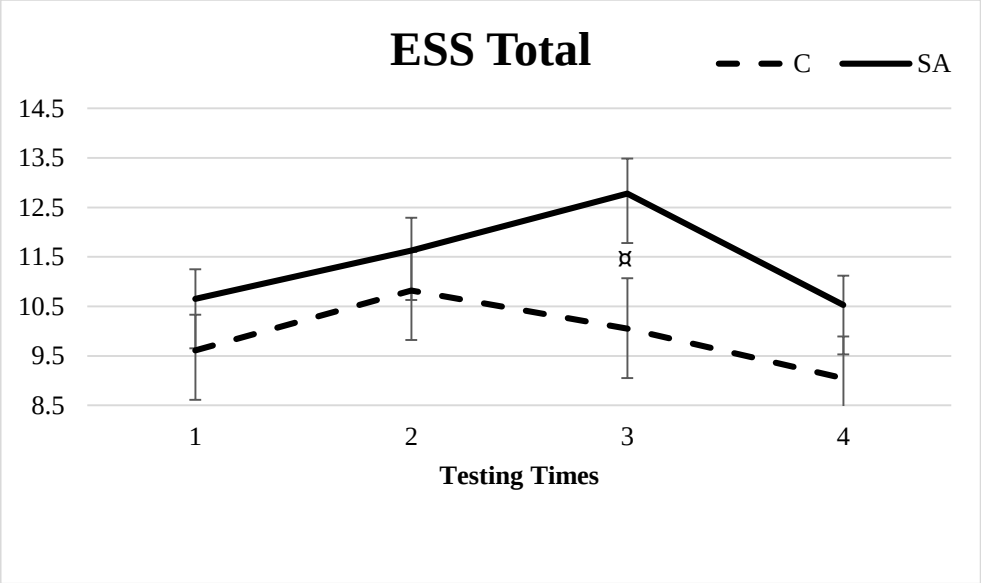


C = Controls, SA = Student-Athlete group
* Indicates significant difference from time point 2 in the control group
† Indicates significant difference from each time point in the student-athlete group
¤ Indicates significant difference between controls and student-athletes

Sleep

For daytime sleepiness, group means and standard deviations are listed in Table 2A of Appendix II and Figure 3. There was a significant main effect of time (F = 6.19, p = 0.001), a nonsignificant main effect for group (F = 1.88, p = 0.177) and a nonsignificant group by time interaction (F = 1.07, p = 0.365). Specifically, during peak training, student-athletes reported significantly greater sleepiness compared to the control group (t = -2.240, p = 0.029). No differences between male and female athletes in ESS scores ($p > 0.05$) were detected at any time point.

Figure 3. Means and standard errors for Epworth Sleeipness Scale



C = Controls, SA = Student-Athlete group
¤ Indicates significant difference between controls and student-athletes

Mood

Total mood disturbance (TMD) scores and effect sizes from the POMS are presented in Table 1 and illustrated in Figure 4. The statistical analyses for TMD revealed a significant main effect of time (F = 13.17, p = 0.000), a significant main effect for group (F = 6.61, p = 0.013) and a significant time by group interaction (F = 4.32, p = 0.006). Simple effects for the SA group

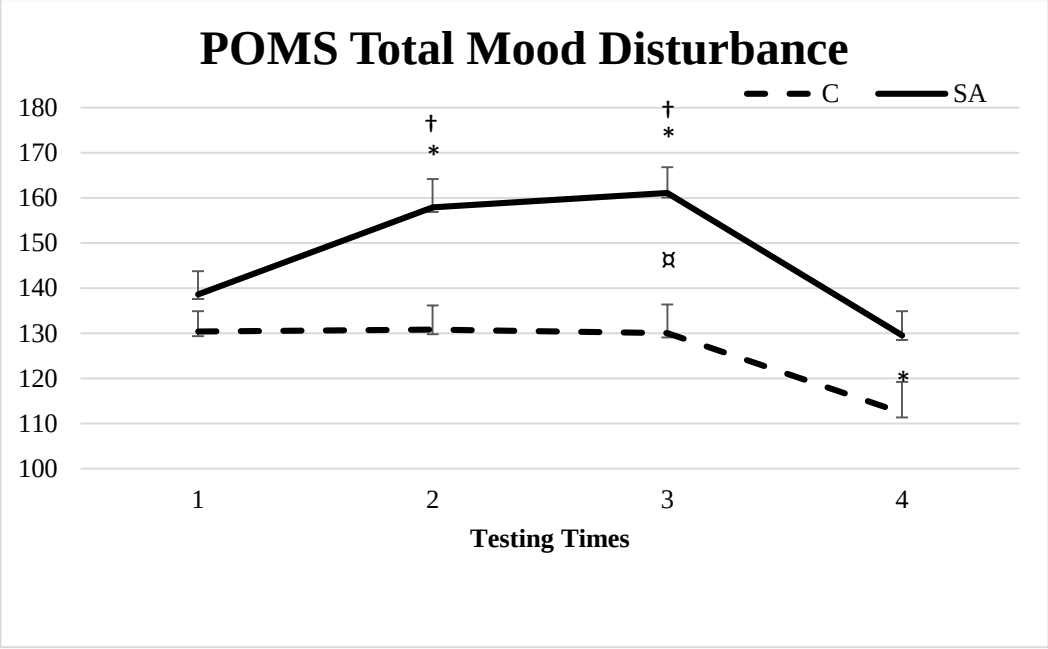
demonstrated that baseline (time 1) differed significantly ($p < 0.05$) from both typical and peak training time points. TMD during typical and peak training were similar and they both significantly differed from recovery (time 4). In controls, the only difference in TMD was between baseline and recovery. At peak training, student-athletes had a significantly greater total mood disturbance compared to controls ($t = -3.399$, $p = 0.001$). No differences between male and female athletes in TMD scores ($p > 0.05$) were detected at any time point.

Table 1. Profile of Mood States Total Mood Disturbance

			Controls		**Student-Athletes**	**Effect Size**
TMD	**Test**	**n**	M ± SD	**n**	M ± SD	Cohen's *d*
	1	23	130.35 ± 21.62	43	138.58 ± 33.85	0.205
	2	22	130.77 ± 25.26	41	157.90 ± 40.05	0.573
	3*	22	130.05 ± 29.64	41	161.07 ± 36.85	0.656
	4	19	112.32 ± 30.01	36	129.50 ± 32.40	0.390

M = mean, SD = standard deviation
*Indicates significant difference between groups at time 3

Figure 4. Group means and standard errors for the Profile of Mood States Total Mood Disturbance



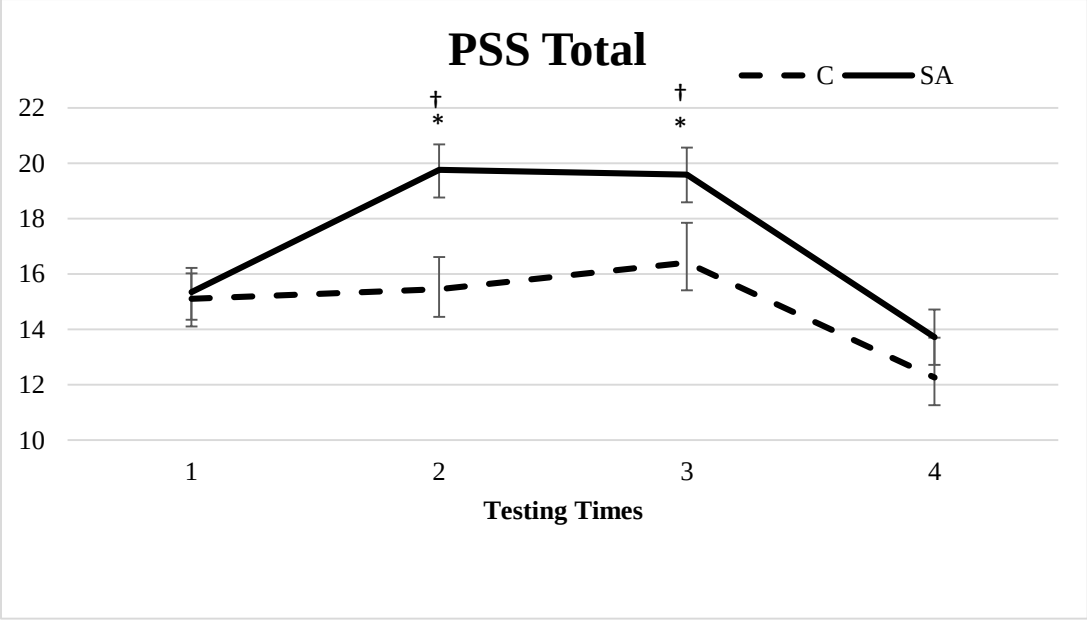
C = Controls, SA = Student-Athletes
* Indicates significant difference compared to time point 1 within each group

† Indicates significant difference compared to time 4 within each group
¤ Indicates significant difference between controls and student-athletes

Stress

The PSS total score is listed in Table 3A of Appendix II and illustrated in Figure 5. There was a significant main effect of time (F = 15.52, p = 0.000), a nonsignificant main effect for group (F = 3.029, p = 0.088) and a significant time by group interaction (F = 3.185, p = 0.026). Simple effects within the student-athlete group demonstrated significant differences ($p < 0.05$) in perceived stress during typical and peak training compared to both baseline and recovery. Baseline did not significantly differ from recovery and typical training did not significantly differ from peak training. In controls, there were no significant differences across time points. During peak training, no significant difference was observed between student-athletes and controls (t = -1.88, p = 0.065). No significant differences between male and female athletes in PSS scores ($p > 0.05$) were detected at any time point.

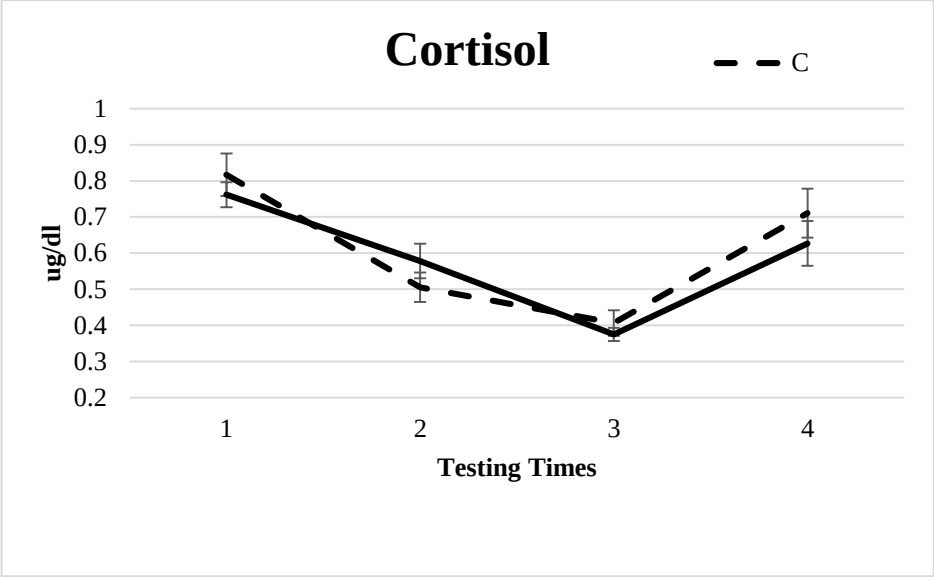
Figure 5. Group means and standard errors on the Perceived Stress Scale



C = Controls, SA = Student-Athletes
* Indicates significant difference compared to time point 1 within each group
† Indicates significant difference compared to time 4 within each group

Cortisol values are listed in Table 4A of Appendix II and illustrated in Figure 6. One athlete cortisol value from time 2 was identified as an outlier (more than two standard deviations about the mean) and removed from the analysis. For resting cortisol, there was a significant main effect of time (F = 18.86, p = 0.000, Huyn-Feldt corrected), a nonsignificant main effect for group (F = 0.112, p = 0.74) and a nonsignificant time by group interaction (F = 0.89, p = 0.44, Huyn-Feldt corrected). During peak training, no significant group difference was observed (t = 0.476, p = 0.642). No significant differences between male and female athletes in cortisol values ($p > 0.05$) were detected at any time point.

Figure 6. Group means and standard errors in resting cortisol (µg/dl)



C = Controls, SA = Student-Athletes

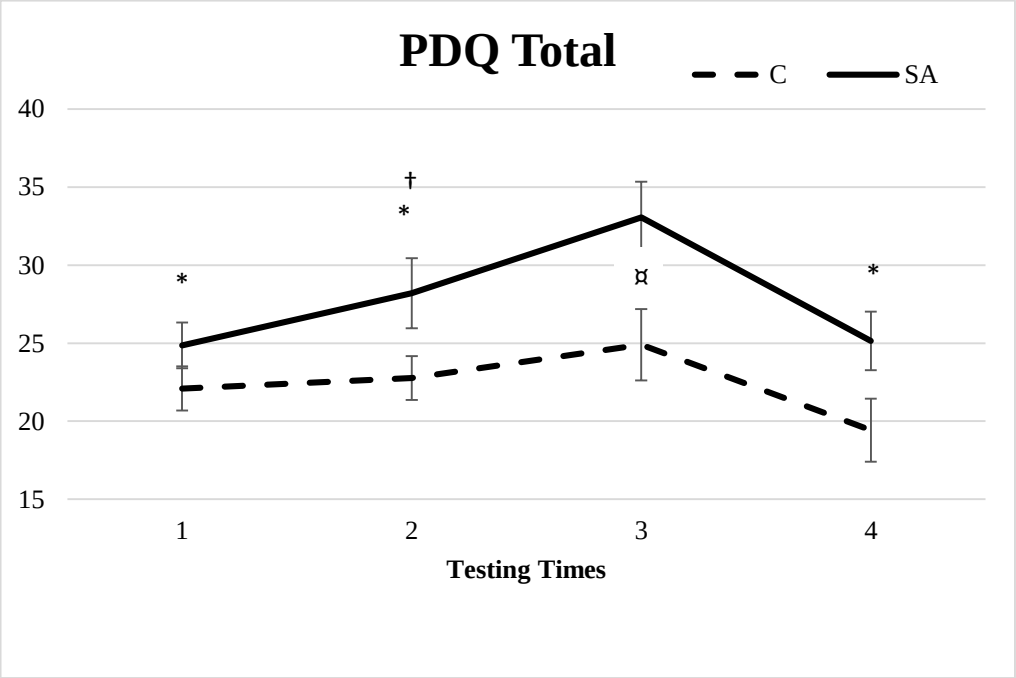
Cognition

Perceived Deficits

Summary scores on the PDQ total are listed in Table 5A of Appendix I and illustrated in Figure 7. There was a significant main effect of time (F = 8.30, p = 0.000), a nonsignificant main effect for group (F = 2.44, p = 0.124) and a significant time by group interaction (F = 3.27, p =

0.023). Simple effects within the student-athlete group demonstrated significant ($p < 0.05$) differences in perceived cognitive deficits during peak training compared to all other times. In addition, baseline significantly differed from typical training but not from recovery. In controls, there were no significant differences across time points. During peak training, student-athletes reported significantly higher PDQ scores compared to the control group ($t = -2.309$, $p = 0.024$). No significant differences between male and female athletes in PDQ total scores ($p > 0.05$) were detected at any time point.

Figure 7. Group means and standard errors on the Perceived Deficits Questionnaire



C = Controls, SA = Student-Athletes
* Indicates significant difference from time point 3 within each group
† Indicates significant difference from time 1 within each group
¤ Indicates significant difference between controls and student-athletes

Stroop Performance

Average response accuracy and reaction time for congruent and incongruent trials at each testing time are presented in Table 6A of Appendix II, response accuracy and reaction times for

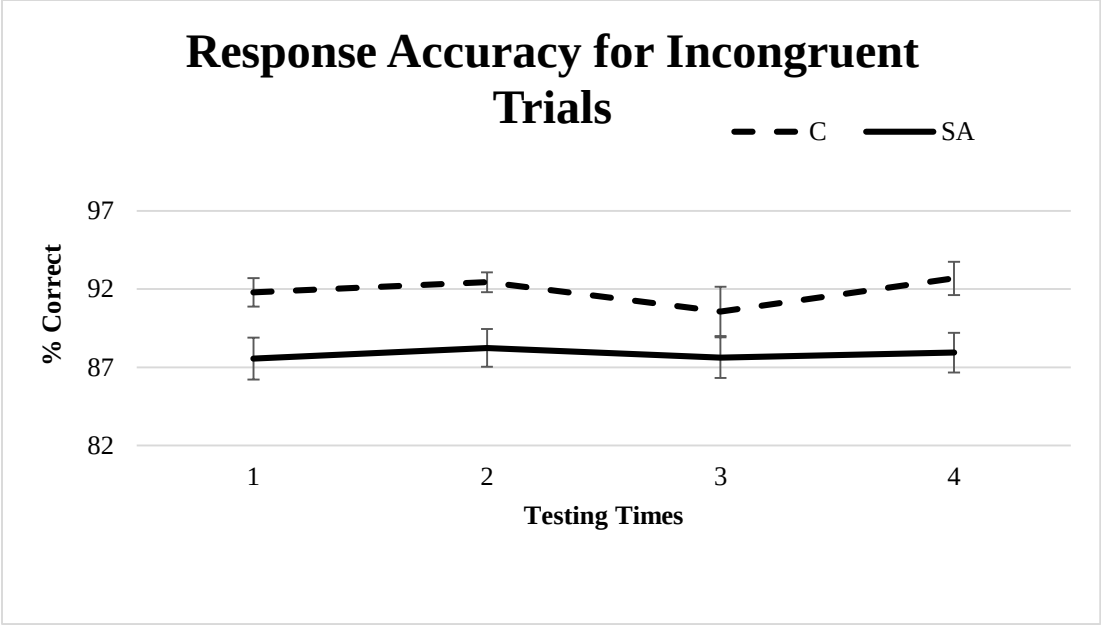
incongruent trials are illustrated in Figures 8 and 9. Response accuracy and reaction times for congruent trials are illustrated in Figures 1A and 2A of Appendix II. Main effects and interactions are presented in Table 2. The response accuracy for incongruent trials during peak training for one student-athlete was removed as it was identified as an outlier. No significant main effect of time or significant interaction was found for any outcome. A significant main effect for group for response accuracy during incongruent trials was observed and Bonferroni adjusted pairwise comparisons revealed that controls were more accurate than student-athletes ($p = 0.020$). At peak training, no significant differences between controls and student-athletes in any Stroop performance variable were observed ($p > 0.05$). No differences between male and female athletes in reaction times or response accuracy for congruent or incongruent trials ($p > 0.05$) were detected at any time point.

Table 2. Results of repeated measures ANOVAs for Stroop Performance

	Time		**Group**		**Interaction**	
	F	***p***	**F**	***p***	**F**	***p***
RA- C	0.411	0.746	0.025	0.874	0.357	0.784
RA-I*	0.576	0.621	5.823	0.020	0.51	0.664
RT-C*	0.878	0.434	1.497	0.227	2.233	0.102
RT-I*	0.523	0.636	1.59	0.213	1.62	0.195

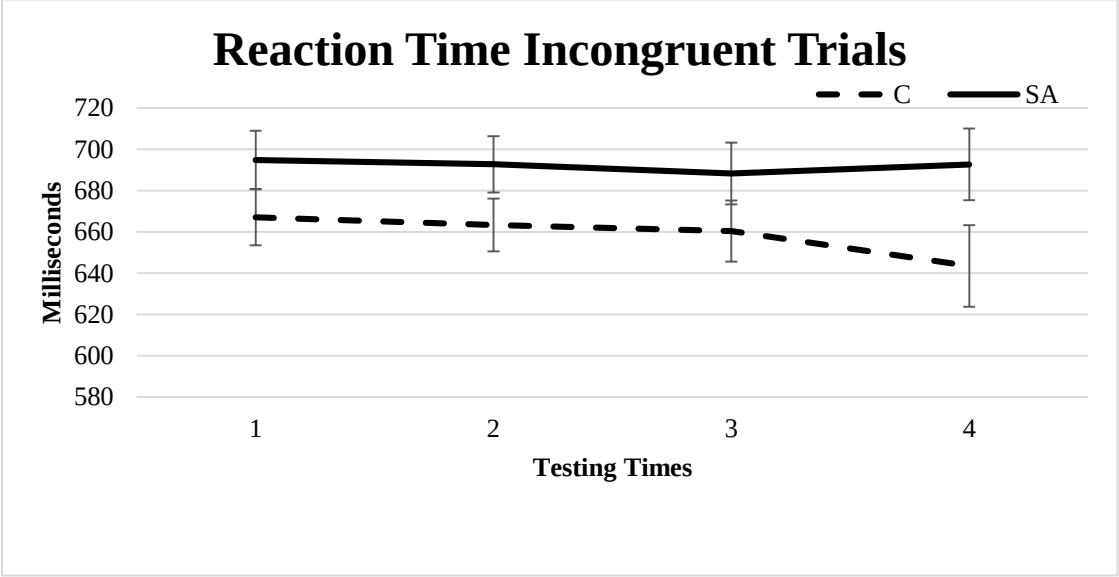
*signifies the data violated sphericity and the Huyn-Feldt correction was applied

Figure 8. Group means and standard errors for Response Accuracy on Incongruent Trials



C = Controls, SA = Student-Athletes

Figure 9. Group means and standard errors for Reaction Time on Incongruent Trials



C = Controls, SA = Student-Athletes

Correlations

Correlations (Pearson's r) across outcomes during peak training are listed in table 3. PSS was significantly and positively related to PDQ total ($p = 0.000$) and POMS TMD ($p = 0.000$), and negatively related to response accuracy for incongruent trials ($p = 0.000$). Thus, higher perceived stress is associated with higher cognitive deficits and total mood disturbance and lower accuracy during the more challenging Stroop trials. Additionally, the PDQ total was positively related to POMS TMD ($p = 0.000$) and negatively associated with response accuracy during incongruent trials ($p = 0.000$) during peak training. No other relationships survived the Holm's procedure to control for multiple comparisons.

Table 3. Relationships amongst outcomes at test time 3 in student-athletes

Test 3	PSS	PDQ	ESS	TMD	Cortisol	RT-C	RT-I	RA-C	RA-I
PSS	1								
PDQ	0.552*	1							
ESS	0.248	0.433	1						
TMD	0.792*	0.537*	0.299	1					
Cort	-0.083	0.071	0.2	-0.076	1				
RT-C	0.131	0.092	0.26	0.152	-0.046	1			
RT-I	0.219	0.065	0.15	0.148	-0.102	0.919*	1		
RA-C	-0.211	-0.225	-0.245	-0.114	0.011	-0.094	-0.078	1	
RA-I	-0.423*	-0.382*	-0.00	-0.350	0.030	-0.063	-0.212	0.398*	1

RT-C = Reaction time for congruent trials, RT-I = Reaction time for incongruent trials, RA-C = Response accuracy for congruent trials, RA-I = Response accuracy for incongruent trials
* Indicates significant relationship at $p < 0.05$

Regression

Multiple regression for variables at testing time 1 that predict TMD at peak training in student-athletes revealed a nonsignificant model including gender, academic load, exercise load,

cortisol and PDQ total and explaining 28.4% of the TMD variance (F = 2.18, p = 0.070). The same variables at typical (time 2) produced a significant model (F =3.03, p = 0.019) explaining 36.9% of the variance in TMD at peak training (Table 6) and PDQ total predicted a significant amount of the variance.

Table 4. Multiple Regression for variables at testing time 2 that predict total mood disturbance at peak training

Time 2	**B**	***t***	***p***
Gender	-9.61	-.87	0.392
Academic Load	.081	.264	0.793
Exercise Load	.47	1.29	0.21
Cortisol	-14.78	-0.835	0.41
ESS	.93	0.639	0.53
PDQ	1.25	2.94	0.006

Exploratory Analysis of Overreached Athletes

Using the criteria of decreased performance and increases in mood disturbance, 10 athletes were identified as OR in this study. Descriptive data and effect sizes for group differences are reported in Table 8. Moderate to large effect sizes (d > 0.50) were observed in perceived stress and total mood disturbance at peak training with OR athletes having higher values and for reaction time to both congruent and incongruent trials at baseline with OR athletes being faster.

Table 5. Means (M) and standard deviations (SD) for variables across a season in well-trained (WT) athletes and those meeting the criteria for overreaching (OR)

		OR		WT		Effect Size
Variable	Test	n	**M ± SD**	n	**M ± SD**	Cohen's *d*

ESS	1	10	10.80 ± 3.79	31	10.45 ± 4.02	0.09
	2	10	12.50 ± 4.60	31	11.35 ± 4.10	0.26
	3	10	13.70 ± 3.95	31	12.48 ± 4.71	0.28
	4	10	10.10 ± 2.81	26	10.69 ± 3.83	-0.18
PSS	1	10	15.30 ± 5.46	31	15.13 ± 5.91	0.03
	2	10	20.60 ± 4.90	31	19.48 ± 6.19	0.20
	3	10	23.00 ± 4.42	31	18.48 ± 6.33	0.83
	4	10	13.10 ± 5.07	26	13.96 ± 6.32	-0.15
TMD	1	10	134 ± 34.17	31	140.13 ± 34.92	-0.18
	2	10	157.8 ± 30.18	31	157.94 ± 38.79	0.00
	3	10	172.6 ± 28.68	31	157.35 ± 38.79	0.45
	4	10	125.9 ± 24.82	26	130.88 ± 35.23	-0.16
PDQ	1	10	23.70 ± 10.66	31	25.10 ± 9.49	-0.14
	2	10	27.70 ± 12.01	31	28.35 ± 15.27	-0.05
	3	10	34.40 ± 12.19	31	32.65 ± 15.44	0.13
	4	10	25.4 ± 9.16	26	25.04 ± 12.16	0.03
Cort	1	9	0.82 ± 0.26	31	0.78 ± 0.25	0.16
	2	10	0.62 ± 0.35	29	0.65 ± 0.57	-0.06
	3	10	0.38 ± 0.12	31	0.41 ± 0.19	-0.19
	4	9	0.77 ± 0.64	25	0.67 ± 0.38	0.19
Response Accuracy Congruent (%)	1	10	95.54 ± 4.54	30	95.54 ± 3.28	0.00
	2	10	96.67 ± 2.36	31	96.11 ± 3.15	0.20
	3	10	94.67 ± 4.91	31	96.16 ± 3.13	-0.36
	4	9	93.62 ± 9.08	25	96.89 ± 2.95	-0.48
Response Accuracy Incongruent (%)	1	10	88.81 ± 4.10	30	87.12 ± 9.75	0.23
	2	10	88.77 ± 4.23	31	88.12 ± 8.83	0.09
	3	9	88.16 ± 3.86	31	87.46 ± 9.16	0.10
	4	9	87.43 ± 7.97	25	88.12 ± 7.31	-0.09
Reaction Time Congruent (ms)	1	10	578.63 ± 73.23	30	618.29 ± 61.73	-0.59
	2	10	587.81 ± 82.09	31	615.16 ± 57.71	-0.39

	3	10	586.38 ± 81.10	31	611.49 ± 64.55	-0.34
	4	9	625.40 ± 105.18	25	619.05 ± 82.23	0.07
Reaction Time Incongruent (ms)	1	10	646.92 ± 76.43	30	711.63 ± 91.91	-0.77
	2	10	666.49 ± 84.19	31	702.76 ± 90.62	-0.41
	3	10	672.91 ± 120.32	31	693.31 ± 88.06	-0.19
	4	9	691.88 ± 107.03	25	693.04 ± 101.80	-0.01

Discussion

This study aimed to characterize psychological and biological adaptations across a competitive season in Division I collegiate rowers to better understand the response of student-athletes to the dynamic demands of academics and athletics. Rather than taking a snapshot of health once during the season, we aimed to collect data on the process of adaptation across a season to identify the expected patterns of change and to predict maladaptive responses. The results of this study show that across the season, athletes demonstrate a pattern of change that is unique from controls. While no gender differences within the athlete group were identified in any outcome at any time, the purpose of the study was to characterize adaptations in the student-athlete population and therefore, combining males and females provided a better summary of that process. The general pattern of athlete adaptation includes healthy baseline values in sleep, mood, stress and cognition followed by a significant increase at either mid-season or peak training and a return to healthy values after the season. This was apparent in every variable studied with the exception of cognitive performance as measured by the Stroop color-naming task and cortisol. Further, several of the stress, mood and cognitive performance outcomes were significantly related during peak training (e.g. stress and perceived cognitive deficits) and perceived cognitive deficits at typical training was able to significantly predicted total mood

disturbance at peak training. Overall, the results of this investigation suggest that the demands of being a student-athlete have a global impact on health at peak training.

Few studies have measured psychobiological outcomes in athletes across a season, leaving the general pattern of adaptation unknown. One of the outcomes that is often discussed but infrequently measured in athlete well-being is sleep. We found that student-athlete daytime sleepiness showed a gradual pattern of increase with the highest values occurring during peak training, surpassing controls. Further, this sleep measurement was not significantly associated with other indices of well-being including cognitive function, suggesting that while there is an increase in perceived sleepiness, it did not impact performance on a challenging cognitive task.

There is evidence that athletes get a similar amount of sleep but of poorer quality compared to controls (Leeder et al., 2012) and the observed values at baseline were similar to those collected at baseline in a sleep intervention study in collegiate basketball players (ESS = 9.64) (Mah et al., 2011). Interestingly, a recent study reported that sleep may be involved in the overtraining process. An intervention study in elite athletes examined whether quantitative or qualitative changes in sleep were involved with the overreaching process in athletes (Hausswirth et al., 2014). One group of athletes completed 3-weeks of an intensified training load while a control group completed their habitual training. Testing occurred after a baseline phase, following the overload phase and after the taper phase. Sleep was quantified using an Actiwatch and self-reported sleep quality. In the intervention group, athletes were labeled as functionally overreached (F-OR) based on decreases in performance with concomitant high fatigue on the POMS. While no differences between the intervention and control groups were observed for any sleep outcome, actual sleep time, sleep efficiency and immobile time following the overload period were significantly decreased compared to baseline in the F-OR group (Hausswirth et al.,

2014). The authors suggest that heavy training may exert a negative effect on sleep quality. Though our study did not quantify aspects of sleep, athletes reported greater daytime sleepiness during peak training compared to controls.

Similar to previous research, collegiate rowers showed positive mental health profiles at the beginning and the end of their athletic season on the POMS. This status has been defined as the iceberg profile with athletes reporting lower levels of anger, depression, tension, confusion and fatigue with higher levels of vigor than population average values (Morgan et al., 1987). Student-athlete rowers in the present study demonstrated an inverted iceberg profile during mid-season and peak training, replicating previous research in endurance sports (Morgan et al., 1987; O'Connor, 1989, Raglin, 1990; Hooper, Flynn, Pizza, Boone, Andres, Michaud, & Rodriguez-Zayas, 1994, Filaire, Slivka, Halson, Hoffman, Berglund, 1994). The total mood disturbance values for collegiate rowers in the present study is similar to what has been reported in longitudinal training studies and was significantly different than controls during peak training demonstrating a unique impact of student-athlete status on mood. We also observed significant changes in mood during typical training, but most athletes recovered by the end of the season. This observed change is interesting because it suggests that total negative mood increases occur earlier in the pattern of adaptation than other measurements such as daytime sleepiness. Total mood disturbance at peak training was also positively and significantly related to perceived stress and perceived cognitive deficits. This constellation of symptoms observed during peak training lends further support a potential global impact of repeated high intensity exercise on mood, perceptions of cognitive deficits and stress.

Quantifying the impact and amount of chronic stress in athletes is a challenging task and perceived stress has not been previously examined across a season in college athletes. The

athletes in this study demonstrated an increase in perceived stress during typical training (time 2) in a similar fashion as the total mood disturbance change. Although the perceived stress at peak training (time 3) did not differ from controls, it was significantly related to a number of variables in student-athletes (perceptions of cognitive deficits, mood disturbance and response accuracy during incongruent trials). Increases in stress have been reported in swimmers (training logs) (Hooper et al., 1995) and rowers (REST-Q) (Kellman et al., 2000) during periods of increased training using different questionnaires. One study reported that perceived stress scores for elite athletes diagnosed as overtrained were significantly higher (PSS = 25) than their healthy athlete counterparts (PSS = 15) (Hynynen, 2008). Our student-athlete group had a score of 19 at peak training suggesting that as a group, they may have been approaching a level of perceived stress associated with clinically meaningful outcomes (e.g. overtraining). Thus, measuring perceived stress may be a simple, useful and effective way to monitor the process of and perhaps prevent some negative aspects of overtraining.

One common biomarker of stress that has been examined as part of the athlete adaptation to training is cortisol. We found that cortisol changed significantly over time but did not differ between groups at any time point and was not related to any other outcome at peak training. These results suggest that resting cortisol is not sensitive to the unique demands of student-athlete rowers. In a previous longitudinal study conducted by O'Connor et al., 1989, collegiate female swimmers had elevated resting cortisol at baseline and during high-intensity training compared to a group of healthy control college students. Additionally, cortisol was significantly related to the POMS depression scores at peak training (O'Connor et al., 1989). We observed similar POMS data but a different pattern of cortisol, which was not associated with mood at peak training. This pattern of cortisol change across time may be sport or season specific.

Previous research in elite rowers has reported no change in cortisol across 7-weeks of intense training (Urhausen, Kullmer & Kindermann, 1987) or across a season in a group of young male national team rowers (Iellamo et al., 200). To our knowledge, this study is the first to report a significant reduction in cortisol at peak training in rowers. Hypocortisolism may be a protective mechanism against chronic elevated levels in athletes, however, a similar pattern was observed for our control participants suggesting something other than student-athlete specific demands were driving this change. For example, when considering that Madison had a very long winter in 2013 and research in endurance athletes has reported decreased overnight urinary cortisol in June when compared to November values in both triathletes and controls (Gouarne et al., 2005), this may be a general seasonal HPA axis response. A recent review of resting salivary cortisol in athletes concluded that no consistent difference has been identified between athletes, sedentary or physically active control participants (Cevada et al., 2014). Based on the research on resting salivary cortisol, a more critical analysis of the usefulness of an absolute resting value may be necessary. Due to the varying types of adaptation that may be occurring throughout the HPA axis (production, release, receptor affinity and number, use, elimination), nuanced changes within an individual over time may be obscured by analyses using group averages.

In order to extend the knowledge of adaptation across a semester and competitive season, perceived deficits in cognition and behavioral changes in an executive function task were measured. Collegiate athletes in this study reported a significant increase in total perceived cognitive deficits beginning early in training (typical training - time 2) followed by another increase at peak training and then a return to similar values as baseline after the season (recovery). The value at peak training differed from controls and was related to mood and response accuracy during incongruent trials suggesting that cognition was being impacted in a

similar manner in response to student-athlete demands. The lack of change in the control group suggests that this is not due to the increased academic demand of the semester. Interestingly, though athletes reported increased perceptions of cognitive difficulty, they were able to maintain their performance on a challenging executive function task. No learning effect was observed and performance did not significantly change across the season/semester. Response times and response accuracy in this study were similar to what has been reported in healthy adults on the Stroop task (Zurron et al., 2009; Liotti et al., 2000). Both our groups demonstrated resiliency to cognitive deficits during the most stressful time of the semester/season. While no group differences were reported in Stroop performance, response accuracy during incongruent trials at peak training was related to stress, mood and cognitive deficits in student-athletes. Thus, while Stroop performance was not sensitive to dynamic influences occurring across a competitive season, response accuracy during the more challenging trials at peak training was related to several indices of central nervous system function.

Previous research assessing cognition in athlete overtraining has highlighted the similarities with major depression and chronic fatigue syndrome and hypothesized that psychomotor speed, or the speed of information processing, will be slower in athletes showing signs of overtraining (Nederhof et al., 2006). Several studies report nonsignificant changes in performance on the more challenging trials of their cognitive task following short duration (10 days-2-weeks) intense training camps (Neverhof et al., 2007; Dupuy et al., 2010). Rietjens, 2005, examined cognition before and after an exercise camp and reported no changes in mood, body mass, body fat, exercise performance or lactate during testing. Faster reaction times during a finger cuing task were found during the post-test for both athletes and controls suggesting a learning effect. Additionally, the learning effect was larger for the control group compared to the

intervention group but only for the most challenging conditions (uncued and neither-cued trials). Thus, intense exercise training may negate a learning effect that would occur in tasks that are more challenging. The authors concluded that central nervous system fatigue precedes peripheral fatigue and that cognitive ability is an early biomarker of overtraining. This group of studies is limited in a number of ways primarily in the small sample sizes, short exercise interventions and selection of cognitive tasks. Thus, clarification of the pattern of adaptation in cognitive function across a competitive season is needed.

In order to further assess differences between well-trained athletes and those who were not adapting positively, we identified 10 athletes (four female) who reported having decreased individual performance in the last month at time 3 or 4 and had increased total mood disturbance. This number of athletes (approximately 25% of the sample) is similar to what has been previously reported (O'Connor et al., 1989). Small effect sizes were observed for reaction time during peak training for both incongruent and congruent trials with OR athletes performing faster, directly contradicting the psychomotor speed hypothesis. Moderate effect sizes were found for perceived stress and mod disturbance during peak training, which is not surprising as mood disturbance change was a criteria for identifying the athlete. Therefore, while our data do not support the psychomotor speed hypothesis in this sample of collegiate athletes, TMD and PSS appear to be different between the well trained athlete group and those meeting our criteria for overreaching during peak training.

This study is limited in a number of ways. First, due to the difficulty of scheduling we missed data from participants at each time point. Specifically, a number of students were unavailable during the final testing point due to starting new jobs, moving or vacationing, and while we tried to get their data during a 2-week period, we missed a few participants. If

participants were unable to return to campus for the cognitive testing and a saliva sample, they were still emailed the questionnaires and some completed them. Second, the Stroop task may not have been challenging or sensitive enough to reveal subtle deficits occurring across a season in cognition. Additionally, as there are no specific criteria for identifying athletes as overreached, we chose self-reported decreases in performance rather than objective markers and we chose an increase in TMD of 10 points as an arbitrary cut-off. Finally, although a healthy physically active control group was used, only collegiate rowers (endurance athletes) were studied so the data are limited to this population. Future research addressing changes in cognition should employ challenging tasks addressing different cognitive systems including executive function, working memory and selective attention to ascertain whether the demands across a season in different sports is having a selective effect on a particular cognitive system.

Endurance athletes undergo increases in training volume during their competitive season. Although many attempts have been made to define the psychobiological pattern across a competitive season, the sequence of central nervous system adaptation remains undefined. Our results suggest that student athletes present a unique psychological pattern compared to physically active student controls, but do not differ in either cortisol or cognitive performance as a function of the competitive athletic season. Our results also suggest that some behavioral responses occur before peak training such as increases in mood disturbance, perceived stress and perceived cognitive deficits and that these variables are related at peak training. Further, while student-athlete experience a general increase in central nervous system symptoms during peak-training, values return to healthy baseline values after the season. Additionally, perceived cognitive deficits were predictive of mood disturbance at peak training suggesting that this measurement is sensitive to the training season and related to one of the major components

involved in athlete overtraining. The literature hypothesizing a behavioral cognitive component to the overreaching process is in its infancy and has significant methodological limitations. In the interest of student-athlete health, more research is needed to characterize objective changes in cognition. The student-athlete is constantly adapting to the demands of their environment and understanding the pattern of change over the season can help to improve student-athlete well-being.

4.3 Fluctuations in Well-Being across a Semester in Physically Active College Students and Student-Athletes

The college student population encounters a range of demands and challenges that fluctuate over the course of the semester including academic, social and personal factors that affect their holistic development (Gayles, 2007). While research supports this time in a young adult's life as important and stress-inducing, little is known about the way that health outcomes differ within subpopulations of college students, such as in student-athletes. Research on collegiate athletes shows both positive and negative consequences of participating in collegiate athletics while highlighting many health similarities to non-athlete college students. However, as data on students are typically collected at one time point, it is unclear how the response to the demands of college is affected across a semester and how that pattern might differ from non-athletes and across genders. Importantly, as the demands of the college environment change over time, it is plausible that fluctuations in college-related demands are reflected in student mental health outcomes such as mood and cognition.

Student-athletes differ from non-athlete college students primarily in the time and physical demands of their sport involvement. The majority of Division I student-athletes report spending more than 30 hours per week on their sport and only 2% report spending less than 20 hours a week (Brown, 2000). Wilson (2005) found that compared to non-athlete freshman, a sample of freshman student-athletes at a small private Division I school reported significantly greater stress from the number of responsibilities, not enough sleep, heavy demands of extracurricular activities, boyfriend/girlfriend's and family with no differences in academic problems. Thus, unique time constraints and sport-related stressors of student-athletes may have important health consequences over the course of their season that differs from their college non-

athlete counterparts. Despite this large time commitment, Division I freshman scholarship athletes go on to have a graduation rate of 82% (www.ncaa.org). Although student-athletes enter college with lower grades and lower standardized test scores than non-athlete students (Aries, 2004), they show similar academic progress (Hood, 1992; Aries, 2004) suggesting that sport involvement does not impede academic success. On one hand, understanding academic success is important in the college athlete population, but performance-based academic outcomes may not the most sensitive measure of exercise-influenced changes in well-being or cognitive function that can occur across a competitive season.

An additional area of research focusing on student-athlete academics and health addresses gender differences. In spite of the androcentric institution of athletics, female student-athletes might benefit from participating in college athletics to a greater degree than male athletes. Division I female freshman student-athletes report more interactions with non-teammates and higher self-concepts compared to male student-athletes (Gaston-Gayles, 2009), are more satisfied with their experience, interact with faculty more frequently and participate in active and collaborative learning experiences more than non-athlete females with similar levels grades (Umbach, 2007). On the contrary, it has been suggested that the female athlete may experience gender role conflict given that the stereotypical definitions of femininity are violated by the assertive and competitive nature of athletics (Allison, 1991). This conceptualization has not been supported in the literature (Allison, 1991; Miller, 1996; Desertrain, 1988). In fact, Ross (2008) reported that in a small group of Division I female athletes, themes such as being comfortable with being more muscular, appreciation of being powerful and confidence emerged over the course of an interview. This literature proposes that participating in athletics might

benefit the overall college experience of female athletes compared to female non-athletes but it remains unknown whether these groups differ in wellness outcomes across a semester.

The negative impact of participating in college athletics on mental health, specifically depression has been assessed. Overall college prevalence rates of depression are typically around 25%, is higher among women and depressed students report significantly higher amounts of stress (Lindsey, 2009; Tupler et al., 2015). Athletes experience psychiatric symptoms at a similar to lesser degree compared to the greater college population (Armstrong, 2009; Donohue, 2004). In athletes, psychiatric symptoms are often measured at the beginning of the season with one study reporting 31% of NCAA football players endorsing elevated depression scores prior to season commencement (Bailey, 2010) and female student-athletes reporting more cognitive, emotional and sleep symptoms than male athletes (Covassin, 2012). Yang (2007) collected psychological data on 257 NCAA athletes and reported that 4% of athletes indicated a history of major depressive disorder with 21% of the total sample reporting elevated symptoms of depression. The largest predictors for depressive symptoms were being female, being a freshman and reporting elevated pain during the preceding week (Yang et al., 2007). Overall, student-athletes report their general well-being as significantly higher than non-athlete college students (Aries, 2004) but there are still individuals who experience mood disturbance. Interestingly, although female student-athletes academic experience may benefit by participating in athletics, they are still at risk for experiencing the higher depressive symptoms found in women in the college student body and the population at large (Alexander, 2007; Munce, 2007).

Research addressing well-being outcomes of student-athletes is useful to inform coaches and supporting athletic staff of important athlete-specific considerations. Unfortunately this literature is limited by data that are collected at one time point (prior to the season), are self-

report, and data from all sports and different institutions are often combined. This method of data collection and analysis may preclude identification of health variables that fluctuate over the course of a competitive season, differ across genders and from non-athlete students. Therefore, the purpose of this study was to test differences in sleep, mood, stress and cognition across a semester in male and female collegiate athletes and physically active healthy male and female college students. It was hypothesized that student-athletes would be similar to non-athlete students at the beginning and end of the semester. However, athletes, but not controls, would show significant change over the semester in all outcomes during peak training. Female athletes were expected to have higher tension at all testing times compared to male athletes.

Methods

This manuscript is an extension of the adaptations paper that allows for a closer assessment of the role of gender in these outcomes. Therefore, the methods are the same and the only unique aspect of this manuscript are the analyses of multiple groups (female and male controls and female and male athletes). For complete methods, the reader is referred to chapter 3 of this dissertation.

Statistical Analyses

Descriptive data are presented as means and standard deviations and frequency when applicable for female controls, male controls, female athletes and male athletes. Differences in characteristics were assessed using one-way analysis of variance (ANOVA) using Bonferroni post-hoc tests. Frequency differences were assessed using Kruskal-Wallis H tests. Within and between group differences in, sleep, mood, stress and cognitive function were analyzed with 4 (time) x 4 (groups) repeated measures analysis of variance (ANOVAs) applying a familywise

alpha of 0.05. Following significant main effects of group or interactions, Tukey's post hoc tests were applied to examine differences across groups. To control for Type I error from multiple comparisons, Holm's sequential procedure was applied to the familywise alpha of 0.05 only for significant main effects of group and interactions. If the assumption of sphericity (significant Mauchly's test) for the within-subjects design was violated, a data-driven adjustment was made using Huynh-Feldt's epsilon correction. For all measures, the level of significance was $p < 0.05$.

Results

Group descriptions are presented in Table 1. For additional descriptive information regarding numbers of exams and papers, illness and injury frequency for each testing time, please see Table 1A and Table 1B in Appendix III titled "Gender Differences". Analyses of the descriptive data revealed that the control groups were more ethnically diverse than the athlete groups and that female ($p = 0.034$) and male athletes ($p = 0.019$) had significantly fewer credits than male controls. Female controls reported significantly less leisure time physical activity compared to both athlete groups ($p < 0.000$). Groups were similar in class distribution, age and body mass index.

Table 1. Group Descriptives for frequency (% yes) and demographics (M = mean, SD = standard deviation).

Time 1	Female Controls	Male Controls	Female Athletes	Male Athletes	Test Statistic	*p*
N	15	8	22	21		
Ethnicity %					$\chi^2 = 8.94$	0.03
Caucasian	86.7	75	100	100		
Asian American	13.3	12.5	0	0		
African American	0	12.5	0	0		
Class %					$\chi^2 = 3.30$	0.348
Sophomore	0	0	18.2	28.6		

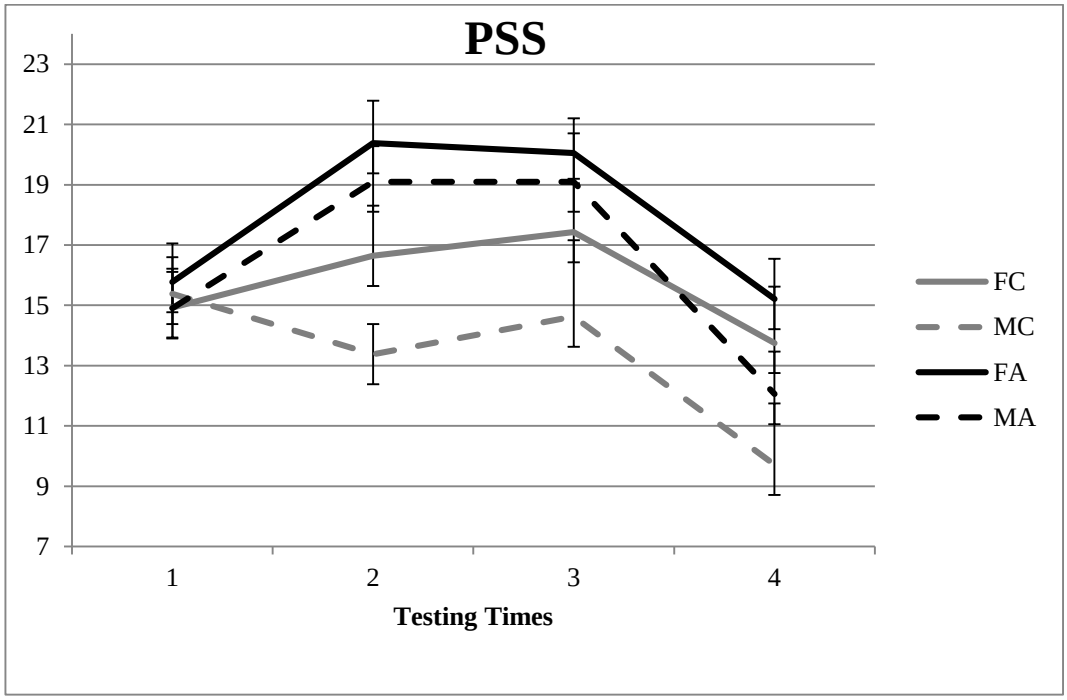
Junior	60	62.5	63.6	19		
Senior	33.3	37.5	18.2	47.6		
Age (M ± SD)	20.60 ± 0.63	21.25 ± 1.04	20.32 ± 0.78	20.71 ± 1.27	F = 1.91	0.137
BMI (M ± SD)	23.18 ± 1.76	22.93 1.82	23.35 1.33	24.21 1.88	F = 1.68	0.181
Credits (M ± SD)	14.33 ± 1.99	15.75 ± 1.28	13.77 ± 1.51	13.62 ± 1.72	F = 3.55	0.019
Godin (M ± SD)	67.07 ± 18.51	87.25 ± 42.42	116.5 ± 37.4	122.9 ± 37.2	F = 9.33	0.000

SA = Student-Athlete, Godin = Godin Leisure Time Physical Activity Questionnaire, BMI = body mass index

Stress

For the PSS, there was a significant main effect of time (F = 15.178, p = 0.000) and nonsignificant interaction (F = 1.587, p = 0.12) and group main effects (F = 1.704, p = 0.18). Average scores for each group are presented in Table 2A of Appendix III and illustrated in Figure 1.

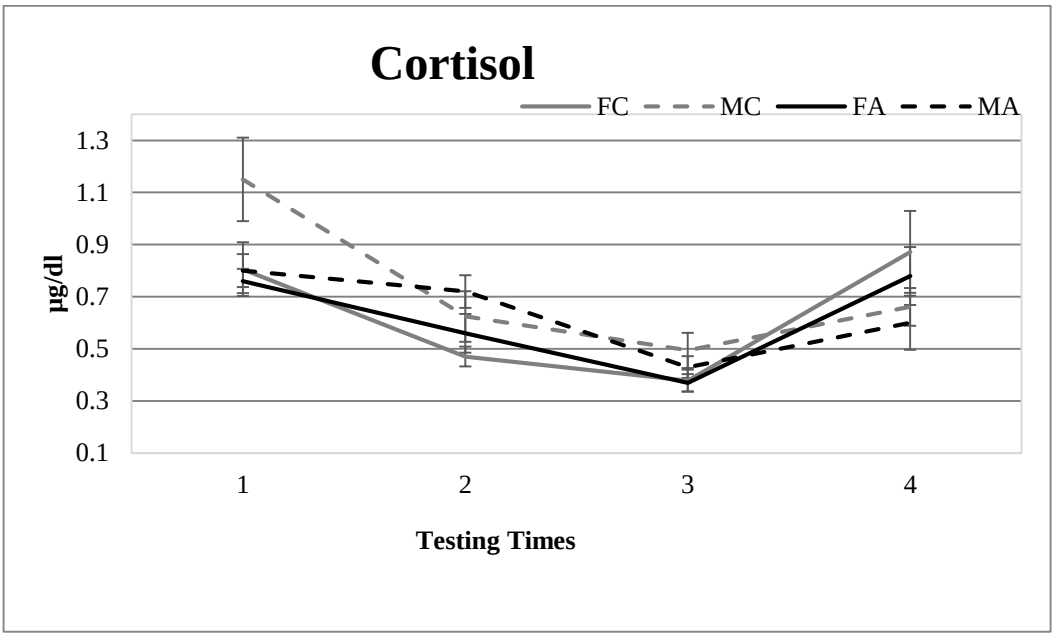
Figure 1. Group means and standard errors on the Perceived Stress Scale



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Analyses of resting cortisol data revealed a significant main effect of time (F = 18.589, p = 0.000, Huynh-Feldt corrected) with nonsignificant interaction (F = 1.103, p = 0.365, Huynh-Feldt corrected) and group main effects (F = .220, p = 0.882). Data for each group are presented in Table 3A of Appendix III and illustrated in Figure 2.

Figure 2. Group means and standard errors in resting cortisol (µg/dl)



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Exercise, Academics, Mental and Physical Fatigue

VAS data for perceived academic load, exercise load, mental and physical fatigue are provided in Table 4A of Appendix III and illustrated in Figures 3-6. The results of the time by group repeated measures ANOVAs are presented in Table 2. There was a significant main effect of group and interaction for exercise load. Tukey's post-hoc tests revealed that FC differed from FA and MA (p < 0.001) and that MC differed from FA (p = 0.021). There was also a significant main effect of group for physical fatigue but the interaction did not survive the Holm's procedure for multiple comparisons. Tukey's post-hoc tests revealed that the two control groups differed

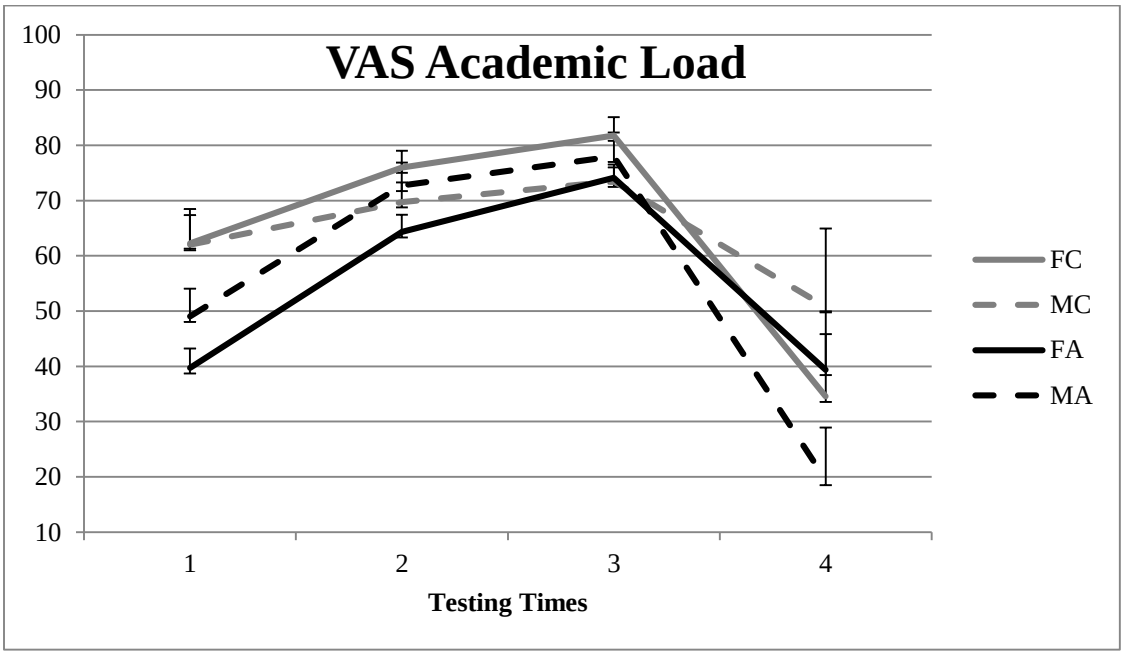
significantly from the athlete groups (FC differed from FA and MA, $p = 0.001$, MC differed from FA and MA, $p = 0.001$).

Table 2. Results of repeated measures ANOVAs for VAS outcomes

	Time		**Group**		**Interaction**	
	F	***p***	**F**	***p***	**F**	***p***
Academic Load*	26.95	0.000	1.36	0.270	1.08	0.385
Exercise Load*	5.50	0.004	9.27	0.000	7.07	0.000
Mental Fatigue*	35.61	0.000	1.17	0.330	1.72	0.094
Physical Fatigue*	6.25	0.000	11.79	0.000	2.53	0.014

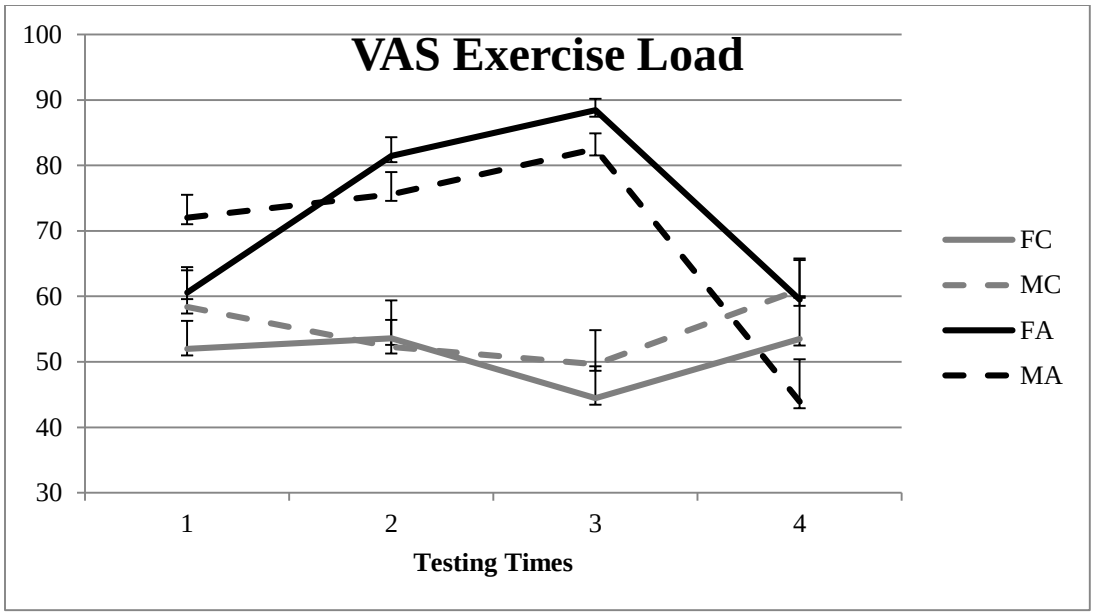
* Signifies that data violated the assumptions of sphericity and the Huynh-Feldt correction was applied

Figure 3. Group means and standard errors on the Visual Analog Scale for academic load



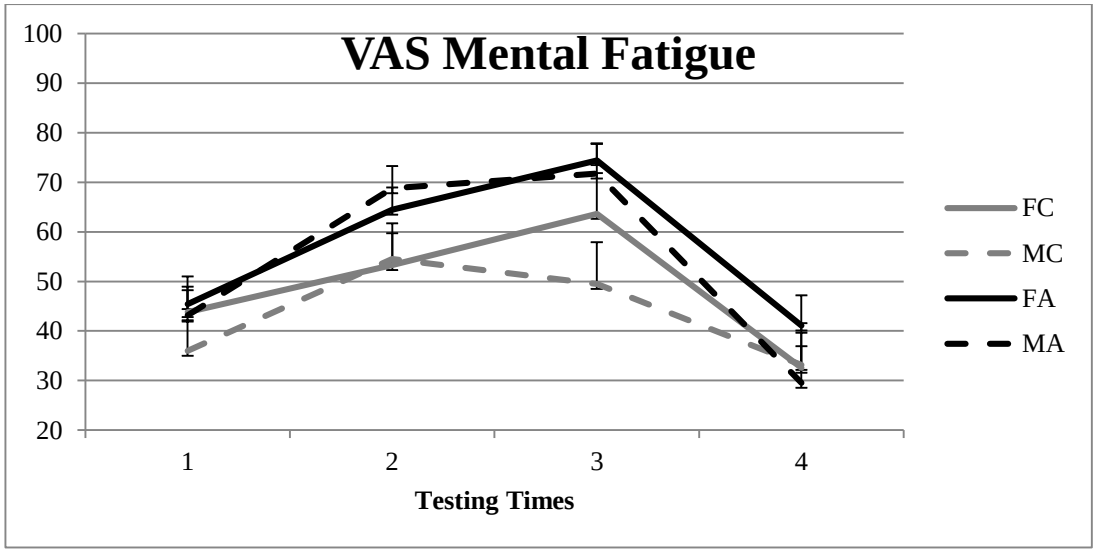
FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Figure 4. Group means and standard errors on the Visual Analog Scale for exercise load



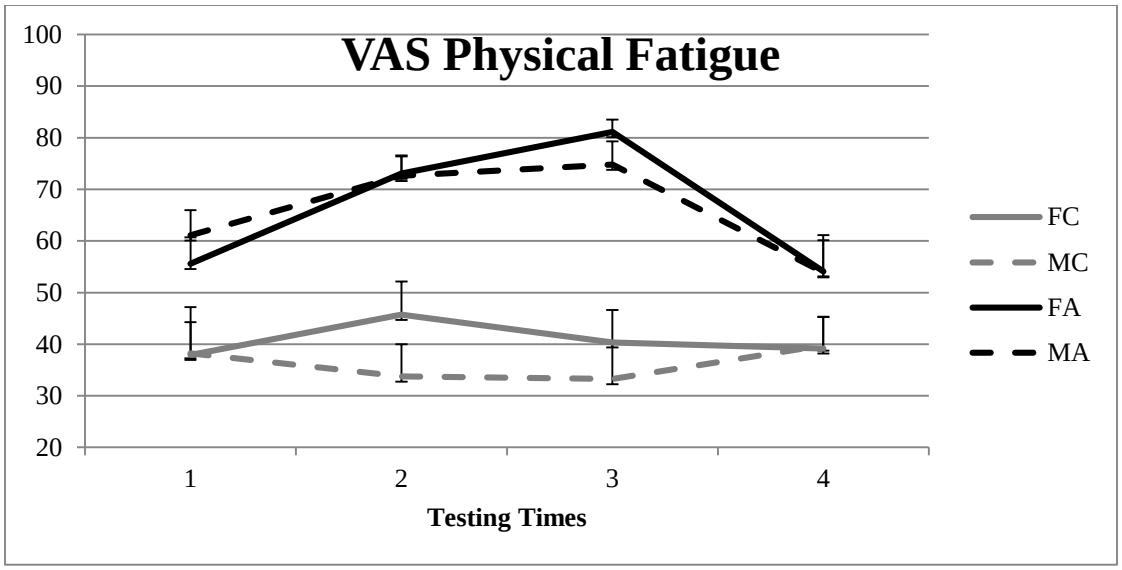
FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Figure 5. Group means and standard errors for Visual Analog Scale for mental fatigue



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Figure 6. Group means and standard errors for Visual Analog Scale for physical fatigue

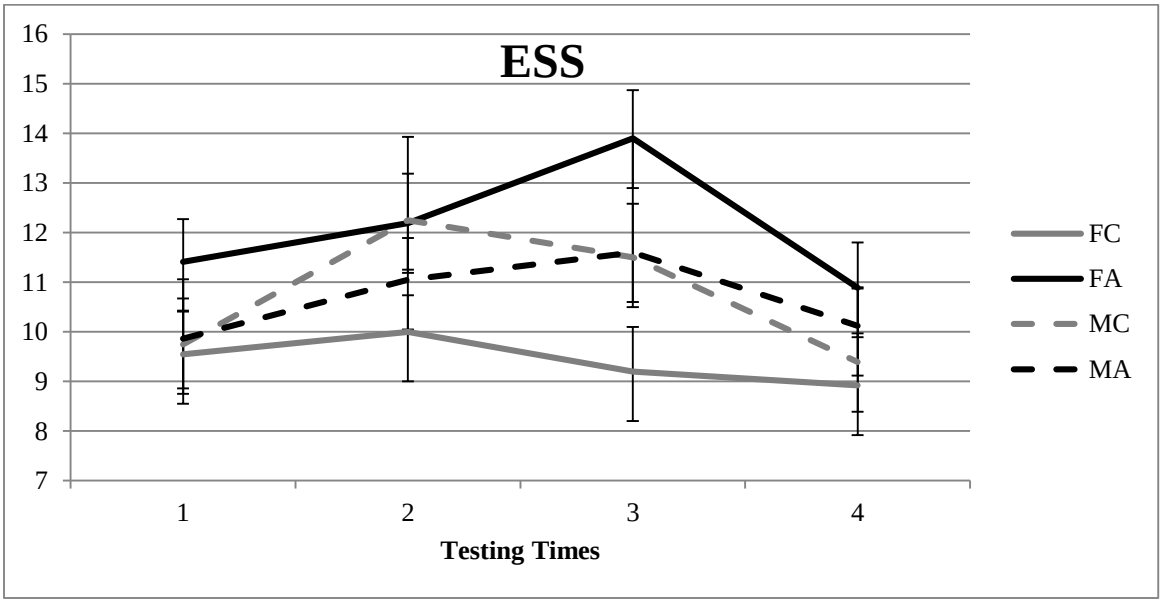


FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Sleep

Daytime sleepiness demonstrated a main effect time (F = 6.375, p = 0.000) and nonsignificant interaction (F = .945, p = 0.488) and group main effects (F = 1.232, p = 0.308). ESS total values are presented in Table 5A of Appendix III and illustrated in Figure 7.

Figure 7. Group means and standard errors on the Epworth Sleepiness Scale



FC = Female Controls, MC = Male Controls, FA = Female-Athletes and MA = Male Athletes

Cognition

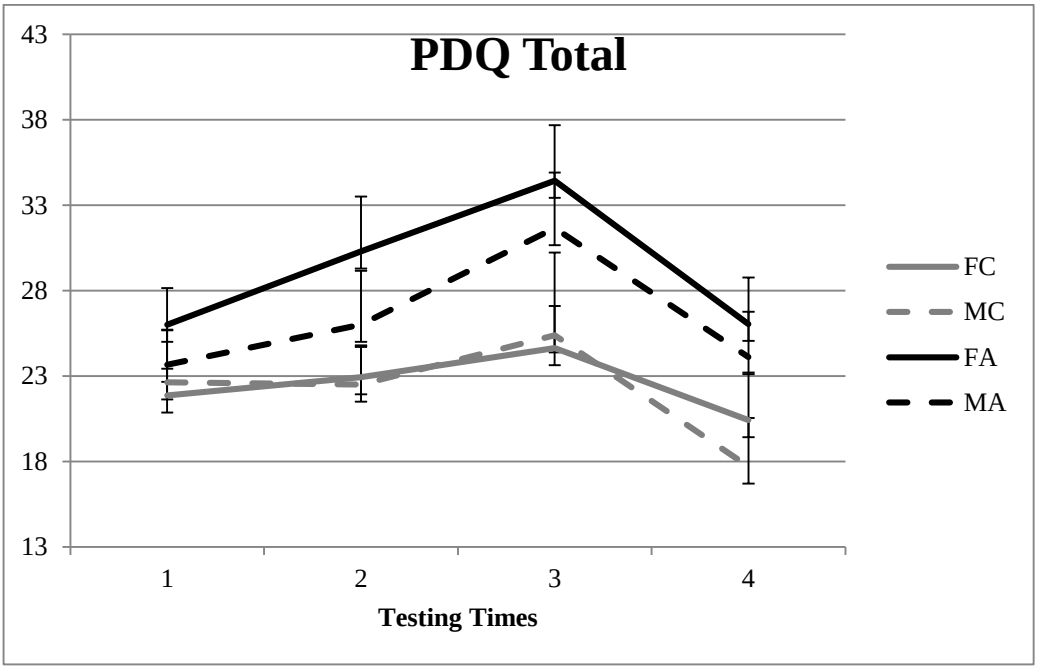
Values for the subscales on the PDQ are presented in Table 6A of Appendix III and illustrated in Figure 8. The results of the time by group repeated measures ANOVAs are presented in Table 3. Tukey's post hoc tests following the significant interaction of the attention subscale revealed no significant differences across groups ($p > 0.05$).

Table 3. Results of repeated measures ANOVAs Perceived Deficits Questionnaire

	Time		**Group**		**Interaction**	
	F	***p***	**F**	***p***	**F**	***p***
Attention	14.69	0.000	1.19	0.324	2.654	0.007
Retrospective Memory*	4.36	0.006	0.774	0.514	1.81	0.072
Prospective Memory	6.97	0.000	1.79	0.162	1.36	0.212
Planning	6.67	0.000	1.29	0.289	0.785	0.63
Total	8.28	0.000	1.23	0.31	1.44	0.177

* Signifies that data violated the assumptions of sphericity and the Huynh-Feldt correction was applied

Figure 8. Group means and standard errors on the Perceived Deficits Questionnaire



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

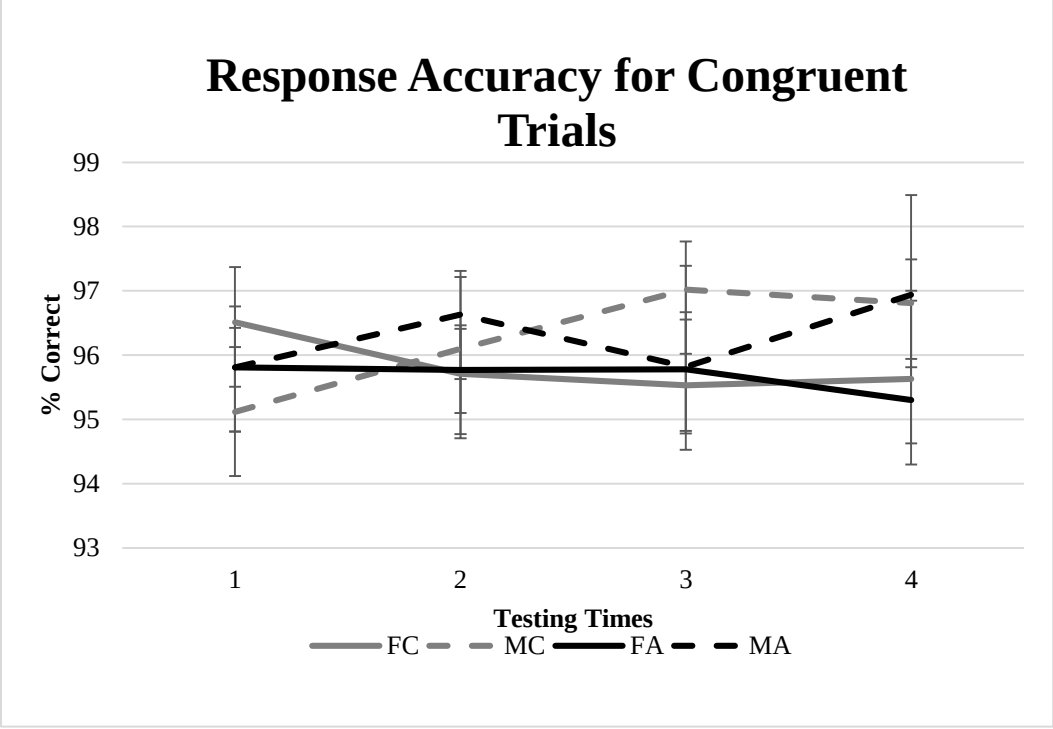
Behavioral results of the Stroop task are presented in Table 7A of the Gender Difference Appendix and illustrated in Figures 9-12. The results of the time by group RM ANOVA are presented in Table 4. There were no significant main effects or interactions in response accuracy or reaction time to either congruent or incongruent trials.

Table 4. Results of repeated measures ANOVAs for Stroop Performance

	Time		**Group**		**Interaction**	
	F	***p***	**F**	***p***	**F**	***p***
RA- C*	0.481	0.692	1.956	0.134	0.497	0.871
RA-I*	0.503	0.681	0.226	0.878	0.816	0.603
RT-C*	0.91	0.424	0.558	0.645	0.861	0.546
RT-I*	0.398	0.729	0.614	0.609	0.693	0.695

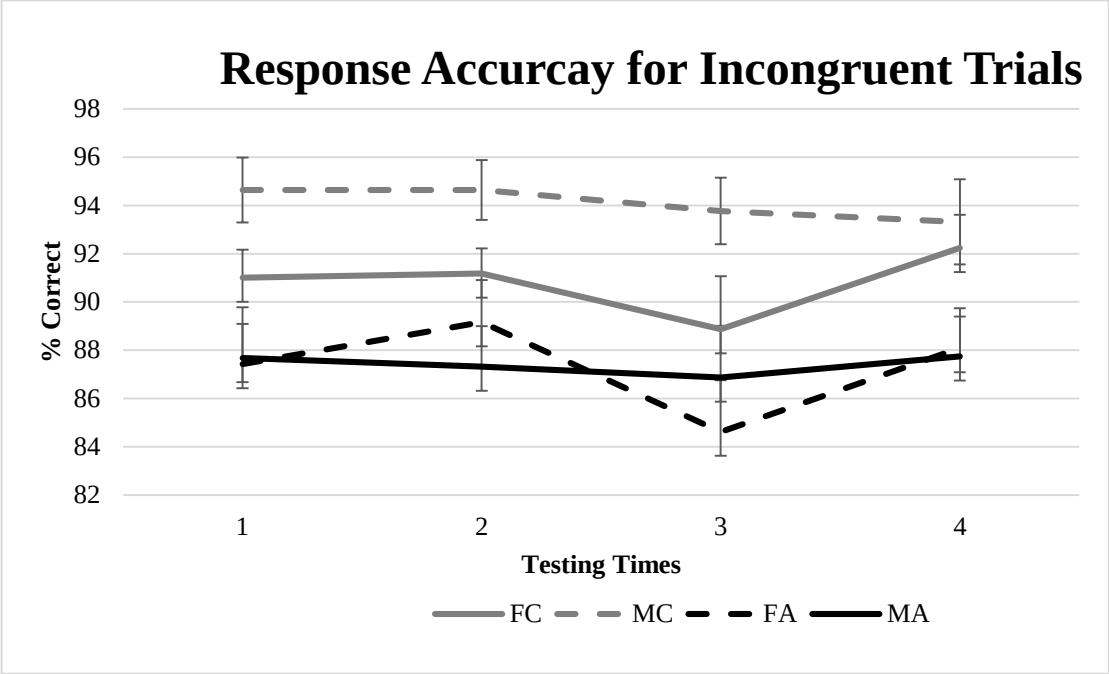
* Signifies that data violated the assumptions of sphericity and the Huynh-Feldt correction was applied
RA-C = Response Accuracy for Congruent Trials, RA-I = Response Accuracy for Incongruent Trials, RT-C = Reaction Time Congruent Trials, RT-I = Reaction Time Incongruent Trials

Figure 9. Group means and standard errors for Response Accuracy on Congruent Trials



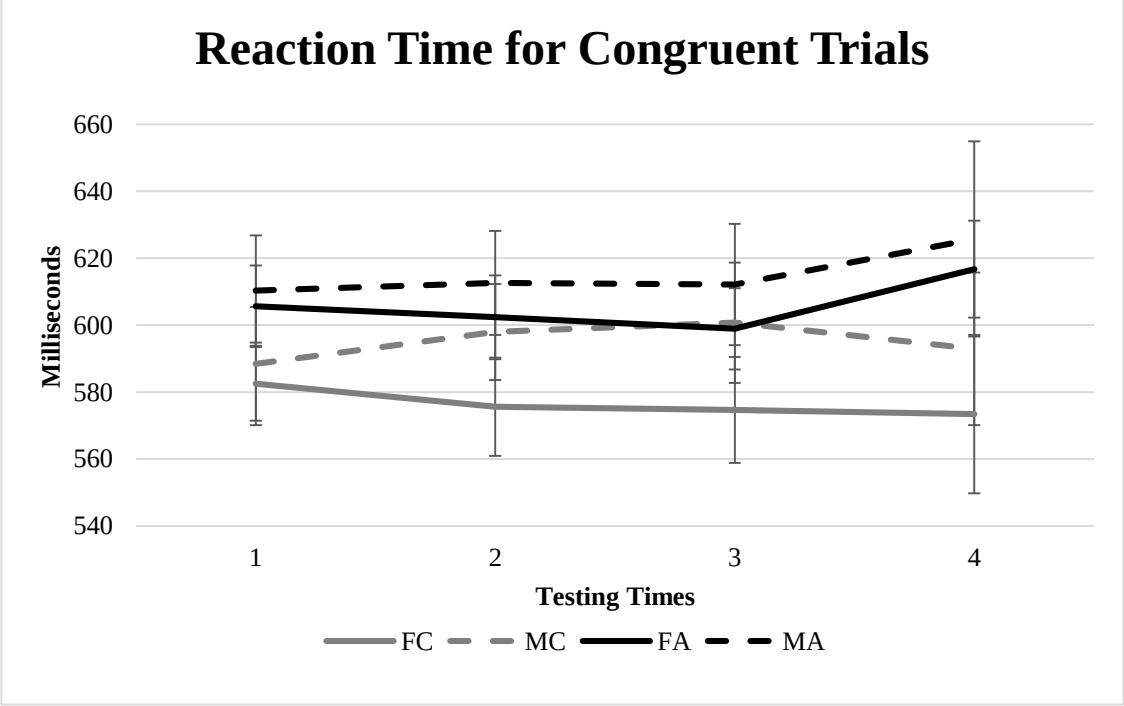
FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Figure 10. Group means and standard errors for Response Accuracy on Incongruent Trials



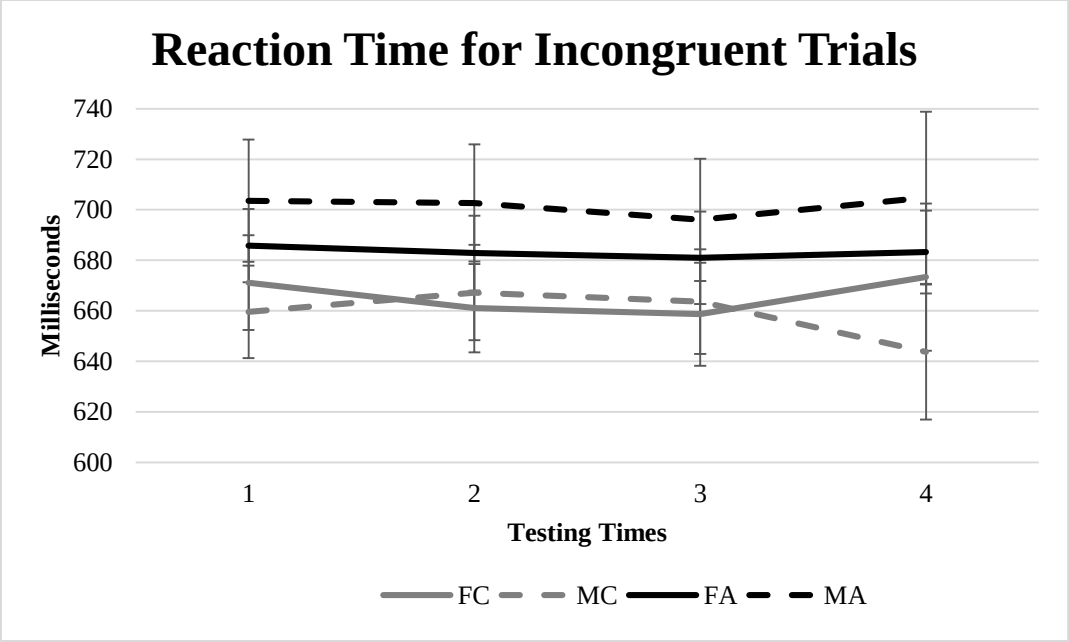
FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Figure 11. Group means and standard errors for Reaction Time on Congruent Trials



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Figure 12. Group means and standard errors for Reaction Time on Incongruent Trials



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Mood

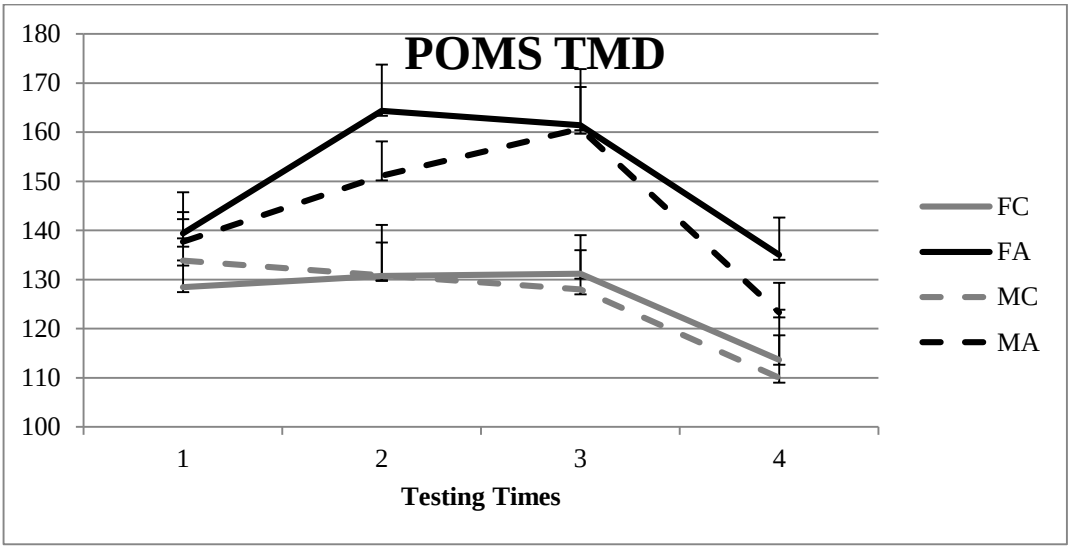
Group averages for the POMS subscales are presented in Table 8A of the Appendix and Figure 13 illustrates group means for the total mood disturbance (TMD) scores across the competitive season and academic semester. The results of the group by time repeated measures ANOVAs are presented in Table 5. The group main effects of anger and vigor did not survive the correction for multiple comparisons procedure. There was a significant main effect of Group (p =0.015) and a significant Group by Time interaction ($p < 0.001$) for Fatigue. Tukey's post hoc tests indicated that FC had significantly lower fatigue than FA ($p = 0.045$).

Table 5. Results of repeated measures ANOVAs for the Profile of Mood States

	Time		**Group**		**Interaction**	
POMS	**F**	***p***	**F**	***p***	**F**	***p***
Tension	13.37	0.000	0.81	0.50	1.38	0.20
Depression	6.28	0.000	1.07	0.369	1.44	0.176
Anger	2.44	0.067	3.55	0.021	1.89	0.06

Fatigue	15.50	0.000	3.841	0.015	2.44	0.001
Vigor	13.04	0.000	3.03	0.038	0.73	0.69
Confusion	12.23	0.000	0.72	0.54	1.34	0.22
TMD	13.04	0.000	2.29	0.09	1.83	0.068

Figure 13. Group means and standard errors for the Profile of Mood States Total Mood Disturbance



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Discussion

The results of the present study demonstrate that although measurements of stress, mood, sleep and cognitive function change significantly over the course of a semester, the patterns of change are similar across athlete and non-athlete groups and genders. While the patterns of changes are similar, the magnitude of changes appears to be greater in student-athletes and these differences may have been significant with larger sample sizes. The only significant interactions that were observed were for perceived exercise load and physical fatigue and these values were higher in the student-athlete groups compared to the controls. Considering that part of the inclusion criteria for control participants was being physically active and being involved in at

least 20 hours of extracurricular activities, the samples were well matched for time commitments and academic load. Overall, the groups demonstrated similar fluctuations in well-being outcomes across a semester.

In regard to stress outcomes, our study demonstrated that both perceived stress and resting cortisol changed over the course of the semester, but did not differ significantly by group. Because of their responsibilities, student-athletes report that the causes of stress stem from both academic and athletic loads, time constraints, relationships and finances (Humphrey, 2000); greater amount of stress could be perceived by student-athletes. However, as the control group was well matched for academic load and extracurricular activities, no group differences were observed. Further, perceived stress scores as high as 35 have been reported in college students (Baghurst, 2014) supporting the healthy and relatively low stress profiles of our general sample with scores ranging from 13 to 20 at peak training. A consistent gender difference in perceived stress has been reported across the lifespan (Cohen et al., 2012). Specifically, during college, scores on the perceived stress scale are higher for women (Ansar et al., 2014; Gambetta-Tessini et al., 2013) and related to a variety of other health outcomes such as psychological distress, pains/aches and gastro-intestinal complaints in college students (Ansar, 2014). The data from this study are inconsistent with these findings, likely due to the healthy profile and homogeneity of the students who volunteered.

Another measure of stress that showed a significant effect of time was resting cortisol levels. Similar to perceived stress, no interaction or main effect of group was observed showing that cortisol changed in a similar pattern across time in non-athlete and student-athlete males and females. A previous study measured changes in resting afternoon cortisol and POMS across a season in collegiate female swimmers and female control students (O'Connor et al., 1991). The

results of this study showed reported that female athletes had significantly higher resting afternoon cortisol (.340 µg/100ml) compared a college control group (.225µg/100ml) during baseline and peak training. The values in the present study are higher in both student-athletes (0.76 µg/dl) and the control sample (0.81 µg/dl) at baseline than what was reported in that study with no differences between groups. Previous research has also reported higher resting cortisol values in college-aged men compared to women in all phases of their menstrual cycle (Walder, 2010; Maestripier, 2010). College-aged women have also shown greater cortisol responses to acute psychological and academic stressors (Maestripier, 2010; Weekes, 2008). The lack of gender and group differences in this study may be due to the healthy but busy nature of both groups.

Similarly, daytime sleepiness changed significantly over the semester, but groups did not differ. Typical Epworth Sleepiness scores in college students range from 7 to 9.5 suggesting that our sample (9-13) may be at the higher end of the sleepiness spectrum (Lund et al., 2010; Moo-Estrella). There are data to support lower sleep quality in elite athletes compared to controls with male athletes spending more time in bed (Leeder, 2012). Moreover, decreases in sleep quality in a portion of athletes have been reported following large increases in training volume (Hausswirth, 2012). Previous gender research has reported that college women have earlier bed times, earlier wake times but also more awakenings during the night, lower sleep quality and higher daytime sleepiness (Tais; Moo-Estrella; Singleton, 2008). Additionally, college women have scored higher on daytime sleepiness compared to men but this score did not relate to GPA or academics (Howell, 2004). Our data do not support a difference between athletes and physically active student controls, nor males and females, in daytime sleepiness. Interestingly, all perceptions of cognitive function (attention, aspects of memory and planning) changed

significantly across the semester while performance on the Stroop tasks did not change. No significant differences were observed between groups or genders suggesting similar perceptions of cognitive stress across the semester. Although some studies have reported changes in cognitive function in athletes showing negative signs of adaptation (Reitjens et al., 2005; Nederhoff et al., 2007; Hynynen et al., 2008; Dupuy et al., 2010), no research has addressed natural fluctuations of perceptions of cognitive deficits or objective changes in cognition across a semester in student-athletes. Collegiate athletes report their sport involvement as a barrier to academic achievement and experience the negative stigma associated with being a student-athlete from professors, teaching assistants and their fellow students (Simons, 2007; Hood, 1992). However, our data suggest that participating in their sport (in this case rowing) is not affecting their cognition differently that non-athlete students across a season. Additionally, although there is some evidence to support a gender differences in impulse control and Stroop performance favoring women (Else-Quest, 2006; Janssen, 2009; Spinella, 2005;Mekarski, 1996), no gender differences in perception or Stroop performance were observed replicating other previous research (Daniel, 2000; MacLeod, 1992).

Changes in mood states over the course of a competitive season were similar to previous research demonstrating an inversion of the healthy mood state profile at peak training compared to baseline followed by a recovery after the season (O'Connor; Raglin; Morgan). The POMS data revealed that contrary to previous research, no gender differences in any mood state were present in our sample. Other research in female college athletes reported significantly higher TMD (TMD = 170) compared to a female college control (TMD = 110) group only at peak training (O'Connor et al., 1989). We did not observe any group differences in TMD values though they were similar to those reported in the previous study (TMD female controls = 131.21)

(TMD female athletes = 161.43). Additionally, it has been reported that while negative mood states respond to increases in training loads across the season in a similar dose-response manner for female and male student-athletes', females have significantly higher tension across the season (Raglin et al., 1991). Our data do not support this finding. In the college population at large, symptoms of depression are common and more frequent in women (Wright, 2004). Recently a nationwide survey collecting data on the presence of psychiatric illnesses and symptoms was sent to over 1,000 Universities (Tupler et al., 2015). Strikingly, nearly 75% of the 29,000 participants reported elevated depressive symptoms and a greater percentage of women endorsed these symptoms (Tupler, 2015). The POMS mood state differs from a more stable measurement of depressive symptoms and this may be, in part, why no gender differences were observed in depression scores at any time point during the study.

One variable that may contribute to the lack of gender differences in these comparisons is the high amount of physical activity across groups. Although perceptions of exercise load differed across the control and athlete groups, all participants were highly physically active. Our control participants reported engaging in at least 15-minutes of strenuous exercise approximately four times a week, at least 15-minutes of moderate exercise four times a week and at least 15-minutes of mild exercise six times each week equaling approximately 74 metabolic equivalents (METs) per week. This value is higher than what has been reported in a college population (men = 51. 2, women = 49.9) (Joseph, 2014). The similar amounts of physical activity in female and male control groups are interesting for a number of reasons. First, objective and self-report physical activity levels during adolescence and college decrease to a greater degree in women (Troiano et al., 2007; Kimm et al., 2002). Kimm and colleagues (2002) reported an 83% decrease in self-reported leisure time physical activity from the ages of 10-19 in girls and women. Second,

decreases in physical activity and depressive symptoms are related in college women. In one study, women were less likely to meet the national recommendations of vigorous physical activity and more likely to report poor mental health and higher PSS (VanKim et al., 2013). In a group of young adolescent girls, decreases in self-reported physical activity were negatively associated with depressive symptoms over a two-year (Motl, 2004) and three-year period (Raudsepp, 2012). Even though female control participants reported less leisure time physical activity compared to both athlete groups at baseline, they were still engaging in recommended levels of physical activity (Ehrman, 2012) and thus they may have been protected from some of the mental health discrepancies related to decreased physical activity that has been observed in previous research.

This study was limited in a number of important ways. First, only eight male controls and 15 female controls were enrolled in the study. Controls were recruited via flyers posted around campus and although efforts were made to contact more students, we were unsuccessful and thus comparisons in these groups should be considered with caution. Next, no objective data were collected to quantify physical activity or sleep in an effort to limit the already onerous time commitments of this busy college sample of men and women. These self-report measures are at risk for reporting bias. Finally, while the Stroop task was selected because it is an executive function task sensitive to both acute and chronic exercise and fatigue, it may not have been challenging or sensitive enough to elicit fatigue-related changes in performance in this particular highly active and academically strong sample..

This study demonstrated that although numerous measurements of well-being fluctuate over the course of a semester, few differences between student-athletes and physically active males and females were identified. All students showed increases in perceived cognitive deficits

yet maintained their Stroop performance suggesting a degree of cognitive resiliency. Additionally, while female athletes reported higher fatigue than female controls, few other gender differences were observed. This is likely explained by the high amount of physical activity performed by all participants and the nature of their extensive involvement in school and extracurricular activities. Future research should include larger sample sizes and sedentary control groups to further assess the influence of physical activity on these variables over the course of a semester.

References

Alexander, J. L. (2007). Quest for timely detection and treatment of women with depression. *Journal of Managed Care Pharmacy: JMCP*, *13*(9 Suppl A), S3–11.

Allison, M. T. (1991). Role conflict and the female athlete: Preoccupations with little grounding. *Journal of Applied Sport Psychology*, *3*(1), 49–60.

Aries, E., McCarthy, D., Salovey, P., & Banaji, M. R. (2004). A Comparison of Athletes and Non-Athletes at Highly Selective Colleges: Academic Performance and Personal Development. *Research in Higher Education*, *45*(6), 577–602.

Baddeley, A. (1996). Exploring the Central Executive. *The Quarterly Journal of Experimental Psychology Section A*, *49*(1), 5–28.

Badzakova-Trajkov, G., Barnett, K. J., Waldie, K. E., & Kirk, I. J. (2009). An ERP investigation of the Stroop task: The role of the cingulate in attentional allocation and conflict resolution. *Brain Research*, *1253*, 139–148.

Baghurst, T., & Kelley, B. C. (2014). An examination of stress in college students over the course of a semester. *Health Promotion Practice*, *15*(3), 438–447.

Bailey, C. M., Samples, H. L., Broshek, D. K., Freeman, J. R., & Barth, J. T. (2010). The Relationship Between Psychological Distress and Baseline Sports-Related Concussion Testing. *Clinical Journal of Sport Medicine*, *20*(4), 272–277.

Banich, M. T. (2009). Executive Function The Search for an Integrated Account. *Current Directions in Psychological Science*, *18*(2), 89–94.

Barwick, F., Arnett, P., & Slobounov, S. (2012). EEG correlates of fatigue during administration of a neuropsychological test battery. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, *123*(2), 278–284.

Başar, E. (2008). Oscillations in "brain-body-mind"--a holistic view including the autonomous system. *Brain Research*, *1235*, 2–11.

Belanger, H. G., & Cimino, C. R. (2002). The lateralized Stroop: A meta-analysis and its implications for models of semantic processing. *Brain and Language*, *83*(3), 384–402.

Benwell, N. M., Sacco, P., Hammond, G. R., Byrnes, M. L., Mastaglia, F. L., & Thickbroom, G. W. (2006). Short-interval cortical inhibition and corticomotor excitability with fatiguing hand exercise: a central adaptation to fatigue? *Experimental Brain Research. Experimentelle Hirnforschung. Expérimentation Cérébrale*, *170*(2), 191–198.

Bertolucci, P. H. F., & Oliveira, F. F. de. (2013). Cognitive Impairment in Fibromyalgia. *Current Pain and Headache Reports*, *17*(7), 1–9.

Blinde, E. M., Taub, D. E., & Han, L. (1993). Sport Participation and Women's Personal Empowerment: Experiences of the College Athlete. *Journal of Sport & Social Issues*, *17*(1), 47–60.

Boenke, L. T., Ohl, F. W., Nikolaev, A. R., Lachmann, T., & Leeuwen, C. van. (2009). Different time courses of Stroop and Garner effects in perception--an event-related potentials study. *NeuroImage*, *45*(4), 1272–1288.

Boksem, M. A. S., Meijman, T. F., & Lorist, M. M. (2005). Effects of mental fatigue on attention: an ERP study. *Brain Research. Cognitive Brain Research*, *25*(1), 107–116.

Brouwer, H., Fitz, H., & Hoeks, J. (2012). Getting real about Semantic Illusions: Rethinking the functional role of the P600 in language comprehension. *Brain Research*, *1446*, 127–143.

Brown, C., Glastetter-Fender, C., & Shelton, M. (2000). Psychosocial Identity and Career Control in College Student-Athletes. *Journal of Vocational Behavior*, *56*(1), 53–62.

Budgett, R., Newsholme, E., Lehmann, M., Sharp, C., Jones, D., Jones, T., … White, P. (2000).

Redefining the overtraining syndrome as the unexplained underperformance syndrome. *British Journal of Sports Medicine*, *34*(1), 67–68.

Cannon, R., Congedo, M., Lubar, J., & Hutchens, T. (2009). Differentiating a network of executive attention: LORETA neurofeedback in anterior cingulate and dorsolateral prefrontal cortices. *The International Journal of Neuroscience*, *119*(3), 404–441.

Carpenter, P. A., Just, M. A., & Reichle, E. D. (2000). Working memory and executive function: evidence from neuroimaging. *Current Opinion in Neurobiology*, *10*(2), 195–199.

Cevada, T., Vasques, P. E., Moraes, H., & Deslandes, A. (2014). Salivary cortisol levels in athletes and nonathletes: a systematic review. *Hormone and Metabolic Research = Hormon- Und Stoffwechselforschung = Hormones et Metabolisme*, *46*(13), 905–910.

Chang, Y.-K., Tsai, C.-L., Huang, C.-C., Wang, C.-C., & Chu, I.-H. (2013). Effects of acute resistance exercise on cognition in late middle-aged adults: General or specific cognitive improvement? *Journal of Science and Medicine in Sport / Sports Medicine Australia*.

Chaudhuri, A., & Behan, P. O. (2004). Fatigue in neurological disorders. *The Lancet*, *363*(9413), 978–988.

Chen, R., Lozano, A. M., & Ashby, P. (1999). Mechanism of the silent period following transcranial magnetic stimulation. Evidence from epidural recordings. *Experimental Brain Research. Experimentelle Hirnforschung. Expérimentation Cérébrale*, *128*(4), 539–542.

Cohen, S., & Janicki-Deverts, D. (2012). Who's Stressed? Distributions of Psychological Stress in the United States in Probability Samples from 1983, 2006, and 20091. *Journal of Applied Social Psychology*, *42*(6), 1320–1334.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*(4), 385–396.

Colcombe, S. J., Kramer, A. F., Erickson, K. I., Scalf, P., McAuley, E., Cohen, N. J., … Elavsky, S. (2004). Cardiovascular fitness, cortical plasticity, and aging. *Proceedings of the National Academy of Sciences of the United States of America*, *101*(9), 3316–3321.

Colcombe, S., & Kramer, A. F. (2003). Fitness effects on the cognitive function of older adults: a meta-analytic study. *Psychological Science*, *14*(2), 125–130.

Collette, F., & Van der Linden, M. (2002). Brain imaging of the central executive component of working memory. *Neuroscience & Biobehavioral Reviews*, *26*(2), 105–125.

Cook, D. B., O'Connor, P. J., Lange, G., & Steffener, J. (2007). Functional neuroimaging correlates of mental fatigue induced by cognition among chronic fatigue syndrome patients and controls. *NeuroImage*, *36*(1), 108–122.

Costill, D. L., Flynn, M. G., Kirwan, J. P., Houmard, J. A., Mitchell, J. B., Thomas, R., & Park, S. H. (1988). Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Medicine and Science in Sports and Exercise*, *20*(3), 249–254.

Coutts, A. J., Wallace, L. K., & Slattery, K. M. (2007). Monitoring changes in performance, physiology, biochemistry, and psychology during overreaching and recovery in triathletes. *International Journal of Sports Medicine*, *28*(2), 125–134.

Covassin, T., Elbin, R. J., Larson, E., & Kontos, A. P. (2012). Sex and Age Differences in Depression and Baseline Sport-Related Concussion Neurocognitive Performance and Symptoms. *Clinical Journal of Sport Medicine*, *22*(2), 98–104.

Crabbe, J. B., & Dishman, R. K. (2004a). Brain electrocortical activity during and after exercise: a quantitative synthesis. *Psychophysiology*, *41*(4), 563–574.

Crowley, K. E., & Colrain, I. M. (2004). A review of the evidence for P2 being an independent

component process: age, sleep and modality. *Clinical Neurophysiology*, *115*(4), 732–744.
Dale, A. M., Liu, A. K., Fischl, B. R., Buckner, R. L., Belliveau, J. W., Lewine, J. D., & Halgren, E. (2000). Dynamic Statistical Parametric Mapping: Combining fMRI and MEG for High-Resolution Imaging of Cortical Activity. *Neuron*, *26*(1), 55–67.
Daniel, D. B., Pelotte, M., & Lewis, J. (2000). Lack of sex differences on the Stroop Color-Word Test across three age groups. *Perceptual and Motor Skills*, *90*(2), 483–484.
DeLuca, J. (Ed.). (2005). *Fatigue as a window to the brain*. MIT Press.
Desertrain, G. S., & Weiss, M. R. (1988). Being female and athletic: A cause for conflict? *Sex Roles*, *18*(9-10), 567–582.
Dishman, R. K., Berthoud, H.-R., Booth, F. W., Cotman, C. W., Edgerton, V. R., Fleshner, M. R., … Zigmond, M. J. (2006). Neurobiology of Exercise. *Obesity*, *14*(3), 345–356.
Donohue, B., Covassin, T., Lancer, K., Dickens, Y., Miller, A., Hash, A., & Genet, J. (2004). Examination of Psychiatric Symptoms in Student Athletes. *The Journal of General Psychology*, *131*(1), 29–35.
Duclos, M., Guinot, M., & Le Bouc, Y. (2007). Cortisol and GH: odd and controversial ideas. *Applied Physiology, Nutrition, and Metabolism*, *32*(5), 895–903.
Duncan, J., & Owen, A. M. (2000). Common regions of the human frontal lobe recruited by diverse cognitive demands. *Trends in Neurosciences*, *23*(10), 475–483.
Dupuy, O., Renaud, M., Bherer, L., & Bosquet, L. (2010). Effect of functional overreaching on executive functions. *International Journal of Sports Medicine*, *31*(9), 617–623.
El Ansari, W., Oskrochi, R., & Haghgoo, G. (2014a). Are students' symptoms and health complaints associated with perceived stress at university? Perspectives from the United Kingdom and Egypt. *International Journal of Environmental Research and Public Health*, *11*(10).
Else-Quest, N. M., Hyde, J. S., Goldsmith, H. H., & Van Hulle, C. A. (2006). Gender differences in temperament: a meta-analysis. *Psychological Bulletin*, *132*(1).
Faber, L. G., Maurits, N. M., & Lorist, M. M. (2012). Mental fatigue affects visual selective attention. *PloS One*, *7*(10), e48073.
Fallah, N., Hsu, C. L., Bolandzadeh, N., Davis, J., Beattie, B. L., Graf, P., & Liu-Ambrose, T. (2013a). A multistate model of cognitive dynamics in relation to resistance training: the contribution of baseline function. *Annals of Epidemiology*, *23*(8), 463–468.
Fedewa, A. L., & Ahn, S. (2011). The Effects of Physical Activity and Physical Fitness on Children's Achievement and Cognitive Outcomes. *Research Quarterly for Exercise and Sport*, *82*(3), 521–535.
Filaire, E., Bernain, X., Sagnol, M., & Lac, G. (2001). Preliminary results on mood state, salivary testosterone:cortisol ratio and team performance in a professional soccer team. *European Journal of Applied Physiology*, *86*(2), 179–184.
Flor-Henry, P., Lind, J. C., & Koles, Z. J. (2010). EEG source analysis of chronic fatigue syndrome. *Psychiatry Research*, *181*(2), 155–164.
Flynn, M., Pizza, F., Boone, J., Andres, F., Michaud, T., & Rodriguez-Zayas, J. (2008a). Indices of Training Stress During Competitive Running and Swimming Seasons. *International Journal of Sports Medicine*, *15*(01), 21–26.
Fragala, M. S., PhD, W. J. K., Denegar, C. R., Maresh, C. M., Mastro, A. M., & Volek, J. S. (2011). Neuroendocrine-Immune Interactions and Responses to Exercise. *Sports Medicine*, *41*(8), 621–639.
Frisch, S., Kotz, S. A., von Cramon, D. Y., & Friederici, A. D. (2003). Why the P600 is not just

a P300: the role of the basal ganglia. *Clinical Neurophysiology*, *114*(2), 336–340.
Funahashi, S. (2001). Neuronal mechanisms of executive control by the prefrontal cortex. *Neuroscience Research*, *39*(2), 147–165.
Furr, S. R., Westefeld, J. S., McConnell, G. N., & Marshall, J. (2001a). Suicide and depression among college students: A decade later. *Professional Psychology: Research and Practice*, *32*(1), 97–100.
Gambetta-Tessini, K., Marino, R., Morgan, M., Evans, W., & Anderson, V. (2013). Stress and health-promoting attributes in Australian, New Zealand, and Chilean dental students. *Journal of Dental Education*, *77*(6), 801–809.
Garavan, H., Ross, T. J., Murphy, K., Roche, R. A. P., & Stein, E. A. (2002a). Dissociable Executive Functions in the Dynamic Control of Behavior: Inhibition, Error Detection, and Correction. *NeuroImage*, *17*(4), 1820–1829.
Garber, C. E., Blissmer, B., Deschenes, M. R., Franklin, B. A., Lamonte, M. J., Lee, I.-M., … Swain, D. P. (2011). American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Medicine and Science in Sports and Exercise*, *43*(7), 1334–1359.
Gaston-Gayles, J. L. (2004). Examining Academic and Athletic Motivation Among Student Athletes at a Division I University. *Journal of College Student Development*, *45*(1), 75–83.
Gayles, J. G., & Hu, S. (2009). Athletes as students: Ensuring positive cognitive and affective outcomes. *New Directions for Higher Education*,*2009*(148), 101-107.
Godin, G., & Shephard, R. J. (1985). A simple method to assess exercise behavior in the community. *Canadian Journal of Applied Sport Sciences. Journal Canadien Des Sciences Appliquees Au Sport*, *10*(3), 141–146.
Gouarné, C., Groussard, C., Gratas-Delamarche, A., Delamarche, P., & Duclos, M. (2005). Overnight urinary cortisol and cortisone add new insights into adaptation to training. *Medicine and Science in Sports and Exercise*, *37*(7), 1157–1167.
Haenschel, C., Baldeweg, T., Croft, R. J., Whittington, M., & Gruzelier, J. (2000). Gamma and beta frequency oscillations in response to novel auditory stimuli: A comparison of human electroencephalogram (EEG) data with in vitro models. *Proceedings of the National Academy of Sciences of the United States of America*, *97*(13), 7645–7650.
Hahne, A., & Friederici, A. D. (1999). Electrophysiological Evidence for Two Steps in Syntactic Analysis: Early Automatic and Late Controlled Processes. *Journal of Cognitive Neuroscience*, *11*(2), 194–205.
Häkkinen, A., Pakarinen, A., Hannonen, P., Kautiainen, H., Nyman, K., Kraemer, W. J., & Häkkinen, K. (2013). Effects of prolonged combined strength and endurance training on physical fitness, body composition and serum hormones in women with rheumatoid arthritis and in healthy controls. *Clinical and Experimental Rheumatology*, *23*(4), 505–512.
Hallez, H., Vanrumste, B., Grech, R., Muscat, J., De Clercq, W., Vergult, A., … Lemahieu, I. (2007). Review on solving the forward problem in EEG source analysis. *Journal of NeuroEngineering and Rehabilitation*, *4*(1), 46.
Halson, S. L., Bridge, M. W., Meeusen, R., Busschaert, B., Gleeson, M., Jones, D. A., & Jeukendrup, A. E. (2002). Time course of performance changes and fatigue markers during intensified training in trained cyclists. *Journal of Applied Physiology*, *93*(3), 947–

956.
Halson, S. L., & Jeukendrup, A. E. (2004). Does overtraining exist? An analysis of overreaching and overtraining research. *Sports Medicine (Auckland, N.Z.)*, *34*(14), 967–981.
Hanks, R. A., Rapport, L. J., Millis, S. R., & Deshpande, S. A. (1999). Measures of executive functioning as predictors of functional ability and social integration in a rehabilitation sample. *Archives of Physical Medicine and Rehabilitation*, *80*(9), 1030–1037.
Hanslmayr, S., Pastötter, B., Bäuml, K.-H., Gruber, S., Wimber, M., & Klimesch, W. (2007). The Electrophysiological Dynamics of Interference during the Stroop Task. *Journal of Cognitive Neuroscience*, *20*(2), 215–225.
Harrison, B. J., Shaw, M., Yücel, M., Purcell, R., Brewer, W. J., Strother, S. C., … Pantelis, C. (2005). Functional connectivity during Stroop task performance. *NeuroImage*, *24*(1), 181–191.
Hasson, D., & Arnetz, B. B. (2005). Validation and Findings Comparing VAS vs. Likert Scales for Psychosocial Measurements. *International Electronic Journal of Health Education*, *8*, 178–192.
Hausswirth, C., Louis, J., Aubry, A., Bonnet, G., Duffield, R., & LE Meur, Y. (2014). Evidence of disturbed sleep and increased illness in overreached endurance athletes. *Medicine and Science in Sports and Exercise*, *46*(5), 1036–1045.
Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, *25*(1), 1–35. 9
Hillman, C. H., Kramer, A. F., Belopolsky, A. V., & Smith, D. P. (2006). A cross-sectional examination of age and physical activity on performance and event-related brain potentials in a task switching paradigm. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *59*(1), 30–39.
Hillman, C. H., Motl, R. W., Pontifex, M. B., Posthuma, D., Stubbe, J. H., Boomsma, D. I., & de Geus, E. J. C. (2006). Physical activity and cognitive function in a cross-section of younger and older community-dwelling individuals. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association*, *25*(6), 678–687.
Hillyard, S. A., Teder-Sälejärvi, W. A., & Münte, T. F. (1998). Temporal dynamics of early perceptual processing. *Current Opinion in Neurobiology*, *8*(2), 202–210.
Hilty, L., Langer, N., Pascual-Marqui, R., Boutellier, U., & Lutz, K. (2011). Fatigue-induced increase in intracortical communication between mid/anterior insular and motor cortex during cycling exercise. *The European Journal of Neuroscience*, *34*(12), 2035–2042.
Hindin, S. B., & Zelinski, E. M. (2012). Extended Practice and Aerobic Exercise Interventions Benefit Untrained Cognitive Outcomes in Older Adults: A Meta-Analysis. *Journal of the American Geriatrics Society*, *60*(1), 136–141.
Hoffman, J. R., Bar-Eli, M., & Tenenbaum, G. (1999). An examination of mood changes and performance in a professional basketball team. *The Journal of Sports Medicine and Physical Fitness*, *39*(1), 74–79.
Hood, A. B., Craig, A. F., & Ferguson, B. W. (1992). The impact of athletics, part-time employment, and other activities on academic achievement. *Journal of College Student Development*, *33*(5), 447–453.
Hooper, S. L., MacKinnon, L. T., Gordon, R. D., & Bachmann, A. W. (1993). Hormonal responses of elite swimmers to overtraining. *Medicine and Science in Sports and*

Exercise, *25*(6), 741–747.

Howell, A. J., Jahrig, J. C., & Powell, R. A. (2004). Sleep quality, sleep propensity and academic performance. *Perceptual and Motor Skills*, *99*(2), 525–535.

Humphrey, J. H., Yow, D. A., & Bowden, W. W. (2000). *Stress in College Athletics: Causes, Consequences, Coping.* Haworth Half-Court Press, Inc., 10 Alice Street, Binghamton, NY 13904-1580 (paperback: ISBN-0-7890-0935-8 ($24.95); clothbound: ISBN-0-7890-0934-X, $59.95). Tel: 800-429-6784 (Toll Free); Fax: 800-895-0582 (Toll Free); Web site:

Hynynen, E., Uusitalo, A., Konttinen, N., & Rusko, H. (2008). Cardiac autonomic responses to standing up and cognitive task in overtrained athletes. *International Journal of Sports Medicine*, *29*(7), 552–558.

Ibanez, A., Melloni, M., Huepe, D., Helgiu, E., Rivera-Rei, A., Canales-Johnson, A., … Moya, A. (2012). What event-related potentials (ERPs) bring to social neuroscience? *Social Neuroscience*, *7*(6), 632–649.

Iellamo, F., Legramante, J. M., Pigozzi, F., Spataro, A., Norbiato, G., Lucini, D., & Pagani, M. (2002). Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. *Circulation*, *105*(23), 2719–2724.

Janssen, G. T. L., De Mey, H. R. A., & Egger, J. I. M. (2009). Executive functioning in college students: evaluation of the Dutch executive function index (EFI-NL). *The International Journal of Neuroscience*, *119*(6).

Johns, M. W. (1991). A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*, *14*(6), 540–545.

Jongen, P. J., Ter Horst, A. T., & Brands, A. M. (2012). Cognitive impairment in multiple sclerosis. *Minerva Medica*, *103*(2), 73–96.

Joseph, R. P., Royse, K. E., Benitez, T. J., & Pekmezi, D. W. (2014). Physical activity and quality of life among university students: exploring self-efficacy, self-esteem, and affect as potential mediators. *Quality of Life Research : An International Journal of Quality of Life Aspects of Treatment, Care and Rehabilitation*, *23*(2), 659–667.

Kamijo, K., & Takeda, Y. (2010). Regular physical activity improves executive function during task switching in young adults. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *75*(3), 304–311.

Kato, Y., Endo, H., & Kizuka, T. (2009a). Mental fatigue and impaired response processes: event-related brain potentials in a Go/NoGo task. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *72*(2), 204–211.

Kellmann, M. (2010). Preventing overtraining in athletes in high-intensity sports and stress/recovery monitoring. *Scandinavian Journal of Medicine & Science in Sports*, *20 Suppl 2*, 95–102.

Kellmann, M., & Gunther, K. D. (2000). Changes in stress and recovery in elite rowers during preparation for the Olympic Games. *Medicine and Science in Sports and Exercise*, *32*(3), 676–683.

Kimball, A., & Freysinger, V. J. (2003). Leisure, Stress, and Coping: The Sport Participation of Collegiate Student-Athletes. *Leisure Sciences*, *25*(2-3), 115–141.

Kimm, S. Y. S., Glynn, N. W., Kriska, A. M., Barton, B. A., Kronsberg, S. S., Daniels, S. R., … Liu, K. (2002). Decline in physical activity in black girls and white girls during adolescence. *The New England Journal of Medicine*, *347*(10), 709–715.

Kindt, M., Bierman, D., & Brosschot, J. F. (1996). Stroop versus Stroop: Comparison of a card format and a single-trial format of the standard color-word Stroop task and the emotional Stroop task. *Personality and Individual Differences*, *21*(5), 653–661.

Kirwan, J. P., Costill, D. L., Flynn, M. G., Mitchell, J. B., Fink, W. J., Neufer, P. D., & Houmard, J. A. (1988). Physiological responses to successive days of intense training in competitive swimmers. *Medicine and Science in Sports and Exercise*, *20*(3), 255–259.

Klimesch, W. (1999). EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis. *Brain Research. Brain Research Reviews*, *29*(2-3), 169–195.

Knoop, H., Prins, J. B., Moss-Morris, R., & Bleijenberg, G. (2010). The central role of cognitive processes in the perpetuation of chronic fatigue syndrome. *Journal of Psychosomatic Research*, *68*(5), 489–494.

Kok, A. (1997). Event-related-potential (ERP) reflections of mental resourcês: a review and synthesis. *Biological Psychology*, *45*(1–3), 19–56.

Kuoppasalmi, K., Näveri, H., Härkönen, M., & Adlercreutz, H. (1980). Plasma cortisol, androstenedione, testosterone and luteinizing hormone in running exercise of different intensities. *Scandinavian Journal of Clinical & Laboratory Investigation*, *40*(5), 403–409.

Kutas, M., & Federmeier, K. D. (2011). Thirty Years and Counting: Finding Meaning in the N400 Component of the Event-Related Brain Potential (ERP). *Annual Review of Psychology*, *62*(1), 621–647.

Leeder, J., Glaister, M., Pizzoferro, K., Dawson, J., & Pedlar, C. (2012). Sleep duration and quality in elite athletes measured using wristwatch actigraphy. *Journal of Sports Sciences*, *30*(6), 541–545.

Lee, R. S. C., Hermens, D. F., Porter, M. A., & Redoblado-Hodge, M. A. (2012). A meta-analysis of cognitive deficits in first-episode Major Depressive Disorder. *Journal of Affective Disorders*, *140*(2), 113–124.

Lehmann, M., Dickhuth, H., Gendrisch, G., Lazar, W., Thum, M., Kaminski, R., … Keul, J. (2008). Training - Overtraining. A Prospective, Experimental Study with Experienced Middle- and Long-Distance Runners*. *International Journal of Sports Medicine*, *12*(05), 444–452.

Lehmann, M., Foster, C., & Keul, J. (1993). Overtraining in endurance athletes: A brief review. *Medicine & Science in Sports & Exercise*, *25*(7), 854–862.

Leung, H.-C., Skudlarski, P., Gatenby, J. C., Peterson, B. S., & Gore, J. C. (2000). An Event-related Functional MRI Study of the Stroop Color Word Interference Task. *Cerebral Cortex*, *10*(6), 552–560.

Lewandowski, K. E., Cohen, B. M., Keshavan, M. S., Sperry, S. H., & Öngür, D. (2013). Neuropsychological functioning predicts community outcomes in affective and non-affective psychoses: A 6-month follow-up. *Schizophrenia Research*.

Lindsey, B. J., Fabiano, P., & Stark, C. (2009). The Prevalence and Correlates of Depression among College Students. *College Student Journal*, *43*(4), 999–1014.

Liotti, M., Woldorff, M. G., Perez III, R., & Mayberg, H. S. (2000). An ERP study of the temporal course of the Stroop color-word interference effect. *Neuropsychologia*, *38*(5), 701–711.

Lori, S., Portaccio, E., Zipoli, V., Giannini, M., Scarpelli, S., Goretti, B., & Amato, M. P. (2011). Cognitive impairment and event-related potentials in paediatric multiple sclerosis: 2-year

study. *Neurological Sciences: Official Journal of the Italian Neurological Society and of the Italian Society of Clinical Neurophysiology*, *32*(6), 1043–1046.

Luger, A., Deuster, P. A., Kyle, S. B., Gallucci, W. T., Montgomery, L. C., Gold, P. W., … Chrousos, G. P. (1987). Acute Hypothalamic–Pituitary–Adrenal Responses to the Stress of Treadmill Exercise. *New England Journal of Medicine*, *316*(21), 1309–1315.

Lund, H. G., Reider, B. D., Whiting, A. B., & Prichard, J. R. (2010). Sleep patterns and predictors of disturbed sleep in a large population of college students. *The Journal of Adolescent Health : Official Publication of the Society for Adolescent Medicine*, *46*(2), 124–132.

MacLeod, C. M. (1992). The Stroop task: The "gold standard" of attentional measures. *Journal of Experimental Psychology: General*, *121*(1), 12–14.

Maestripieri, D., Baran, N. M., Sapienza, P., & Zingales, L. (2010). Between- and within-sex variation in hormonal responses to psychological stress in a large sample of college students. *Stress (Amsterdam, Netherlands)*, *13*(5), 413–424.

Mah, C. D., Mah, K. E., Kezirian, E. J., & Dement, W. C. (2011). The effects of sleep extension on the athletic performance of collegiate basketball players. *Sleep*, *34*(7), 943–950.

Main, L. C., Dawson, B., Grove, J. R., Landers, G. J., & Goodman, C. (2009). Impact of training on changes in perceived stress and cytokine production. *Research in Sports Medicine (Print)*, *17*(2), 121–132.

Maluf, S. A. A.-M. (n.d.).

Markela-Lerenc, J., Ille, N., Kaiser, S., Fiedler, P., Mundt, C., & Weisbrod, M. (2004). Prefrontal-cingulate activation during executive control: which comes first? *Cognitive Brain Research*, *18*(3), 278–287.

Marrie, R. A., Miller, D. M., Chelune, G. J., & Cohen, J. A. (2003). Validity and reliability of the MSQLI in cognitively impaired patients with multiple sclerosis. *Multiple Sclerosis (Houndmills, Basingstoke, England)*, *9*(6), 621–626.

Martin, D. T., Andersen, M. B., & Gates, W. (2000). Using Profile of Mood States (POMS) to monitor high-intensity training in cyclists: group versus case studies. *Sport Psychologist*, *14*(2), 138–156.

Mastorakos, G., & Pavlatou, M. (2005). Exercise as a Stress Model and the Interplay Between the Hypothalamus-pituitary-adrenal and the Hypothalamus-pituitary-thyroid Axes. *Hormone and Metabolic Research*, *37*(9), 577–584.

McNair, D., Lorr, M., & Droppleman, L. (1989). Profile of Mood States (POMS). Retrieved from http://ubir.buffalo.edu/xmlui/handle/10477/1888

McSweeny, A. J., Grant, I., Heaton, R. K., Prigatano, G. P., & Adams, K. M. (1985). Relationship of neuropsychological status to everyday functioning in healthy and chronically Ill persons. *Journal of Clinical and Experimental Neuropsychology*, *7*(3),

Mecklinger, A., & Ullsperger, P. (1995). The P300 to novel and target events: a spatiotemporal dipole model analysis. *Neuroreport*, *7*(1), 241–245.

Meeusen, R., Duclos, M., Foster, C., Fry, A., Gleeson, M., Nieman, D., … American College of Sports Medicine. (2013). Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. *Medicine and Science in Sports and Exercise*, *45*(1), 186–205. h

Meeusen, R., Nederhof, E., Buyse, L., Roelands, B., de Schutter, G., & Piacentini, M. F. (2010). Diagnosing overtraining in athletes using the two-bout exercise protocol. *British Journal*

of Sports Medicine, *44*(9), 642–648.
Meeusen, R., Piacentini, M. F., Busschaert, B., Buyse, L., De Schutter, G., & Stray-Gundersen, J. (2004). Hormonal responses in athletes: the use of a two bout exercise protocol to detect subtle differences in (over)training status. *European Journal of Applied Physiology*, *91*(2-3), 140–146.
Meeusen, R., Watson, P., Hasegawa, H., Roelands, B., & Piacentini, M. F. (2007). Brain neurotransmitters in fatigue and overtraining. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquée, Nutrition et Métabolisme*, *32*(5), 857–864.
Mekarski, J. E., Cutmore, T. R. H., & Suboski, W. (1996). Gender differences during processing of the Stroop Task. *Perceptual and Motor Skills*, *83*(2), 563–568.
Melendez, M. C. (2006). The influence of athletic participation on the college adjustment of freshman and sophomore student athletes. *Journal of College Student Retention: Research, Theory and Practice*, *8*(1), 39–55.
Michiels, V., & Cluydts, R. (2001a). Neuropsychological functioning in chronic fatigue syndrome: a review. *Acta Psychiatrica Scandinavica*, *103*(2), 84–93.
Miller, J. L., & Levy, G. D. (1996). Gender role conflict, gender-typed characteristics, self-concepts, and sport socialization in female athletes and nonathletes. *Sex Roles*, *35*(1-2), 111–122.
Mj, S., E, H., R, B., Me, S., & H, A. (2001). Perceived cognitive deficits, emotional distress and disability following whiplash injury. *Pain Research & Management : The Journal of the Canadian Pain Society = Journal de La Societe Canadienne Pour Le Traitement de La Douleur*, *7*(3), 120–126.
Moo-Estrella, J., Perez-Benitez, H., Solis-Rodriguez, F., & Arankowsky-Sandoval, G. (2005). Evaluation of depressive symptoms and sleep alterations in college students. *Archives of Medical Research*, *36*(4), 393–398.
Moraes, H., Deslandes, A., Silveira, H., Ribeiro, P., Cagy, M., Piedade, R., … Laks, J. (2011). The effect of acute effort on EEG in healthy young and elderly subjects. *European Journal of Applied Physiology*, *111*(1), 67–75.
Morgan, W. P., Brown, D. R., Raglin, J. S., O'Connor, P. J., & Ellickson, K. A. (1987). Psychological monitoring of overtraining and staleness. *British Journal of Sports Medicine*, *21*(3), 107–114.
Morgan, W. P., Costill, D. L., Flynn, M. G., Raglin, J. S., & O'Connor, P. J. (1988). Mood disturbance following increased training in swimmers. *Medicine and Science in Sports and Exercise*, *20*(4), 408–414.
Munce, S. E. P., & Stewart, D. E. (2007). Gender Differences in Depression and Chronic Pain Conditions in a National Epidemiologic Survey. *Psychosomatics*, *48*(5), 394–399.
NCAA Research Staff (2014, October, 22). Trends in graduation success rates and Federal graduation rates at NCAA Division I institutions. http://www.ncaa.org
Nederhof, E., Lemmink, K. A. P. M., Visscher, C., Meeusen, R., & Mulder, T. (2006). Psychomotor speed: possibly a new marker for overtraining syndrome. *Sports Medicine (Auckland, N.Z.)*, *36*(10), 817–828.
Nederhof, E., Lemmink, K., Zwerver, J., & Mulder, T. (2007). The effect of high load training on psychomotor speed. *International Journal of Sports Medicine*, *28*(7), 595–601.
Nederhof, E., Zwerver, J., Brink, M., Meeusen, R., & Lemmink, K. (2008). Different diagnostic tools in nonfunctional overreaching. *International Journal of Sports Medicine*, *29*(7), 590–597.

Neu, D., Kajosch, H., Peigneux, P., Verbanck, P., Linkowski, P., & Le Bon, O. (2011). Cognitive impairment in fatigue and sleepiness associated conditions. *Psychiatry Research*, *189*(1), 128–134.

Nieuwenhuis, S., Yeung, N., Wildenberg, W. van den, & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. *Cognitive, Affective, & Behavioral Neuroscience*, *3*(1), 17–26.

O'Connor, P. J., Morgan, W. P., Raglin, J. S., Barksdale, C. M., & Kalin, N. H. (1989). Mood state and salivary cortisol levels following overtraining in female swimmers. *Psychoneuroendocrinology*, *14*(4), 303–310.

Pardo, J. V., Pardo, P. J., Janer, K. W., & Raichle, M. E. (1990). The anterior cingulate cortex mediates processing selection in the Stroop attentional conflict paradigm. *Proceedings of the National Academy of Sciences*, *87*(1), 256–259.

Pascual-Marqui, R. D. (2002). Standardized low-resolution brain electromagnetic tomography (sLORETA): technical details. *Methods and Findings in Experimental and Clinical Pharmacology*, *24 Suppl D*, 5–12.

Piras, M. R., Magnano, I., Canu, E. D. G., Paulus, K. S., Satta, W. M., Soddu, A., … Aiello, I. (2003). Longitudinal study of cognitive dysfunction in multiple sclerosis: neuropsychological, neuroradiological, and neurophysiological findings. *Journal of Neurology, Neurosurgery, and Psychiatry*, *74*(7), 878–885.

Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology*, *118*(10), 2128–2148.

Polich, J., Moore, A. P., & Wiederhold, M. D. (1995). P300 assessment of chronic fatigue syndrome. *Journal of Clinical Neurophysiology: Official Publication of the American Electroencephalographic Society*, *12*(2), 186–191.

Pontifex, M. B., Hillman, C. H., Fernhall, B., Thompson, K. M., & Valentini, T. A. (2009). The effect of acute aerobic and resistance exercise on working memory. *Medicine and Science in Sports and Exercise*, *41*(4), 927–934.

Prasher, D., Smith, A., & Findley, L. (1990). Sensory and cognitive event-related potentials in myalgic encephalomyelitis. *Journal of Neurology, Neurosurgery, and Psychiatry*, *53*(3), 247–253.

Predovan, D., Fraser, S. A., Renaud, M., & Bherer, L. (2012). The effect of three months of aerobic training on stroop performance in older adults. *Journal of Aging Research*, *2012*, 269815.

Purvis, D., Gonsalves, S., & Deuster, P. A. (2010). Physiological and psychological fatigue in extreme conditions: overtraining and elite athletes. *PM & R: The Journal of Injury, Function, and Rehabilitation*, *2*(5), 442–450.

Raglin, J., Morgan, W., & O'Connor, P. (1991). Changes in Mood States during Training in Female and Male College Swimmers. *International Journal of Sports Medicine*, *12*(06), 585–589.

Raglin, J. S. (1990). Exercise and Mental Health. *Sports Medicine*, *9*(6), 323–329.

Rebai, M., Bernard, C., & Lannou, J. (1997). The Stroop's test evokes a negative brain potential, the N400. *The International Journal of Neuroscience*, *91*(1-2), 85–94.

Richard, S., & Aries, E. (1999). The Division III Student-Athlete: Academic Performance, Campus Involvement and Growth. *Journal of College Student Development*, *40*(3), 211–18.

Rietjens, G. J. W. M., Kuipers, H., Adam, J. J., Saris, W. H. M., van Breda, E., van Hamont, D., & Keizer, H. A. (2005). Physiological, Biochemical and Psychological Markers of Strenuous Training-Induced Fatigue. *International Journal of Sports Medicine*, *26*(01/02), 16–26.

Rivier, C., & Rivest, S. (1991). Effect of stress on the activity of the hypothalamic-pituitary-gonadal axis: peripheral and central mechanisms. *Biology of Reproduction*, *45*(4), 523–532.

Ryan, F. J. (1989). Participation in intercollegiate athletics: Affective outcomes. *Journal of College Student Development*, *30*(2), 122–128.

Sammer, G., Blecker, C., Gebhardt, H., Bischoff, M., Stark, R., Morgen, K., & Vaitl, D. (2007). Relationship between regional hemodynamic activity and simultaneously recorded EEG-theta associated with mental arithmetic-induced workload. *Human Brain Mapping*, *28*(8), 793–803.

Sansgiry, S. S., & Sail, K. (2006). Effect of Students' Perceptions of Course Load on Test Anxiety. *American Journal of Pharmaceutical Education*, *70*(2).

Schaul, N. (1998). The fundamental neural mechanisms of electroencephalography. *Electroencephalography and Clinical Neurophysiology*, *106*(2), 101–107.

Scheffers, M. K., Johnson, R., Jr, Grafman, J., Dale, J. K., & Straus, S. E. (1992). Attention and short-term memory in chronic fatigue syndrome patients: an event-related potential analysis. *Neurology*, *42*(9), 1667–1675.

Scisco, J. L., Leynes, P. A., & Kang, J. (2008). Cardiovascular fitness and executive control during task-switching: an ERP study. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *69*(1), 52–60.

Sherlin, L., Budzynski, T., Kogan Budzynski, H., Congedo, M., Fischer, M. E., & Buchwald, D. (2007). Low-resolution electromagnetic brain tomography (LORETA) of monozygotic twins discordant for chronic fatigue syndrome. *NeuroImage*, *34*(4), 1438–1442.

Silton, R. L., Heller, W., Towers, D. N., Engels, A. S., Spielberg, J. M., Edgar, J. C., … Miller, G. A. (2010). The time course of activity in dorsolateral prefrontal cortex and anterior cingulate cortex during top-down attentional control. *NeuroImage*, *50*(3), 1292–1302.

Simons, H. D., Bosworth, C., Fujita, S., & Jensen, M. (2007). The athlete stigma in higher education. *College Student Journal*, *41*(2), 251–273.

Singleton, R. A. J., & Wolfson, A. R. (2009). Alcohol consumption, sleep, and academic performance among college students. *Journal of Studies on Alcohol and Drugs*, *70*(3), 355–363.

Slivka, D. R., Hailes, W. S., Cuddy, J. S., & Ruby, B. C. (2010). Effects of 21 days of intensified training on markers of overtraining. *Journal of Strength and Conditioning Research / National Strength & Conditioning Association*, *24*(10), 2604–2612.

Smith, E. E., & Jonides, J. (1999). Storage and Executive Processes in the Frontal Lobes. *Science*, *283*(5408), 1657–1661.

Smith, P. J., Blumenthal, J. A., Hoffman, B. M., Cooper, H., Strauman, T. A., Welsh-Bohmer, K., … Sherwood, A. (2010). Aerobic Exercise and Neurocognitive Performance: A Meta-Analytic Review of Randomized Controlled Trials. *Psychosomatic Medicine*, *72*(3), 239–252.

Spinella, M. (2005). Self-rated executive function: development of the executive function index. *The International Journal of Neuroscience*, *115*(5), 649–667.

St Clair Gibson, A., Lambert, E. V., Rauch, L. H. G., Tucker, R., Baden, D. A., Foster, C., &

Noakes, T. D. (2006). The role of information processing between the brain and peripheral physiological systems in pacing and perception of effort. *Sports Medicine (Auckland, N.Z.)*, *36*(8), 705–722.

Stroth, S., Hille, K., Spitzer, M., & Reinhardt, R. (2009). Aerobic endurance exercise benefits memory and affect in young adults. *Neuropsychological Rehabilitation*, *19*(2), 223–243.

Sullivan, M., Edgley, K., & Dehoux, E. (1990). A survey of multiple sclerosis. Part I: perceived cognitive problems and compensatory strategy use., *4 SP - 99 EP - 105 JF - Canadian Journal of Rehabilitation M1 - Journal Article ER -*.

Tanaka, M., Shigihara, Y., Ishii, A., Funakura, M., Kanai, E., & Watanabe, Y. (2012). Effect of mental fatigue on the central nervous system: an electroencephalography study. *Behavioral and Brain Functions: BBF*, *8*, 48.

Terry, P. ., Lane, A. ., & Fogarty, G. . (2003a. Construct validity of the Profile of Mood States — Adolescents for use with adults. *Psychology of Sport and Exercise*, *4*(2), 125–139.

Themanson, J. R., Hillman, C. H., & Curtin, J. J. (2006). Age and physical activity influences on action monitoring during task switching. *Neurobiology of Aging*, *27*(9), 1335–1345.

Themanson, J. R., Pontifex, M. B., & Hillman, C. H. (2008). Fitness and action monitoring: Evidence for improved cognitive flexibility in young adults. *Neuroscience*, *157*(2), 319–328.

Tiersky, L. A., DeLuca, J., Hill, N., Dhar, S. K., Johnson, S. K., Lange, G., … Natelson, B. H. (2001). Longitudinal Assessment of Neuropsychological Functioning, Psychiatric Status, Functional Disability and Employment Status in Chronic Fatigue Syndrome. *Applied Neuropsychology*, *8*(1), 41–50.

Tomporowski, P. D., & Hatfield, B. D. (2005a). Effects of exercise on neurocognitive functions. *International Journal of Sport and Exercise Psychology*, *3*(3), 363–379.

Tonner, P. H., & Bein, B. (2006). Classic electroencephalographic parameters: Median frequency, spectral edge frequency etc. *Best Practice & Research Clinical Anaesthesiology*, *20*(1), 147–159.

Tremblay, M. S., Copeland, J. L., & Helder, W. V. (2005). Influence of exercise duration on post-exercise steroid hormone responses in trained males. *European Journal of Applied Physiology*, *94*(5-6), 505–513.

Troiano, R. P., Berrigan, D., Dodd, K. W., Masse, L. C., Tilert, T., & McDowell, M. (2008). Physical activity in the United States measured by accelerometer. *Medicine and Science in Sports and Exercise*, *40*(1), 181–188.

Tsai, L.-L., & Li, S.-P. (2004). Sleep patterns in college students: gender and grade differences. *Journal of Psychosomatic Research*, *56*(2), 231–237.

Tupler, L. A., Hong, J. Y., Gibori, R., Blitchington, T. F., & Krishnan, K. R. R. (2015). Suicidal ideation and sex differences in relation to 18 major psychiatric disorders in college and university students: anonymous web-based assessment. *The Journal of Nervous and Mental Disease*, *203*(4), 269–278.

Umbach, P. D., Palmer, M. M., Kuh, G. D., & Hannah, S. J. (2006). Intercollegiate Athletes and Effective Educational Practices: Winning Combination or Losing Effort? *Research in Higher Education*, *47*(6), 709–733.

Urhausen, A., Gabriel, H. H., Weiler, B., & Kindermann, W. (n.d.). Ergometric and Psychological Findings During Overtraining: A Long-Term Follow-Up Study in Endurance Athletes. *International Journal of Sports Medicine*, *19*(2), 114–120.

Urhausen, A., Kullmer, T., & Kindermann, W. (1987). A 7-week follow-up study of the

behaviour of testosterone and cortisol during the competition period in rowers. *European Journal of Applied Physiology and Occupational Physiology*, *56*(5), 528–533.

Uusitalo, A. L., Huttunen, P., Hanin, Y., Uusitalo, A. J., & Rusko, H. K. (1998). Hormonal responses to endurance training and overtraining in female athletes. *Clinical Journal of Sport Medicine : Official Journal of the Canadian Academy of Sport Medicine*, *8*(3), 178–186

Vankim, N. A., & Nelson, T. F. (2013). Vigorous physical activity, mental health, perceived stress, and socializing among college students. *American Journal of Health Promotion : AJHP*, *28*(1).

Verhaeghen, P., & De Meersman, L. (1998). Aging and the Stroop effect: A meta-analysis. *Psychology and Aging*, *13*(1), 120–126.

Vervoorn, C., Quist, A., Vermulst, L., Erich, W., de Vries, W., & Thijssen, J. (2008). The Behaviour of the Plasma Free Testosterone/Cortisol Ratio during a Season of Elite Rowing Training. *International Journal of Sports Medicine*, *12*(03), 257–263.

Vetter RE, S. M. (2010a). Correlations between injury, training intensity, and physical and mental exhaustion among college athletes. *Journal of Strength and Conditioning Research / National Strength & Conditioning Association*, *24*, 587–96.

Vogt, B. A., & Laureys, S. (2005). Posterior cingulate, precuneal and retrosplenial cortices: cytology and components of the neural network correlates of consciousness. *Progress in Brain Research*, *150*, 205–217.

Walder, D. J., Statucka, M., Daly, M. P., Axen, K., & Haber, M. (2012a\). Biological sex and menstrual cycle phase modulation of cortisol levels and psychiatric symptoms in a non-clinical sample of young adults. *Psychiatry Research*, *197*(3), 314–321.

Wearden, A. J., & Appleby, L. (1996). Research on cognitive complaints and cognitive functioning in patients with chronic fatigue syndrome (CFS): What conclusions can we draw? *Journal of Psychosomatic Research*, *41*(3), 197–211.

Weekes, N. Y., Lewis, R. S., Goto, S. G., Garrison-Jakel, J., Patel, F., & Lupien, S. (2008). The effect of an environmental stressor on gender differences on the awakening cortisol response. *Psychoneuroendocrinology*, *33*(6), 766–772.

Wilson, G. S., & Pritchard, M. (2005a. Comparing Sources of Stress in College Student Athletes and Non-Athletes. *Athletic Insight*.

Wolniak, G. C., Pierson, C. T., & Pascarella, E. T. (2001). Effects of Intercollegiate Athletic Participation on Male Orientations toward Learning. *Journal of College Student Development*, *42*(6), 604–24.

Wöstmann, N. M., Aichert, D. S., Costa, A., Rubia, K., Möller, H.-J., & Ettinger, U. (2013). Reliability and plasticity of response inhibition and interference control. *Brain and Cognition*, *81*(1), 82–94.

Yang, J., Peek-Asa, C., Corlette, J. D., Cheng, G., Foster, D. T., & Albright, J. (2007). Prevalence of and Risk Factors Associated With Symptoms of Depression in Competitive Collegiate Student Athletes. *Clinical Journal of Sport Medicine*, *17*(6), 481–487.

Zouhal, D. H., Jacob, C., Delamarche, P., & Gratas-Delamarche, A. (2008). Catecholamines and the Effects of Exercise, Training and Gender. *Sports Medicine*, *38*(5), 401–423.

Zurrón, M., Pouso, M., Lindín, M., Galdo, S., & Díaz, F. (2009). Event-Related Potentials with the Stroop colour-word task: Timing of semantic conflict. *International Journal of Psychophysiology*, *72*(3), 246–252.

Appendix I. Cognitive Function

Table 1A. Means (M) and standard deviations (SD) for number of exams, papers or projects, frequency of illness and injury for the prior month at each testing time

		n	Exams (M ± SD)	Papers or Projects (M ± SD)	Injury (% yes)	Illness (% yes)
Time 1	Controls	23	1.22 ± 1.24	1.48 ± 1.41	13	30.4
	Student-Athletes	43	0.47 ± 0.88	1.09 ± 1.15	16.3	48.8
Time 2	Controls	22	3.62 ± 0.97	2.36 ± 1.68	13.6	18.2
	Student-Athletes	42	3.21 ± 1.22	2.36 ± 1.34	9.5	47.6
Time 3	Controls	22	3.27 ± 1.35	3.45 ± 2.54	4.5	22.7
	Student-Athletes	41	3.23 ± 1.29	2.61 ± 1.24	7.3	17.1
Time 4	Controls	20	1.55 ± 2.11	0.85 ± 1.39	10	10
	Student-Athletes	36	1.08 ± 1.66	.75 ± 1.46	25	5.6

Table 2A. Means (M) and standard deviations (SD) for subscales of the Perceived Deficits Questionnaire

PDQ		Student-Athletes	
	Test	N	M ± SD
Attention	1	43	7.91 ± 3.40
	2	41	8.66 ± 4.50
	3	41	9.81 ± 4.20
	4	36	7.08 ± 3.66
Retrospective	1	43	4.58 ± 2.81
	2	41	5.22 ± 3.60
	3	41	6.81 ± 4.04
	4	36	5.81 ± 3.30
Prospective	1	43	4.95 ± 2.48
	2	41	5.37 ± 3.49
	3	41	6.76 ± 3.63
	4	36	5.00 ± 2.45
Planning	1	43	7.42 ± 3.27
	2	41	8.95 ± 4.19
	3	41	9.71 ± 4.26
	4	36	7.25 ± 3.33
Total	1	43	24.86 ± 9.65

	2	41	28.20 ± 14.41
	3	41	33.07 ± 14.59
	4	36	25.14 ± 11.28

Table 3A. Means (M) and standard deviations (SD) for ERP amplitude (μv) during congruent Stroop trials

			Controls			**Student- Athletes**	
Congruent	**Test**	**n**	M	SD	**n**	M	SD
N1	1	17	0.12	2.31	39	-0.40	2.04
	2	22	-0.68	1.58	42	-0.53	1.66
	3	18	-1.23	1.71	40	-0.99	2.23
	4	17	-1.18	1.59	33	-0.97	1.50
N2	1	17	-1.59	1.81	39	-1.90	1.72
	2	22	-2.37	1.56	42	-2.04	1.41
	3	18	-3.12	1.72	40	-2.25	1.97
	4	17	-2.48	1.66	33	-2.09	1.55
N4	1	17	-2.77	1.44	39	-2.63	1.47
	2	22	-3.02	1.42	42	-2.58	1.27
	3	18	-3.05	1.67	40	-2.83	1.93
	4	17	-3.32	1.58	33	-2.53	1.44
P1	1	17	3.01	2.29	39	2.17	2.00
	2	22	2.46	2.02	42	2.06	1.90
	3	18	2.24	2.01	40	1.52	1.99
	4	17	1.69	1.68	33	1.09	1.62
P2	1	17	3.91	2.27	39	3.20	2.29
	2	22	3.17	1.85	42	2.89	1.86
	3	18	3.47	1.40	40	2.42	1.61
	4	17	2.53	1.62	33	2.11	1.39
P3	1	17	3.03	1.00	39	2.67	1.04
	2	22	3.02	1.22	42	2.81	1.06
	3	18	3.66	1.34	40	3.08	1.46
	4	17	3.61	1.08	33	2.72	1.02
P6	1	17	0.08	1.76	39	0.62	1.81
	2	22	0.27	2.48	42	1.22	2.29
	3	18	0.49	2.29	40	0.27	2.27
	4	17	-0.35	1.78	33	0.07	1.78

Table 4A. Means (M) and standard deviations (SD) for ERP amplitudes (μv) during incongruent Stroop trials

			Controls			**Student-Athletes**	
Incongruent	**Test**	**n**	M	SD	**n**	M	SD
N1	1	17	0.12	1.69	39	-0.32	2.04
	2	22	-0.60	2.38	42	-0.75	1.66
	3	18	-1.36	2.65	40	-1.37	2.23
	4	17	-0.83	2.75	33	-0.77	1.50
N2	1	17	-1.51	1.10	39	-1.92	1.72
	2	22	-2.43	1.86	42	-1.99	1.41
	3	18	-2.90	1.86	40	-2.51	1.97
	4	17	-2.47	2.04	33	-1.91	1.55
N4	1	17	-3.02	1.97	39	-2.49	1.47
	2	22	-2.46	1.23	42	-2.40	1.27
	3	18	-2.69	1.45	40	-2.98	1.93
	4	17	-3.28	2.13	33	-2.43	1.44
P1	1	17	3.14	2.09	39	2.20	2.00
	2	22	2.23	1.71	42	1.97	1.90
	3	18	2.17	2.52	40	1.26	1.99
	4	17	2.01	2.14	33	1.34	1.62
P2	1	17	4.13	2.02	39	3.22	2.29
	2	22	3.17	2.08	42	2.70	1.86
	3	18	2.84	2.31	40	2.28	1.61
	4	17	2.48	1.89	33	2.15	1.59
P3	1	17	2.78	1.01	39	2.52	1.48
	2	22	2.80	1.01	42	2.71	1.06
	3	18	3.02	1.12	40	3.03	1.46
	4	17	3.29	1.52	33	2.74	1.02
P6	1	17	-0.16	2.03	39	0.87	1.81
	2	22	0.14	1.51	42	1.20	2.29
	3	18	-0.02	2.06	40	0.27	2.27
	4	17	-0.48	3.14	33	0.28	1.78

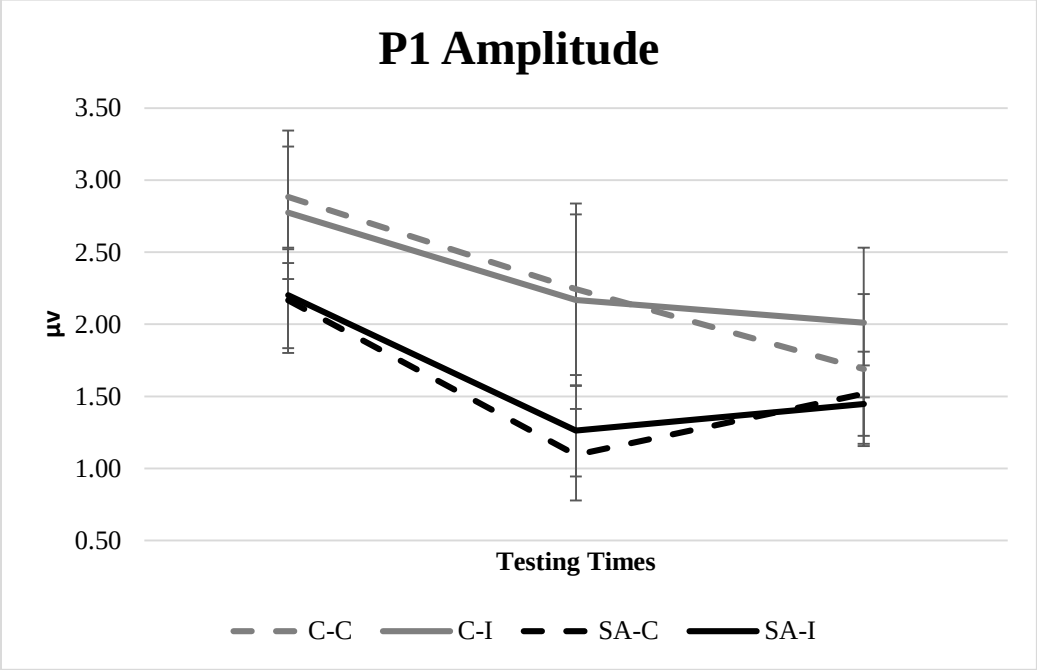
Table 5A. Means (M) and standard deviations (SD) for ERP latency (ms) during congruent Stroop trials

			Controls			**Student-Athletes**	
Congruent	**Test**	**n**	M	SD	**n**	M	SD
N1	1	17	93.99	50.17	39	108.50	28.47
	2	22	93.48	51.76	42	110.16	27.39
	3	18	100.99	36.78	40	104.52	27.40
	4	17	98.57	48.74	33	101.31	31.08
N2	1	17	209.18	15.29	39	207.88	11.96
	2	22	208.12	11.81	42	209.37	12.42
	3	18	208.28	17.73	40	206.32	11.19
	4	17	208.32	12.98	33	207.46	12.70
N4	1	17	403.38	24.75	39	398.69	24.05
	2	22	404.28	26.52	42	398.05	22.93
	3	18	405.33	22.85	40	403.85	24.66
	4	17	412.06	29.64	33	407.54	23.16
P1	1	17	70.02	15.05	39	69.71	14.80
	2	22	66.68	15.69	42	69.92	14.62
	3	18	66.99	16.03	40	64.89	15.50
	4	17	65.28	14.92	33	63.41	15.66
P2	1	17	136.87	8.10	39	137.50	11.80
	2	22	137.55	8.78	42	140.74	12.05
	3	18	136.95	9.74	40	138.00	10.26
	4	17	137.12	8.31	33	144.74	13.45
P3	1	17	292.24	23.10	39	296.27	18.70
	2	22	304.74	20.07	42	297.95	22.81
	3	18	296.21	23.31	40	296.89	20.47
	4	17	296.34	27.08	33	298.08	19.84
P6	1	17	496.13	36.86	39	495.85	33.45
	2	22	505.00	28.61	42	509.43	32.99
	3	18	499.48	32.77	40	499.62	29.68
	4	17	493.73	34.54	33	502.93	28.98

Table 6A. Means (M) and standard deviations (SD) for ERP amplitude during incongruent Stroop trials

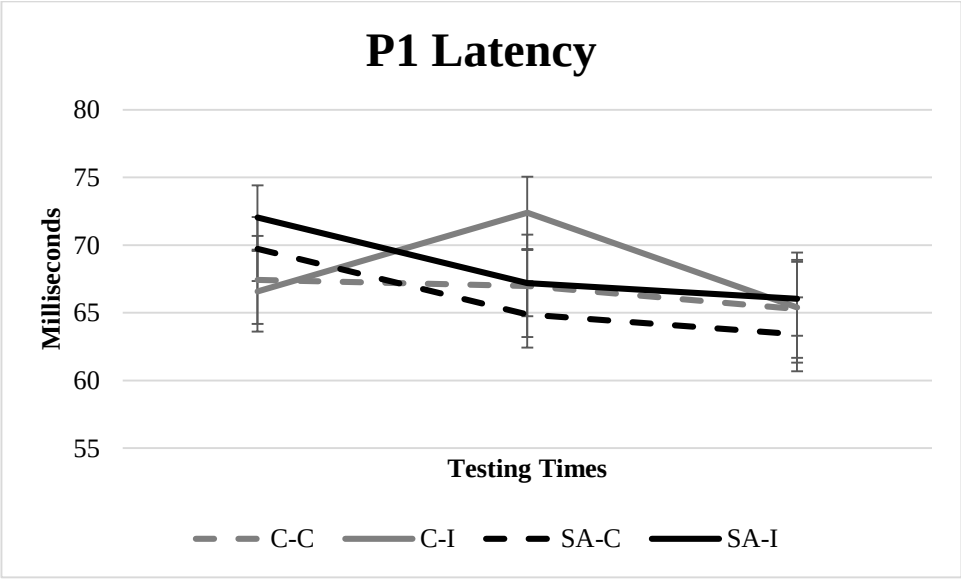
			Controls			**Student-Athletes**	
Incongruent	**Test**	**n**	M	SD	**n**	M	SD
N1	1	17	94.08	52.29	39	103.73	30.18
	2	22	97.53	37.94	42	103.32	40.23
	3	18	105.59	34.19	40	102.00	28.50
	4	17	90.77	50.08	33	93.76	32.49
N2	1	17	209.86	14.59	39	207.57	12.51
	2	22	214.04	15.10	42	210.27	11.52
	3	18	204.93	13.91	40	209.21	12.46
	4	17	209.66	13.83	33	208.39	12.37
N4	1	17	401.14	22.42	39	396.74	24.90
	2	22	405.39	20.64	42	399.38	22.59
	3	18	403.48	24.37	40	410.01	24.29
	4	17	411.89	18.96	33	406.87	22.58
P1	1	17	69.26	13.43	39	72.04	14.42
	2	22	66.42	13.77	42	65.60	17.81
	3	18	72.39	11.32	40	67.19	13.91
	4	17	65.39	16.76	33	66.04	15.92
P2	1	17	137.90	9.71	39	137.86	12.47
	2	22	135.69	8.90	42	141.94	13.19
	3	18	135.06	10.95	40	139.51	10.64
	4	17	137.48	8.94	33	144.16	12.83
P3	1	17	291.02	18.64	39	294.94	17.87
	2	22	298.94	16.27	42	295.85	18.82
	3	18	299.80	19.52	40	294.59	18.67
	4	17	298.91	24.98	33	294.77	19.13
P6	1	17	494.51	35.65	39	492.89	35.71
	2	22	494.42	28.07	42	509.73	27.64
	3	18	499.47	28.50	40	505.23	31.99
	4	17	495.88	29.13	33	498.18	32.26

Figure 1A. P1 Amplitude (μv) at testing times 1, 3 and 4



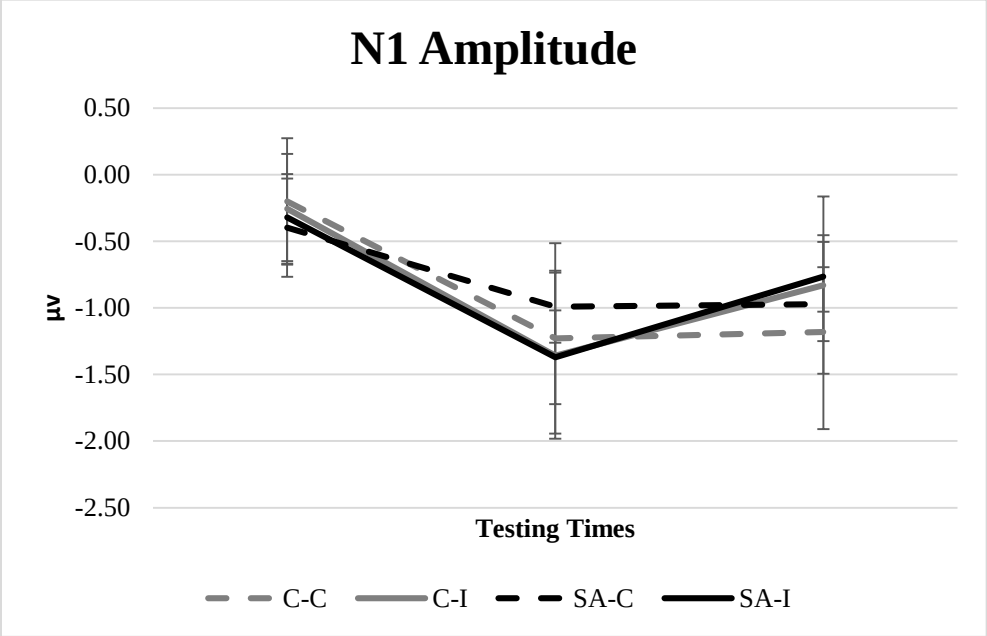
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 2A. P1 Latency (ms) at testing times 1, 3 and 4



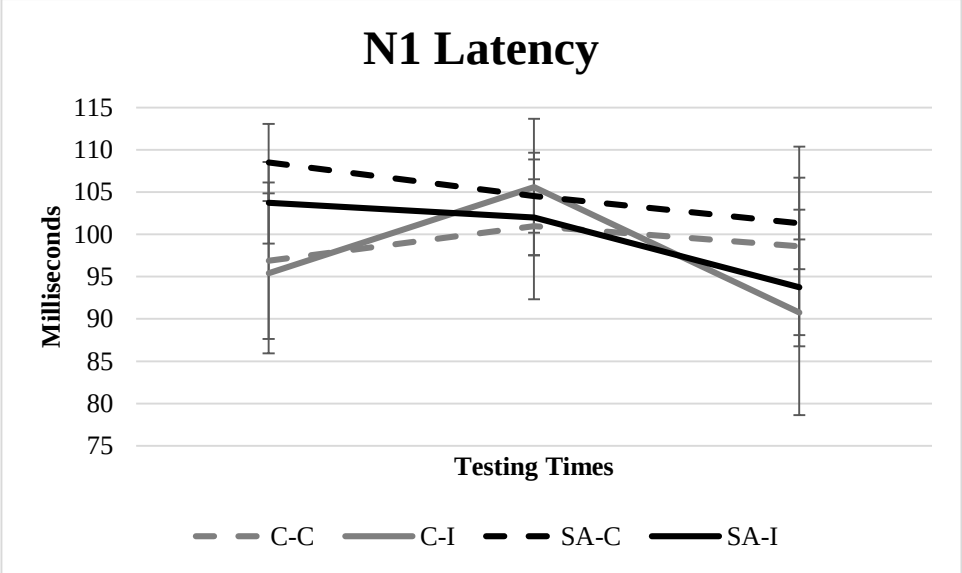
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 3A. N1 Amplitude (μv) at testing times 1, 3 and 4



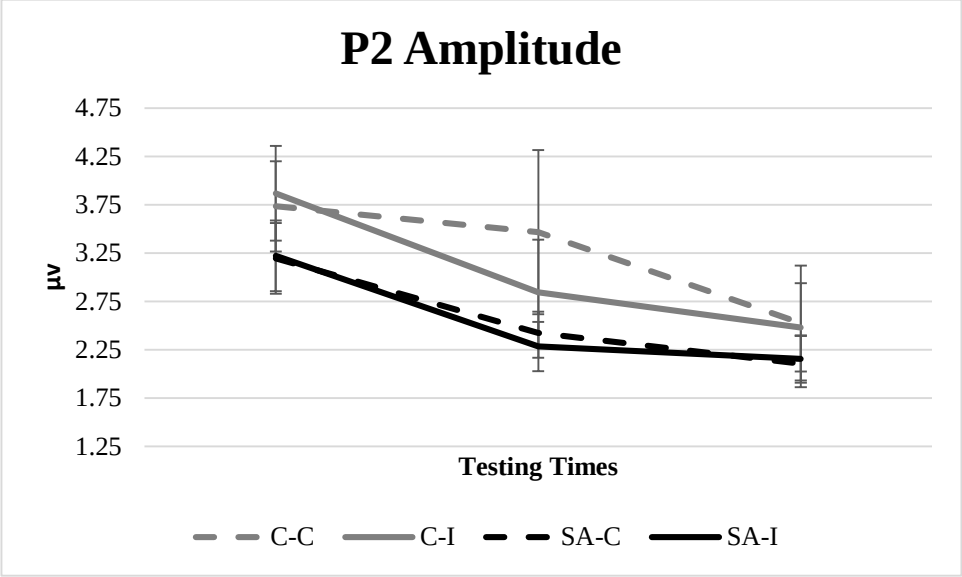
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 4A. N1 Latency (ms) at testing times 1, 3 and 4



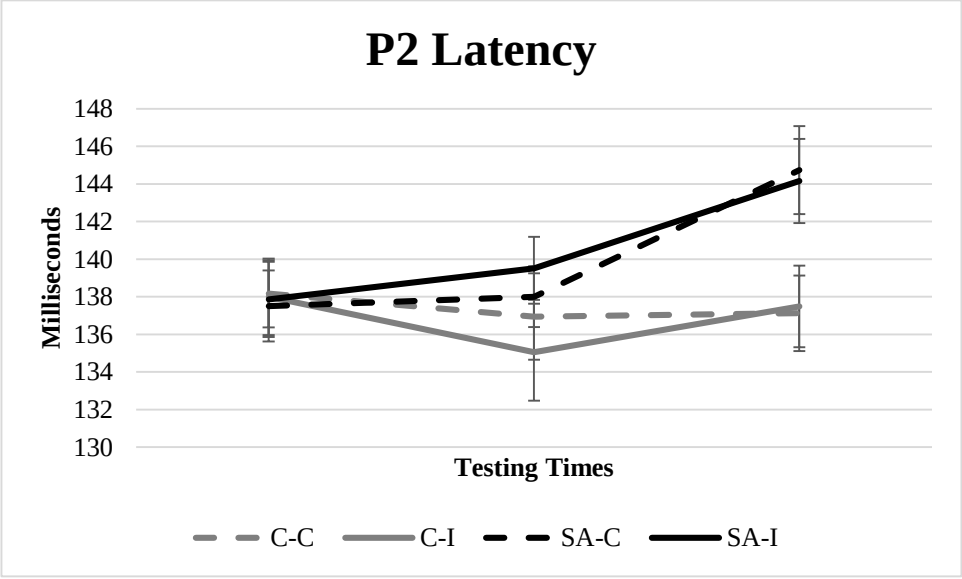
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 5A. P2 Amplitude (μv) at testing times 1, 3 and 4



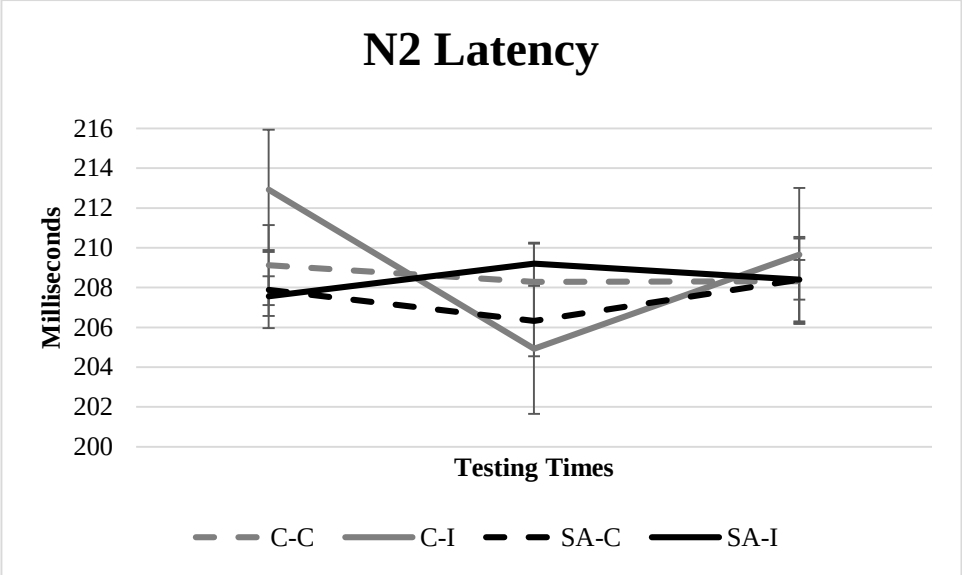
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trial

Figure 6A. P2 Latency (ms) at testing times 1, 3 and 4



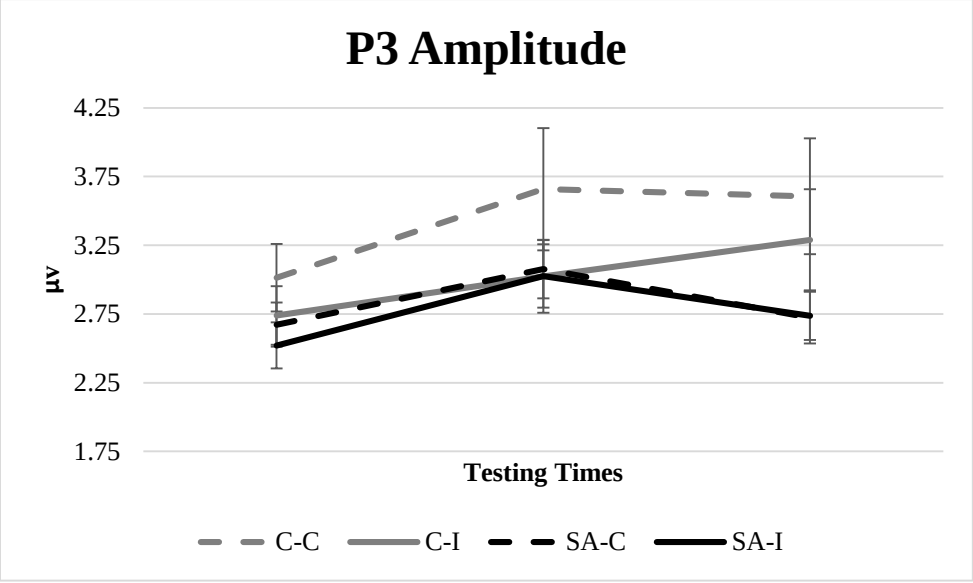
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 7A. N2 Latency (ms) at testing times 1, 3 and 4



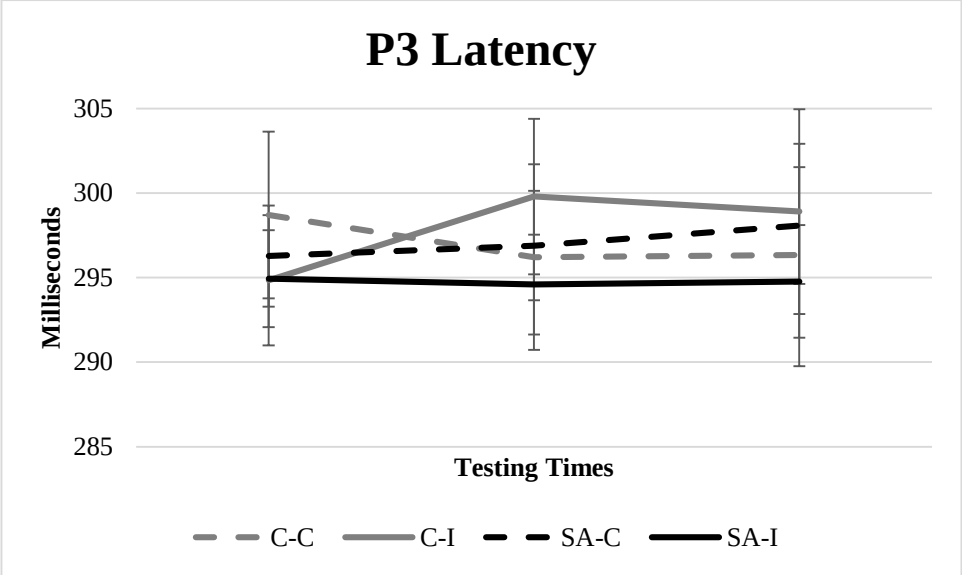
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 8A. P3 Amplitude (μv) for testing times 1, 3 and 4



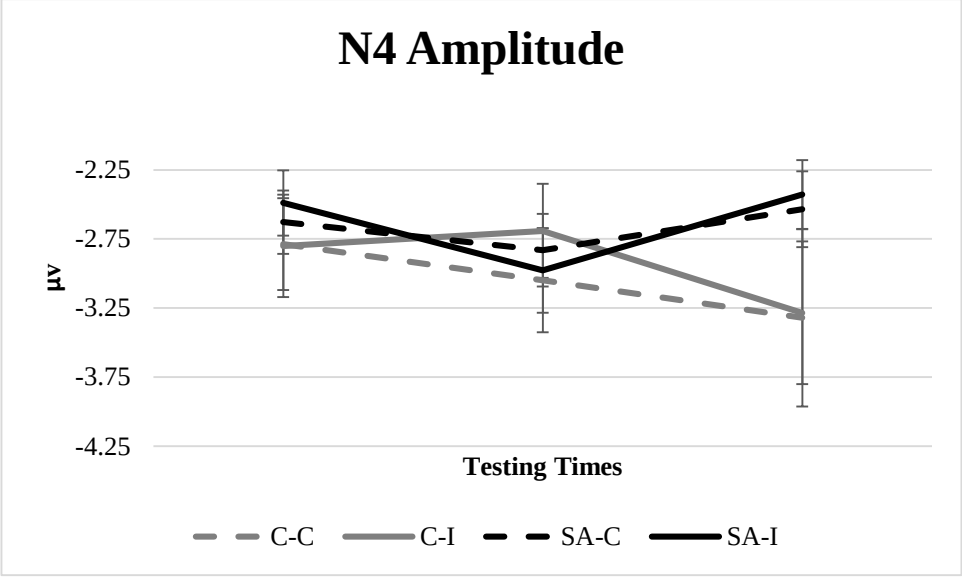
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 9A. P3 Latency (ms) for testing times 1, 3 and 4



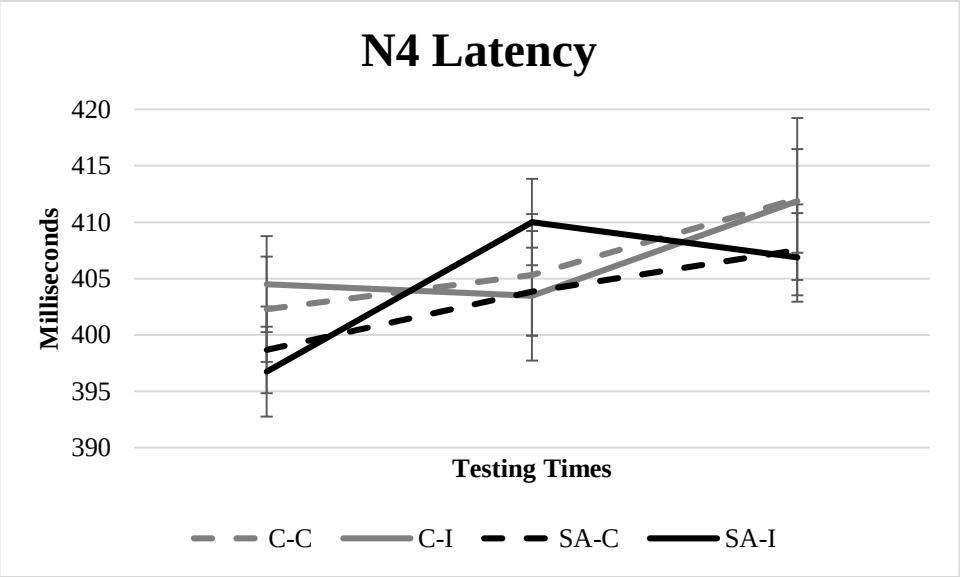
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 10A. N4 Amplitude (μv) during testing times 1, 3 and 4



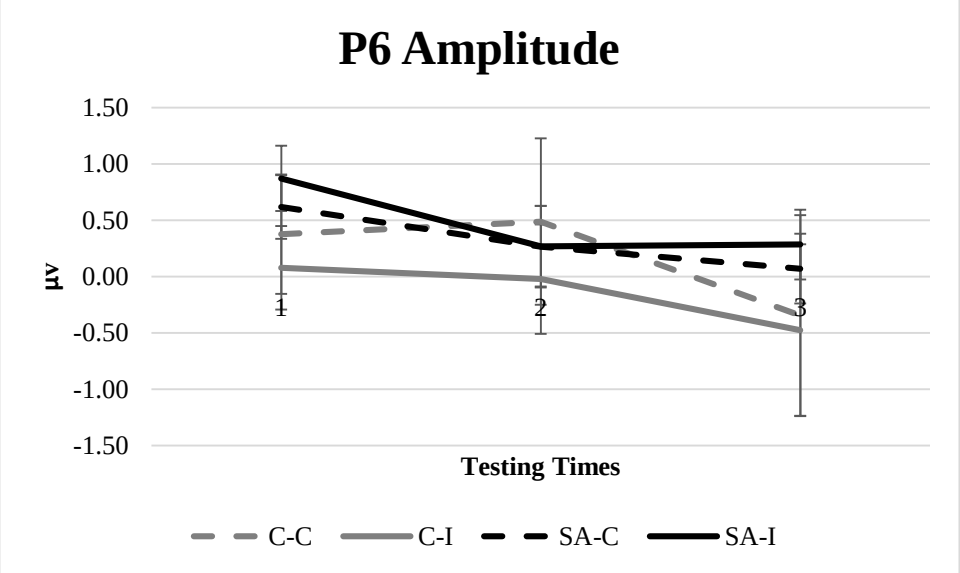
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 11A. N4 Latency (ms) for testing times 1, 3 and 4



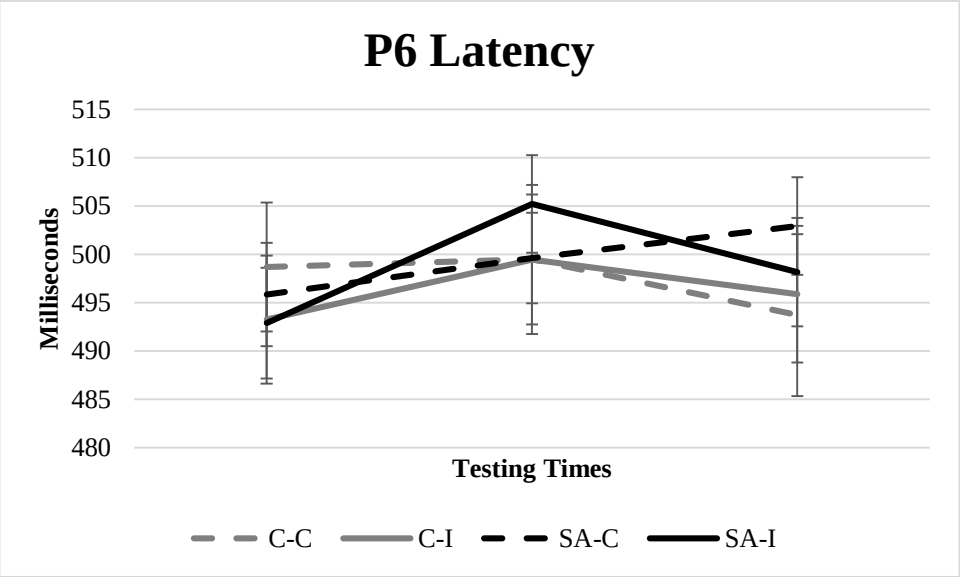
C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

Figure 12A. P6 Amplitude (μv) during testing times 1, 3 and 4



C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

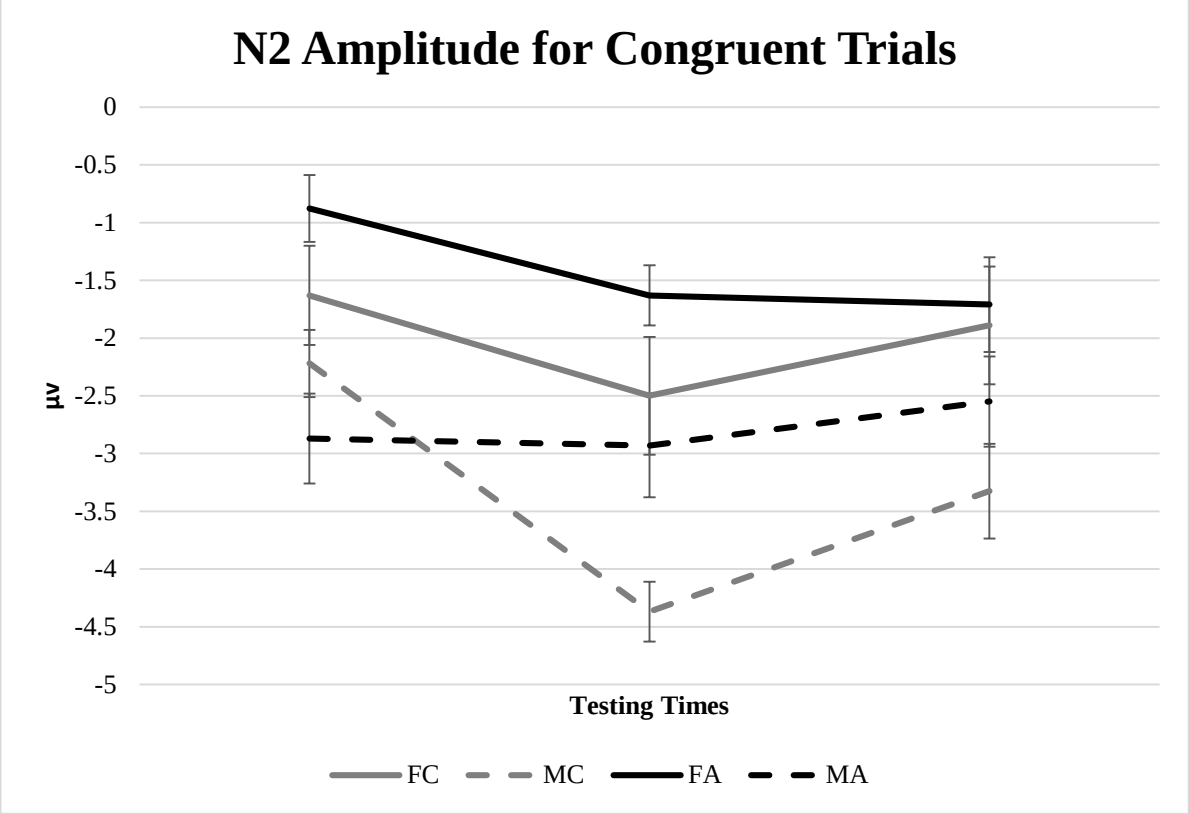
Figure 13A. P6 Latency (ms) during testing times 1, 3 and 4



C-C = Control group congruent trials, CI = Control group incongruent trials, SA-C = Student-Athlete group congruent trials, SA-I = Student-Athlete group incongruent trials

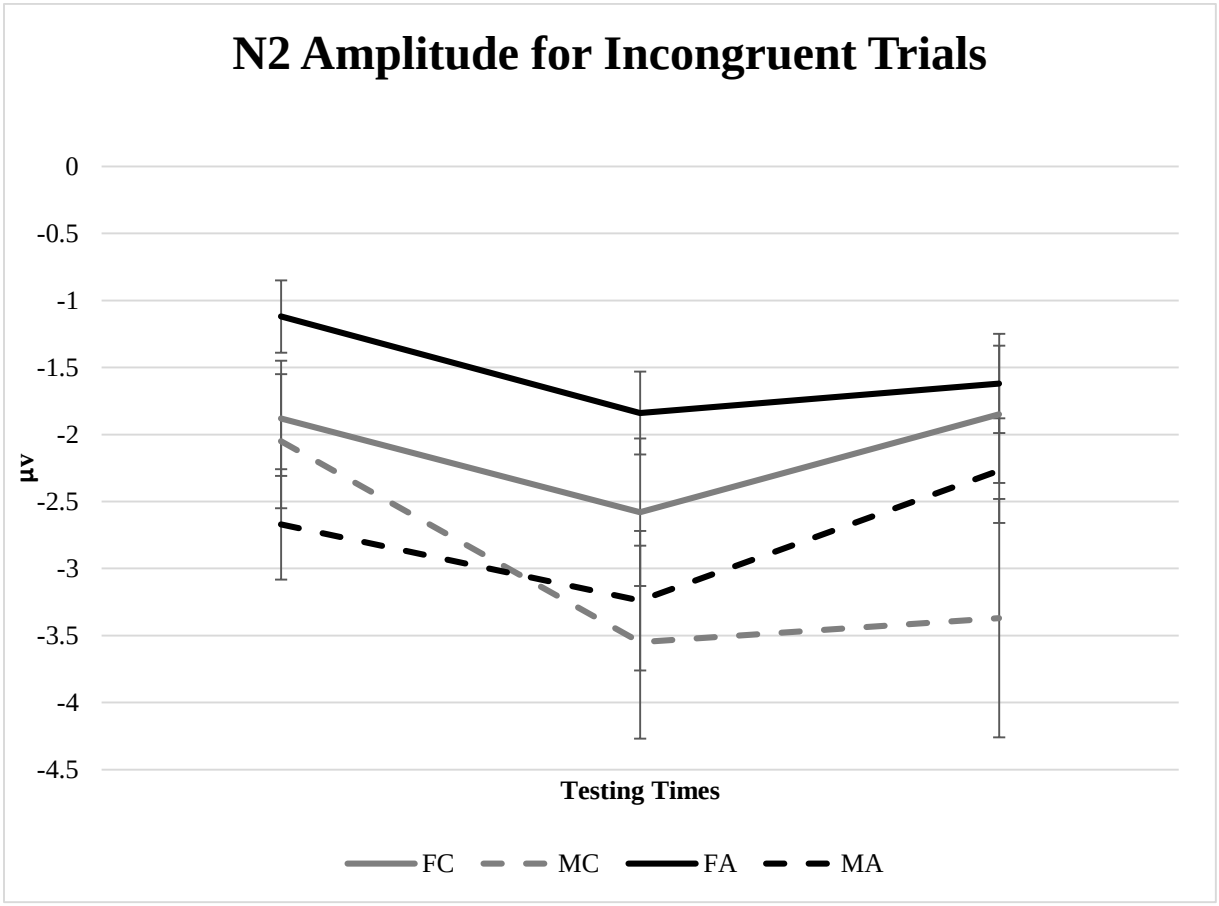
To further investigate the significant interaction of N2 amplitude during congruent and incongruent Stroop trials, a group (female controls (n = 7), male controls (n = 5), female athletes (n = 16) and male athletes (n = 14) x time was performed. Group mean and standard error are presented in Figure 14A for congruent trials and Figure 15A for incongruent trials. The results for N2 amplitude during congruent trials revealed a significant time main effect (F = 17.44, p = 0.000), a significant group main effect (F = 3.14, p = 0.036) and a significant interaction (F = 3.43, p = 0.005). For incongruent trials, a significant main effect of time (F = 16.80, p = 0.000, Huyn-Feldt corrected), a significant interaction (F = 3.28, p = 0.009, Huyn-Feldt corrected) and a nonsignificant group effect (F = 1.76, p = 0.171) were observed.

Figure 14A. N2 Amplitude (μv) during congruent trials across groups and genders



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Figure 15A. N2 Amplitude (μv) during incongruent trials across groups and genders



FC = Female Controls, MC = Male Controls, FA = Female Athletes and MA = Male Athletes

Appendix II. Adaptations Across a Competitive Season

Table 1A. Visual Analog Scale means (M) and standard deviations (SD)

		Controls		Student-Athletes	
	Test	**n**	M ± SD	**n**	M ± SD
Academic	1	23	62.17 ± 18.89	43	46.49 ± 20.81
	2	22	73.73 ± 11.07	42	68.52 ± 17.12
	3	22	78.86 ± 11.26	41	77.93 ± 15.89
	4	14	40.36 ± 32.81	22	36.55 ± 32.20
Exercise	1	23	54.22 ± 16.63	43	65.60 ± 16.70
	2	21	53.14 ± 18.54	42	78.55 ± 14.47
	3	22	46.32 ± 16.88	41	87.56 ± 9.34
	4	20	56.50 ± 18.90	36	57.03 ± 26.19

Table 2A. Epworth Sleepiness Scale means (M) and standard deviations (SD)

	Controls		Student-Athletes	
Test	**n**	M ± SD	**n**	M ± SD
1	23	9.61 ± 3.43	43	10.65 ± 3.92
2	22	10.82 ± 3.67	41	11.63 ± 4.20
3	22	10.05 ± 4.81	41	12.78 ± 4.52
4	19	9.05 ± 3.67	36	10.53 3.55

*Indicates significant difference between groups at time 3

Table 3A. Perceived Stress Scale means (M) and standard deviations (SD)

	Controls		Student-Athletes	
Test	**n**	M ± SD	**n**	M ± SD
1	23	15.09 ± 4.42	43	15.35 ± 5.73
2	22	15.45 ± 5.43	41	19.76 ± 5.86
3	22	16.41 ± 6.78	41	19.59 ± 6.19
4	19	12.26 ± 6.28	36	13.72 ± 5.94

Table 4A. Resting cortisol (µg/dl) with means (M) and standard deviations (SD)

	Controls		Student-Athletes	
Test	**n**	M ± SD	**n**	M ± SD
1	23	0.93 ± 0.44	42	0.7798 ± 0.25
2	22	0.53 ± 0.21	40	0.6436 ± 0.51
3	20	0.42 ± 0.17	41	0.403 ± 0.17
4	17	0.79 ± 0.41	34	0.6991 ± 0.45

Table 5A. Perceived Deficits Questionnaire Total means (M) and standard deviations (SD)

	Controls		Student-Athletes	
Test	**n**	M ± SD	**n**	M ± SD
1	23	22.10 ± 6.85	42	24.86 ± 9.65
2	22	22.77 ± 6.58	40	28.20 ± 14.41
3*	22	24.90 ± 10.72	41	33.07 ± 14.59
4	19	19.42 ± 8.78	34	25.14 ± 11.28

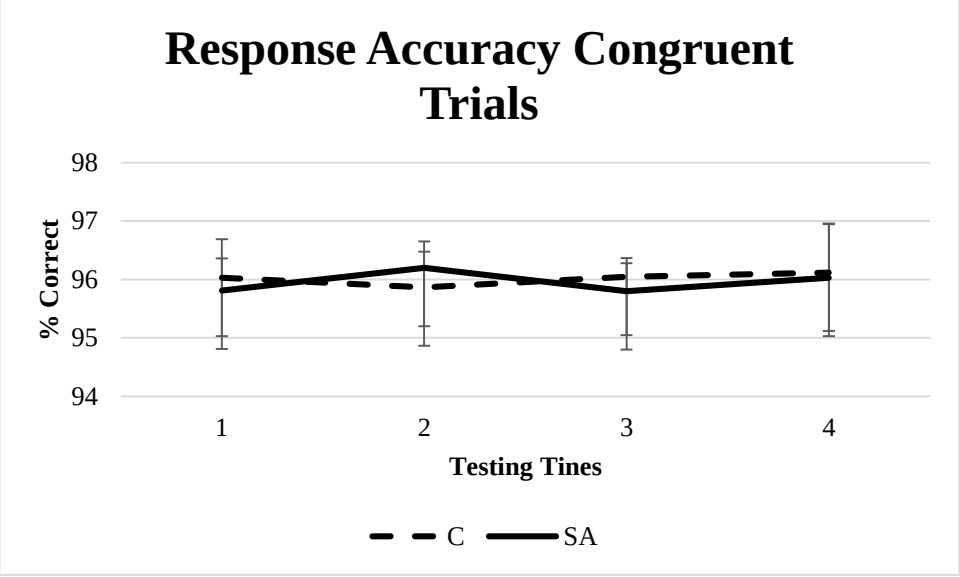
*Indicates significant difference between groups at time 3

Table 6A. Stroop Performance in means (M) and standard deviations (SD)

	Test	**Controls**		**Student-Athletes**	
		n	**M ± SD**	**n**	**M ± SD**
Response Accuracy Congruent (%)	1	23	96.03 ± 3.17	41	95.81 ± 3.54
	2	22	95.87 ± 2.86	42	96.20 ± 2.94
	3	23	96.05 ± 5.92	41	95.80 ± 3.63
	4	17	96.12 ± 3.47	34	96.03 ± 5.33
Response Accuracy Incongruent (%)	1	23	91.79 ± 4.36	41	87.56 ± 8.56
	2	22	92.44 ± 4.05	42	88.24 ± 7.82
	3	23	90.57 ± 7.53	41	87.62 ± 8.22
	4	17	92.68 ± 4.36	34	87.94 ± 7.37

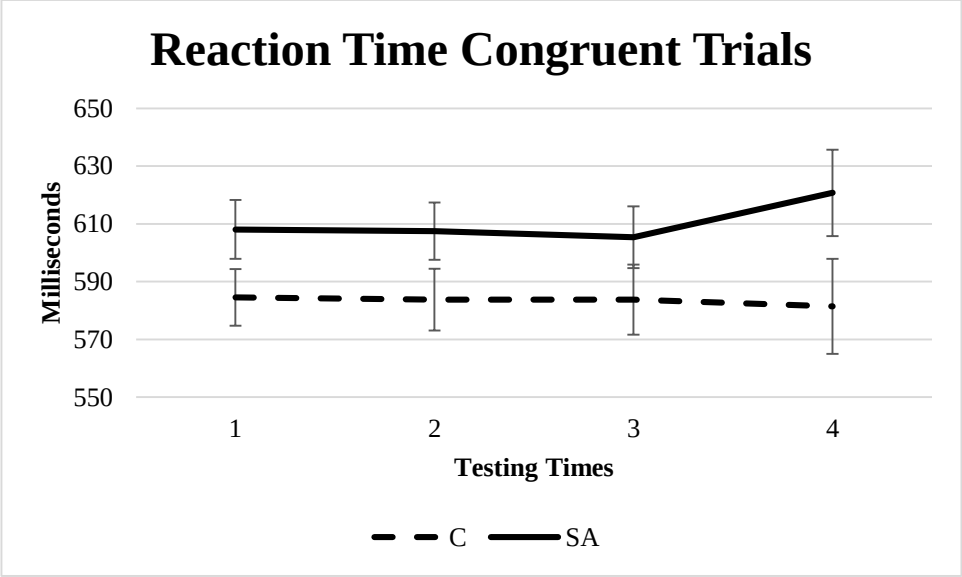
Reaction Time Congruent (ms)	1	23	584.54 ± 46.83	41	608.06 ± 65.34
	2	22	583.77 ± 50.31	42	607.47 ± 64.01
	3	23	583.75 ± 58.14	41	605.37 ± 68.73
	4	17	581.47 ± 67.94	34	620.73 ± 87.22
Reaction Time Incongruent (ms)	1	23	667.15 ± 65.12	41	694.89 ± 90.76
	2	22	663.31 ± 60.04	42	692.77 ± 88.67
	3	23	660.36 ± 71.16	41	688.33 ± 95.66
	4	17	643.49 ± 81.64	34	692.73 ± 101.56

Figure 1A. Group means and standard errors for Response Accuracy on Congruent Trials



C = Controls, SA = Student-Athletes

Figure 2A. Group means and standard errors for Reaction Time on Congruent Trials



C = Controls, SA = Student-Athletes

Appendix III. Gender Differences

Table 1A. Additional Group Descriptives (M = mean, SD = standard deviation) for the past month for each testing time

	Test	Female Controls	Male Controls	Female Athletes	Male Athletes
Exams M ± SD	1	1.33 ± 1.40	1.00 ± .93	0.18 ± 0.66	0.76 ± 1.00
	2	3.64 ± 1.08	3.57 ± 0.79	3.19 ± 1.17	3.24 ± 1.30
	3	3.21 ± 1.58	3.38 ± 0.92	2.81 ± 1.08	3.68 ± 1.38
	4	1.25 ± 2.09	2.00 ± 2.20	1.05 ± 1.78	1.12 ± 1.58
Papers/Projects M ± SD	1	1.20 ± 1.27	2.00 ± 1.60	0.86 ± 1.13	1.33 ± 1.16
	2	2.14 ± 1.41	2.75 ± 2.12	2.38 ± 1.63	2.33 ± 1.02
	3	3.29 ± 2.46	3.75 ± 2.82	2.38 ± 1.16	2.85 ± 1.31
	4	0.92 ± 1.62	0.75 ± 1.04	0.37 ± 1.01	1.18 ± 1.78
Injury (% yes)	1	13.3	12.5	18.2	14.3
	2	21.4	0	9.5	9.5
	3	0	12.5	4.8	10
	4	8.3	12.5	36.8	11.8
Illness (% yes)	1	40	12.5	50	47.6
	2	14.3	25	47.6	42.9
	3	28.6	12.5	19	15
	4	0	25	10.5	0

Table 2A. Group means (M) and standard deviations (SD) on Perceived Stress Scale

		Female Controls		Male Controls		Female Athletes		Male Athletes
Test	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD
1	15	14.93 ± 4.96	8	15.38 ± 3.46	22	15.77 ± 6.00	21	14.90 ± 5.53
2	14	16.64 ± 6.23	8	13.38 ± 2.83	21	20.38 ± 6.45	20	19.10 ± 5.27
3	14	17.43 ± 6.61	8	14.63 ± 7.15	21	20.05 ± 5.26	20	19.10 ± 7.14
4	12	13.75 ± 6.48	7	9.71 ± 5.41	19	15.21 ± 5.79	17	12.06 ± 5.83

Table 3A. Group means (M) and standard deviations (SD) Resting Cortisol Values in µg/dl

		Female Controls		Male Controls		Female Athletes		Male Athletes
Test	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD
1	15	0.8057 ± 0.40	8	1.15 ± 0.45	22	0.76 ± 0.22	20	0.80 ± 0.28
2	14	0.4704 ± 0.14	8	0.6244 ± 0.28	19	0.56 ± 0.32	21	0.72 ± 0.63
3	12	0.3785 ± 0.15	8	0.4941 ± 0.19	21	0.37 ± 0.15	20	0.43 ± 0.19
4	10	0.8721 ± 0.50	7	0.6614 ± 0.19	19	0.78 ± 0.48	15	0.60 ± 0.40

Table 4A. Group means (M) and standard deviations (SD) on Visual Analog Scales

			Female Controls		Male Controls		Female Athletes		Male Athletes
	Test	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD
Academic	1	15	62.27 ± 19.83	8	62.00 ± 18.30	22	39.73 ± 16.45	21	49.05 ± 23.50
	2	14	76.00 ± 11.38	8	69.75 ± 9.92	21	64.33 ± 14.15	21	72.71 ± 19.07
	3	14	81.79 ± 12.34	8	73.50 ± 7.13	21	74.14 ± 10.84	20	78.00 ± 26.00
	4	9	34.56 ± 33.81	5	50.80 ± 31.63	10	39.40 ± 33.33	12	19.52 ± 26.69
Exercise	1	15	52.00 ± 16.44	8	58.38 ± 17.28	22	60.59 ± 15.96	21	72.00 ± 14.61
	2	14	53.57 ± 21.75	7	52.29 ± 10.93	21	81.48 ± 13.04	21	75.60 ± 15.54
	3	14	44.43 ± 18.22	8	49.63 ± 14.79	21	88.43 ± 8.12	20	82.52 ± 21.55
	4	12	53.50 ± 21.80	8	61.00 ± 13.56	19	59.53 ± 26.13	17	43.90 ± 32.40
Mental Fatigue	1	15	43.80 ± 19.96	8	36.00 ± 16.78	19	45.41 ± 26.15	21	43.19 ± 26.12
	2	14	53.29 ± 24.10	8	54.50 ± 20.37	21	64.43 ± 20.81	21	68.81 ± 20.37
	3	14	63.64 ± 30.81	8	49.50 ± 23.84	21	74.43 ± 15.60	20	71.75 ± 26.77
	4	12	32.58 ± 24.37	8	33.13 ± 23.95	19	41.11 ± 26.52	15	29.53 ± 28.57
Physical Fatigue	1	15	37.93 ± 24.45	8	38.25 ± 25.19	22	55.59 ± 23.99	21	61.10 ± 23.23
	2	14	45.71 ± 24.10	8	33.75 ± 17.68	21	73.05 ± 15.34	21	72.62 ± 18.26
	3	14	40.36 ± 23.35	8	33.25 ± 20.08	21	81.10 ± 11.08	20	74.80 ± 20.20
	4	12	39.17 ± 21.16	8	39.75 ± 15.82	19	54.11 ± 26.24	17	53.94 ± 29.60

Table 5A. Group means (M) and standard deviations (SD) for the Epworth Sleepiness Scale

		Female Controls		Male Controls		Female Athletes		Male Athletes
Test	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD
1	15	9.53 ± 3.42	8	9.75 ± 3.69	22	11.41 ± 4.03	21	9.86 ± 3.73
2	14	10.00 ± 2.77	8	12.25 ± 4.74	21	12.19 ± 4.59	20	11.05 ± 3.78
3	14	9.20 ± 3.38	8	11.50 ± 6.65	21	13.90 ± 4.46	20	11.60 ± 4.38
4	12	8.92 ± 3.65	7	9.39 ± 3.99	19	10.89 ± 3.97	17	10.12 ± 3.08

Table 6A. Group means (M) and standard deviations (SD) on the Perceived Deficits Questionnaire

			Female Controls		**Male Controls**		**Female Athletes**		**Male Athletes**
	Test	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD
Attention	1	15	8.07 ± 2.81	8	7.50 ± 2.62	22	8.23 ± 3.87	21	7.57 ± 2.89
	2	14	7.21 ± 3.26	8	7.38 ± 2.33	21	9.57 ± 4.91	20	7.70 ± 3.92
	3	14	7.50 ± 3.37	8	6.88 ± 4.55	21	10.38 ± 4.68	20	9.20 ± 3.65
	4	12	5.67 ± 2.67	7	4.43 ± 2.23	19	7.47 ± 3.79	17	6.65 ± 3.57
Retrospective	1	15	3.33 ± 1.84	8	5.63 ± 3.81	22	4.27 ± 2.51	21	4.90 ± 3.13
	2	14	3.79 ± 1.76	8	4.50 ± 2.93	21	5.10 ± 3.48	20	5.35 ± 3.82
	3	14	4.71 ± 3.31	8	5.63 ± 4.13	21	7.14 ± 3.97	20	6.45 ± 4.20
	4	12	3.75 ± 2.56	7	4.14 ± 2.73	19	5.53 ± 3.44	17	6.12 ± 3.22
Prospective	1	15	3.73 ± 1.44	8	3.00 ± 1.60	22	5.41 ± 2.48	21	4.48 ± 2.44
	2	14	4.93 ± 1.49	8	3.63 ± 1.51	21	5.76 ± 3.33	20	4.95 ± 3.69
	3	14	4.43 ± 1.65	8	5.63 ± 3.07	21	7.00 ± 3.74	20	6.50 ± 3.59
	4	12	4.58 ± 2.35	7	4.00 ± 1.91	19	5.26 ± 2.86	17	4.71 ± 1.93
Planning	1	15	6.73 ± 2.69	8	6.50 ± 1.15	22	8.10 ± 3.29	21	6.71 ± 3.18
	2	14	7.00 ± 2.91	8	7.00 ± 1.41	21	9.86 ± 4.26	20	8.00 ± 4.01
	3	14	8.00 ± 4.08	8	7.25 ± 2.87	21	9.90 ± 3.97	20	9.50 ± 4.64
	4	12	6.42 ± 1.08	7	2.14 ± 2.41	19	7.79 ± 3.46	17	6.65 ± 3.18
PDQ Total	1	15	21.87 ± 6.06	8	22.63 ± 8.58	22	26.00 ± 10.01	21	23.67 ± 9.34
	2	14	22.93 ± 7.01	8	22.5 ± 6.21	21	30.29 ± 14.73	20	26.00 ± 14.09
	3	14	24.64 ± 9.19	8	25.38 ± 13.68	21	34.43 ± 14.86	20	31.65 ± 14.53
	4	12	20.42 ± 9.61	7	17.71 ± 7.52	19	26.05 ± 11.83	17	24.12 ± 10.90

Table 7A. Group means (M) and standard deviations (SD) on the Stroop Color-Naming task

			Female Controls		Male Controls		Female Athletes		Male Athletes
	Test	**n**	**M ± SD**	**n**	**M ± SD**	**n**	**M ± SD**	**n**	**M ± SD**
Response Accuracy	1	15	96.51 ± 3.32	8	95.12 ± 2.84	20	95.81 ± 4.25	21	95.81 ± 2.82
Congruent (%)	2	14	95.71 ± 2.63	8	96.14 ± 3.41	21	95.77 ± 3.18	21	96.63 ± 2.68
	3	15	95.53 ± 7.20	8	97.02 ± 2.13	21	95.78 ± 3.56	20	95.82 ± 3.80
	4	10	95.63 ± 4.32	7	96.81 ± 1.80	19	95.30 ± 6.74	15	96.94 ± 2.65
Response Accuracy	1	15	91.00 ± 4.53	8	93.29 ± 3.82	20	87.43 ± 10.49	21	87.68 ± 6.49
Incongruent (%)	2	14	91.18 ± 3.90	8	94.64 ± 3.50	21	89.16 ± 8.03	21	87.32 ± 7.86
	3	15	88.87 ± 8.51	8	93.77 ± 3.90	20	88.37 ± 6.78	20	86.87 ± 9.58
	4	10	92.24 ± 4.32	7	93.32 ± 4.68	19	88.09 ± 7.19	15	87.74 ± 7.85
Reaction Time	1	15	582.47 ± 47.78	8	588.44 ± 47.97	20	605.66 ± 54.49	21	610.33 ± 75.54
Congruent (ms)	2	14	575.65 ± 54.87	8	597.96 ± 40.52	21	602.34 ± 57.27	21	612.59 ± 71.17
	3	15	574.69 ± 61.35	8	600.72 ± 50.89	21	598.90 ± 55.82	20	612.16 ± 81.06
	4	10	573.43 ± 74.90	7	592.95 ± 60.27	19	616.76 ± 63.17	15	625.75 ± 112.94
Reaction Time	1	15	671.17 ± 72.65	8	659.61 ± 51.69	20	685.76 ± 64.92	21	703.58 ± 110.94
Incongruent (ms)	2	14	661.08 ± 65.36	8	667.22 ± 53.41	21	682.84 ± 68.10	21	702.69 ± 106.18
	3	15	658.63 ± 78.97	8	663.62 ± 58.58	21	681.01 ± 83.89	20	696.02 ± 108.34
	4	10	673.35 ± 92.20	7	643.69 ± 70.86	19	683.25 ± 71.50	15	704.74 ± 132.15

Table 8A. Group means (M) and standard deviations (SD) on the Profile of Mood States

POMS			Female Controls		Male Controls		Female Athletes		Male Athletes
	Test	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD	**n**	M ± SD
Tension	1	15	12.13 ± 5.03	8	13.50 ± 6.57	22	11.91 ± 8.15	21	13.95 ± 5.06
	2	14	11.43 ± 5.69	8	13.13 ± 4.88	21	15.14 ± 7.81	20	13.55 ± 5.47
	3	14	12.50 ± 6.48	8	11.50 ± 4.69	21	15.43 ± 5.71	20	16.10 ± 5.98
	4	12	7.58 ± 6.89	7	8.00 ± 4.73	19	9.74 ± 6.71	17	8.94 ± 4.85
Depression	1	15	6.33 ± 5.90	8	12.50 ± 9.12	22	11.82 ± 13.48	21	10.43 ± 8.56
	2	14	9.43 ± 10.44	8	10.00 ± 8.40	21	18.10 ± 14.70	20	15.05 ± 9.46
	3	14	9.50 ± 9.65	8	10.75 ± 10.99	21	16.81 ± 13.50	20	16.70 ± 12.23
	4	12	7.58 ± 8.73	7	6.57 ± 4.35	19	10.84 ± 12.15	17	8.24 ± 7.26
Anger	1	15	5.73 ± 5.13	8	8.13 ± 4.76	22	10.27 ± 9.50	21	9.86 ± 6.77
	2	14	6.86 ± 5.82	8	7.88 ± 3.76	21	16.81 ± 12.34	20	13.40 ± 8.69
	3	14	5.50 ± 5.13	8	8.00 ± 6.93	21	13.62 ± 10.20	20	16.95 ± 10.71
	4	12	5.42 ± 8.55	7	5.71 ± 3.4	19	14.16 ± 11.16	17	8.06 ± 5.25
Fatigue	1	15	11.20 ± 3.91	8	10.88 ± 4.73	22	11.45 ± 6.00	21	12.29 ± 4.78
	2	14	10.43 ± 5.15	8	10.75 ± 6.34	21	16.10 ± 6.17	20	13.65 ± 5.08
	3	14	10.57 ± 5.06	8	9.00 ± 3.16	21	16.57 ± 4.98	20	15.00 ± 5.99
	4	12	6.67 ± 4.66	7	7.00 ± 2.71	19	9.00 ± 4.92	17	9.29 ± 4.61
Vigor	1	15	14.4 ± 4.72	8	19.38 ± 6.14	22	14.41 ± 4.16	21	15.95 ± 5.16
	2	14	15.57 ± 6.99	8	19.13 ± 8.46	21	12.43 ± 4.68	20	14.80 ± 4.05
	3	14	15.50 ± 6.22	8	19.13 ± 7.22	21	12.48 ± 4.38	20	14.85 ± 4.39
	4	12	19.83 ± 4.95	7	22.14 ± 5.01	19	16.63 ± 4.22	17	18.65 ± 4.82
Confusion	1	15	7.47 ± 3.36	8	8.25 ± 3.62	22	8.36 ± 4.18	21	7.14 ± 4.27
	2	14	8.14 ± 3.66	8	8.25 ± 3.24	21	10.62 ± 6.63	20	10.30 ± 4.85
	3	14	8.64 ± 4.11	8	8.88 ± 3.27	21	11.48 ± 5.02	20	10.80 ± 5.08
	4	12	6.25 ± 4.43	7	4.86 ± 3.67	19	7.95 ± 3.91	17	7.41 ± 3.71
Total	1	15	128.47 ± 21.08	8	133.88 ± 23.62	22	139.41 ± 39.56	21	137.71 ± 27.60
	2	14	130.71 ± 25.48	8	130.88 ± 26.6	21	164.33 ± 46.80	20	151.15 ± 31.26
	3	14	131.21 ± 29.21	8	128.00 ± 32.31	21	161.43 ± 36.64	20	160.70 ± 38.02
	4	12	113.67 ± 35.37	7	110.00 ± 19.94	19	135.05 ± 37.67	17	123.29 ± 24.95