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Samantha Mohler University of Arkansas, Fayetteville

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Examining the Appropriate Recovery Interval Following Maximal Exertion for Baseline Computerized Neurocognitive Testing (CNT)

> A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Kinesiology

> > by

Samantha Mohler University of Arkansas Bachelor of Science in Kinesiology, 2016

> May 2018 University of Arkansas

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This thesis is approved for recommendation to the Graduate Council.

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Abstract

Background: Computerized neurocognitive testing is part of the recommended multi-faceted approach to SRC assessment. Prior research has suggested that maximal exertion negatively effects CNT test scores. **Purpose:** To identify the appropriate timing of the administration of CNT following maximal exertion in healthy college-aged students. **Study Design:** Random cross-over, repeated measures design. **Methods:** Participants will be administered CNT on four different visits, with at least one week between administrations. A $VO₂$ max treadmill test will be performed before CNT administration during three of the four trials. Following the $VO₂$ max test, participants will rest for <2 minutes (immediate), 10-minutes, or 20-minutes before taking CNT. The fourth trial, without maximal exertion preceding CNT administration, will serve as the control. All trials will be randomly-counterbalanced to negate practice effects. **RESULTS:** There was a significant within-subjects effect for prescribed post-exertion recovery intervals on total symptom scores (*Wilks* $\lambda = .62$, *F* [3, 23] = 4.64, $p = .01$, $\eta^2 = .38$). Total symptom scores were significantly higher at the immediate ($p < .002$), 10-minutes ($p = .018$), and 20-minutes (p *=* .011) post-exertion recovery intervals compared to baseline. Additionally, a significantly positive within-subjects effect for prescribed post exertion recovery was observed for processing speed (p=.009, *Wilks* $\lambda = .60$, *F* [3, 27] = 5.9, $\eta^2 = .396$). No significant effect was observed for visual memory ($p = .07$), verbal memory ($p = .06$), or reaction time ($p = .40$). **CONCLUSION:** Baseline symptom scores were negatively influenced processing speed was enhanced by maximal exertion. These changes continue to be elevated 20 minutes post-exertion. Moreover, cognitive performance was not significantly impaired following maximal exercise. To obtain more accurate baseline symptom scores, and allow processing speed composites to return to

normal, sports medicine professionals should wait at least 20 minutes following maximal exertion before administering CNT.

Acknowledgements

I would like to thank members of my committee. Thank you Dr. R.J. Elbin, Dr. Matthew Ganio, and Dr. Brendon McDermott.

Thank you Dr. Elbin for your mentorship and support. Your passion for research is contagious. Over the last few years, you have taught me endless professional, and life skills. Thank you for being a "Rock-star".

A special thank you is extended to Cory Butts for going above and beyond to help me. Even in the "wee" hours of the morning, I could count on Cory to be a never-ending source of support.

Thank you Abby Hess, the "A-team" (Eric Polomis and Damon Reel), Chase Ladd, Zach Sebghatti, and Katie Stephenson-Brown for helping me with the exertion protocol trials. Thank you to members of the University of Arkansas Office for Sport Concussion Research Lab who have also helped me with this project. Your support, even if not directly related to this project, should not be minimized.

Finally, thank you to Regan Hime for your relentless support, and for pulling late night study sessions with me to "grind it out".

Dedication

This thesis is dedicated to my parents, Michael and Sherry Mohler.

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Introduction

Approximately 1.6–3.8 million sport and recreation-related concussions occur each year in the United States (Langlois, Rutland-Brown, & Wald, 2006). Sport-related concussion (SRC) can negatively affect the physical, emotional, social, and cognitive functioning of athletes. If clinically mismanaged, the consequences of SRC on long-term health can be catastrophicresulting in chronic post-concussion symptoms, permanent brain damage, and although very rare, result in death in younger athlete populations. Therefore, to ensure that athletes with concussions receive proper care and avoid poor recovery outcomes, a multi-faceted, objective assessment approach for SRC management is recommended (McCrory, et al., 2013; McCrory, et al., 2017).

Approximately 63% of all sport-related concussions go unreported (McCrea, Hammeke, Olsen, Leo, & Guskiewicz, 2004). Traditionally, sports medicine professionals primarily relied on self-reported symptoms to assess and manage concussion. However, many athletes fail to disclose or minimize their symptoms because of eagerness to return to play, lack of knowledge, or fear of letting their teammates down (McCrea et al., 2004). Due to the subjectivity and lack of accuracy associated with self-reported symptoms, objective testing is needed. Objective tests, used in conjunction with self-reported symptoms, better quantify impairment and provide a visual representation of recovery following a concussion. One test currently used for assessment of sport-related concussion is computerized neurocognitive testing (CNT), which objectively measures several aspects of cognitive functioning (Van Kampen, Lovell, Pardini, Collins & Fu, 2006).

Computerized neurocognitive testing is part of the recommended multi-faceted approach to SRC assessment. These assessments includes batteries of cognitive tasks measuring different domains of cognitive function: verbal memory, visual design memory, concentration, processing

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speed, and reaction time (Covassin, Elbin, Stiller-Ostrowski, & Kontos, 2009). Similar to "old fashioned" paper and pencil neurocognitive tests, technological improvements have advanced neurocognitive testing used for assessing SRC. Governed by a computer, CNT affords clinicians the ability to administer multiple versions, generates automated scoring, and standardized administration. Additionally, CNT enables one clinician to administer multiple tests simultaneously to athletes in a group setting. Computerized neurocognitive testing is best administered before season (baseline) and after suspected concussion. In recent SRC literature CNT has been coined the "cornerstone" of SRC assessment and management (Broglio, et al., 2014). Pre-injury (baseline) and post-injury CNT scores can be compared allowing the athlete to serve as their own control. Scores can be analyzed by sports medicine professionals to better evaluate cognitive function and recovery. Quantitative data from this test can provide a visual depiction of an individual's condition helping to bridge the gap between the athlete, coaches, parents, academic personnel, and the clinician (Broglio, et al., 2014). Ensuring the accuracy of the baseline CNT assessment is critical to SRC management.

The accuracy of baseline CNT is a key component of SRC management (Collins, Kontos, Reynolds, Murawski, & Fu, 2014). Baseline CNT administration is a "snapshot" of an individual cognitive performance. It is imperative sports medicine professionals conduct baseline testing in an environment and at a time that will enable athletes to put forth their best effort, and perform at their maximum potential. Researchers have identified several factors that negatively influence CNT baseline scores including learning disabilities, attention deficit hyperactivity disorder, (Elbin, et al., 2013), concussion history (Broglio, et al., 2014), testing environment (Moser, Schatz, Neidzwski, & Ott, 2011), and prior exertion (Covassin, Weiss, Powell, &

Womack, 2007). Sports medicine professionals should attempt to control, or at minimum consider these factors when interpreting baseline CNT performance.

The relation between prior physical exertion and baseline CNT scores is understudied. Furthermore, much of the literature exploring the interaction of maximal exercise and its effect on cognitive performance is contradictory. Some studies report a negative relationship between the effects of maximal exercise and cognitive performance (Covassin, et al., 2007; Dietrich 2006; Nada, Balde, & Manjunatha 2013). These projects analyzed cognition after a bout of maximal (Covassin, et al., 2007), high-intensity (Nada, Balde, & Manjunatha) and locally fatigued state muscles (Dietrich, 2006). All three studies detected a negative change in cognitive function when compared to baseline or controls. In contrast, other studies have found moderate physical activity to actually facilitate cognitive performance (Brisswalter, Collardeau, & Rene, 2002; Hillman, Snook, and Jerome, 2003; Pontifex, Hillman, Fernhall, & Thompson, 2009). Other studies examining the effects of maximal exertion on cognitive performance have produced mixed findings (Coles & Tomporowski, 2008). This inconsistency is most likely due to differing methodologies, exertion protocols, and outcome measures used when assessing cognition.

Covassin and colleagues (2007) reported that maximal exercise has negative effects on CNT baseline scores when compared to the non-exerted controls. Covassin (2007) administered CNT immediately following the completion of a maximal exertion protocol. When compared to a non-exerted control group, the experimental group performed significantly lower on verbal memory composite scores during the post-exertion CNT. The experimental group also scored significantly lower following maximal exercise when compared to their own baseline. The results of Covassin's study implies that CNT should not be administered directly after exertion.

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This study suggests maximal exercise negatively affects the outcome on CNT; however, the optimal recovery time following a bout of maximal exertion has not been examined.

This research is significant as anecdotal reports from sports medicine professionals suggest baseline testing is often administered in the short time following a bout of physical activity (e.g., strength and conditioning workout) due to time constraints of a rigorous sport environment. Pre-season baseline testing is often an afterthought for many coaches during the start of the new season, and is squeezed into a demanding schedule. No study has examined how long the sports medicine professional should wait before administering baseline CNT following maximal exertion.

If baseline CNT scores are artificially lower because the test was administered immediately after maximal physical activity, athletes sustaining a concussion may go unnoticed. Cognitive deficits caused by SRC may not be as evident if the "snapshot" of an individual's baseline cognitive performance is inaccurate. The wait-time for administering CNT following a bout of maximal exertion is unknown and there is currently no recommended time interval to wait before testing. In order to outline "best practice" guidelines for baseline CNT administration, additional research concerning maximal exercise exertion and recovery is needed. This data will directly impact CNT baseline-testing practices of sports medicine professionals.

Little research has examined maximal exercise and CNT outcomes and a consensus in current literature is mixed. The recovery intervals (immediate, 10 minutes, and 20 minutes post exercise) for this study were determined based on meta-analyses examining exercise and neurocognitive function. A meta-analysis conducted by Chang, Labban, Gapin and Etnier (2012) examined primary moderators of acute exercise and cognitive function reported the delay following exercise significantly influenced the effect size of observations; 0-10 minutes recovery intervals resulted in significant negative effects while 11-20 minutes and beyond recovery intervals resulted in decreasing positive effects. A different meta-analysis conducted by Lamburne and Tomporowski (2010) reported significant positive effects of exercise on cognition immediately after and within 15 minutes of cessation of exercise. Cognitive testing performed after 15 minutes did not result in significant effects. Because of equivocal findings neurocognitive outcomes following a 10 minute recovery interval was not hypothesized, and will be an exploratory time point.

Purpose of the Study

The purpose of this study is to identify the appropriate recovery interval following maximal exertion for the administration of CNT in healthy, active college-aged students.

Hypotheses

Hypothesis 1.Verbal memory composite scores will be significantly lower than baseline at the immediate recovery interval, but not at the 20-minute recovery interval.

Hypothesis 2.Visual memory composite scores will be significantly lower than baseline at the immediate recovery interval, but not at the 20-minute recovery interval.

Hypothesis 3. Processing speed composite scores will be significantly lower than baseline at the immediate recovery interval, but not at the 20-minute recovery interval.

Hypothesis 4. Reaction time will be significantly faster than baseline at the immediate recovery interval, but not at the 20-minute recovery interval.

Hypothesis 5. Total PCSS scores will be significantly higher than baseline at the immediate recovery interval, but not at the 20-minute recovery interval.

Review of Literature

Concussion is defined as a complex injury resulting from a cascade of neurometabolic events following biomechanical trauma, resulting in variable symptoms and impairments (Halstead & Walter, 2010). Clinical, pathological, and biomechanical paradigms utilized to help define this injury, according to the $5th$ International Conference on Concussion in 2017, include: transmission of direct or indirect force transmission to the head, onset of short-lived neurological impairments due to functional disturbances, rather that structural injury, and symptoms that normally resolve with adequate rehabilitation (McCrory, et al., 2017). In literature, concussions have also been defined as a mild traumatic brain injury (mTBI); however, some researchers believe that this nomenclature is misleading- as not all mTBI are concussions. Rather, concussions are a distinct, less severe, subclass of mTBIs (Harmon, et al., 2013).

Prevalence

In the United States, it is estimated that over 1.6 million sport related concussions occur each year (Langlois, et al., 2006). A more recently published prevalence study estimates 1.1- 1.9 million sport and recreation related concussions occur in children $(\leq 18 \text{ years})$ in the United States (Bryan, Rowhani-Rahbar, Comstock & Rivara, 2017). This study was exclusive to children; however, by extrapolating logic presented in the discussion this study suggests 1.8-3.1 million sport-related and recreational concussion occur each year in the United States (Bryan, et al., 2017). The risk of concussion is present in almost every sport, with the highest prevalence in contact sports. Among American high school and collegiate sports, football accounts for 40.2% of reported concussions followed by girls soccer (21.5%), boys soccer (15.4%), and girls basketball (9.5%) (Gessel, Fields, Collins, Dick, & Comstock, 2007). When accounting for the number of athletes involved in each sport Zuckerman, et al. (2015) reported men's wrestling and ice hockey (both men and women) to have the highest rates of concussion in all NCAA sports. Additionally, higher rates of concussion are found in both youth (Noble & Hesdorffer, 2013) and female (Marar, Mcllvain, Fields & Comstock, 2012) athletic populations. Left undetected or mismanaged a concussion can cause potentially long-term effects.

Biomechanics

A concussion occurs as a result of deformation of the brain due the collision of the brain with the skull. Historically the term concussion referred to injuries that caused "brain shaking" (Shaw, 2002). According to Newton's third law, force equals mass multiplied by acceleration. Sufficient force, applied over a sufficient surface area, transferred via kinetic energy is responsible for the concussive strains and damage to the delicate brain tissue. The human brain is suspended in cerebral spinal fluid (CSF). The biomechanics of concussive injuries can be generalized into four main categories: linear forces (acceleration or deceleration), rotational forces, skull deformation, and whiplash (Shaw, 2002).

Acceleration and deceleration injuries are a result of impact (direct blow to the head) or impulse (force that sets head in motion without contact). Acceleration injuries that induce damage directly beneath the point of impact are referred to as coup injures (Ommaya & Gennarelli, 1974). Deceleration injures cause damage opposite to the site of impact. These are classified as contre-coup injuries (Ommaya & Gennarelli, 1974). Linear impact occurs when the head is struck while held stationary or the head strikes a stationary object causing acceleration/deceleration of the skull and subsequent brain movement (Broglio, et al., 2010). Rotational impacts occur when the head rotates in response to an angular blow to the head. Shearing and tensile forces at the junction of the cerebrum and the brainstem may be the resulting stresses of rotational force (Holborne, 1943). Less common in SRC, deformation

injuries, resulting in depression of the skull, causing propagation of waves through the CSF (Gurdjian, 1972). Finally, whiplash injuries result from sudden movements of the head about the cervical region causing propulsion of the brain within the skull.

Outside of controlled experimental conditions, mechanical forces inducing SRC are components of both linear and rotational forces. Despite the broad variety of injury mechanisms all SRC injuries involve a near instant method of kinetic energy transfer (Shaw, 2002). The most commonly reported injury mechanism for all NCAA sports was an outcome of player-on-player contact (Zuckerman et al., 2015). Despite efforts to identify force peak rotational/ linear acceleration thresholds responsible for inducing concussion (Greenwald, Gwin, Chu, & Crisco, 2008; Guskiewicz, & Mihalik, 2011; Pellman, Viano, Tucker, Casson, & Waeckerle, 2003) researchers have yet to identify thresholds (Post & Hoshizaki, 2015)

Pathophysiology

The underlying pathophysiology of concussion is comprised of a cascade of neurometabolic events. Defects within brain tissue cannot be seen on a macroscopic level, as a sport-related concussion is a functional injury occurring within individual neurons. Biomechanical forces cause neurons to become stretched or stressed, yielding reduced cerebral blood flow and ions imbalances (Barkhoudarian, Hovda, & Giza, 2011). In efforts to restore normal cerebral membrane potential, sodium-potassium $(K + /Na+)$ pumps work to efflux $K+$ and influx Na+ (Barkhoudarian, et al., 2011). The ion flux followed by neuronal suppression, also known as "spreading depression" predicted to be associated with early loss of consciousness, amnesia, or cognition deficits (Giza & Hovda, 2014). Additionally, resultant overdrive of the K+/Na+ pumps cause increased ATP utilization (Giza & Hovda, 2001).

Immediately following impact, increased glucose utilization is evident in the cortex and hippocampus of the injured brain (Rosenthal, LaManna, Yamada, & Somjen, 1979; Yoshino, Hovda, Tatsuro, & Becker, 1991). Hyperglycolysis and reduced blood flow increases anaerobic metabolism reliance. Eventually a metabolic mismatch occurs as the body is unable to supply the brain with sufficient energy. This mismatch paired with, lactic acid accumulation, decreased magnesium levels, free radical production, and inflammatory response is believed to cause the outward symptoms that we associate with concussion (Giza & Hovda, 2001; Kalimo, Rehncrona, Soderfeld, Olsson & Siesjo, 1981; McIntosh, Faden, Yamakami, & Vink, 1988). Moreover, mitochondrial oxidation capacity is reduced with decreased magnesium levels and calcium imbalances are also affected (Dominques & Raparla, 2014). Decreased mitochondrial levels exacerbate the energy crisis as magnesium functions to regulate mitochondrial membrane potential and ATP production. These biochemical markers have been studied in both animal and human subjects and are thought to have cumulative effects in repeat injuries (Giza & Hovda, 2001).

Signs and Symptoms

A concussion is a heterogeneous injury. Presentation of this injury varies between individuals. For years, loss of consciousness (LOC) was used to identify and diagnose concussion, however LOC is no longer used to diagnose or confirm SRC (Lovell, Iverson, Collins, McKeag, & Maroon, 1999; Ommaya & Gennarelli, 1974). Additionally, many studies report that less than 14% of individuals with a concussion will lose consciousness (Guskiewicz, Weaver, Padua, & Garrett, 2000; Lau, Kontos, Collins, Mucha, & Lovell, 2011; McCrea, et al., 2003). Other symptoms of concussion include: headache, nausea, vomiting, vestibular disturbances, dizziness, fatigue, sleep pattern disturbances, drowsiness, sensitivity to light and or noise, irritability, sadness, emotional, numbness or tingling, feeling slowed or foggy, difficulty concentrating, difficulty remembering, and visual problems (Collins, et al., 2014; Meehan, Pierre, & Comstock, 2010). Headache is the most commonly endorsed symptom (Guskiewicz, et al., 2000; Kontos, et al., 2012). In most cases (approximately 80%) symptoms will resolve within 3 weeks of the injury, but in some cases these symptoms can linger even longer (Lau, Kontos, Collins, Mucha & Lovell, 2011).

Factor analysis of post concussion symptoms (assessed using the Post Concussion Symptom Scale- PCSS) supports the use of symptom clusters to faction related symptoms of SRC(Kontos et al. 2012) . Results from an exploratory-factor analysis conducted by Kontos et al. (2012) proposes four distinct categories accounting for 58.3% of variance: cognitive-fatiguemigraine, emotional (affective), physical (somatic), and sleep-arousal. Similar anecdotal clinical guidelines suggests that symptoms occurring within the first seven days should be divided into primary and secondary symptoms (Collins, et al., 2014). This suggests that patients should be treated similar in the first seven days (Collins, et al., 2014). If symptoms persist beyond seven days, clinical trajectories are recommended to properly assess, track and treat the patient. Collins et al. (2014) recommends the use of six clinical trajectories: vestibular, ocular-motor, cognitive, post-traumatic migraine, cervical, anxiety/mood.

When assessing symptoms, clinicians should consider both age and sex of the individual. Research suggests that male and female athletes commonly experience different symptom trajectories (Covassin, Elbin, Harris, Parker, & Kontos, 2012). In a cohort of college and high school aged athletes Covassin and colleagues (2012) used a 4 (time) x2(sex) x2(age) repeated measures analysis of variance (ANOVA) to assess symptoms following injury. Younger athletes and females were more likely to have lower neurocognitive scores, and women reported more

symptoms across all time points (Covassin et al., 2012). In addition to increased symptom provocation, females also tend to present with more somatic and migraine-cluster symptoms (Covassin, Elbin, Harris, Parker, & Kontos, 2013; Frommer, et al., 2011).

Other research has explored on-field concussion symptoms and recovery prognosis and trajectory. Initially, LOC was thought to be a proxy of concussion severity, however the literature in the last 15 years has questioned the actual relevance of LOC (Lau, Lovell, Collins, Pardini 2009; Collins, Iverson, Lovell, McKeag, Norwig, Maroon, 2003). Rather focus has shifted towards identifying correlates to protracted recovery such as: retrograde/post-traumatic amnesia and dizziness(Lau, et al., 2011). In 2011, a study conducted by Lau and colleagues found on-field dizziness to be a predictor (Odd's Ratio $= 6.34$) of prolonged recovery. Similarly, dizziness has also been correlated to prolonged social impairments following concussion (Yang, Tu, Hua, & Huang, 2007). Chronic and sub-acute symptoms can cause student athletes to perform poorly in school, become socially withdrawn, and become depressed. The psychological components of recovery play major roles in rehabilitation (Wiese-Bjornstal, White, Russel, & Smith, 2015).

Nevertheless, preventing and treating long-lasting effects of concussion is the focus for many clinicians. These effects are more likely to occur if the individual returns to play without properly recovering from their concussion. Elbin and colleagues (2016) reported that athletes reportedly returning-to-play despite sustaining a concussion were 8.8 times more likely to have a protracted recovery lasting longer than 20 days. After adjusting for other predictors of protracted recovery (eg. age, sex, post-traumatic migraine) the risk of continuing to play with a concussion was exacerbated, resulting in an adjusted odds ratio of 14.2 (Elbin, et al., 2016).

Another risk associated with continuing to play with a concussion or returning to play too quickly is second impact syndrome. This is an extremely rare catastrophic brain injury that results in edema and a breakdown of the blood-brain barrier. This syndrome can occur when an athlete receives another concussive blow before fully recovering from the first concussion. Many researchers believe that the brain is more susceptible to concussion, when in the hypermetabolic stage (Laurer et al., 2001). Normal cellular response following concussion results in vasoconstriction, however during second impact syndrome this vascular regulatory function is impaired resulting in quickly diffusing edema (McCrory, 2001).The excessive edema can cause brainstem compression and hematomas (Le, & Gean, 2009). Onset of this syndrome can take less than 5 minutes to be in full effect (Reilly, 2001). For this reason, it is important to accurately detect, monitor, and fully rehabilitate concussions to prevent further damage.

Management Approaches for Sport-Related Concussion

After a suspected SRC, immediate removal from play is recommended (Elbin, et al., 2016; McCrory, et al., 2013). This recommendation is aimed to prevent successive SRC impacts and protect potentially compromised brain tissue. Concussions are heterogeneous injuries and may present in a variety of ways. A multidisciplinary approach is recommended in order to best account for the variety of presentations (Johnson, Kegel, & Collins, 2011; McCrory, et al., 2013). Four main facets of post-concussion evaluation aim to assess: neurocognitive function, vestibular-ocular function, balance performance, and symptoms. In more recent years efforts to promote objective measures of post-injury deficits have been endorsed in consensus statements; however, symptom assessments remain an essential cog in management of concussion (McCrory, et al., 2017).

Symptom reporting can be assessed in a more relaxed clinical interview format or in a structured intake form. The Post-Concussion Symptom Scale (PCSS) is an intake form used to assess self-reported symptoms. Twenty-two symptoms are scored on a 7-point Likert scale (0-6), these symptoms can be grouped into specific categories. In 2016, a study conducted by Elbin et al., compared methods of symptom reporting in a cohort of adolescent athletes. In a cohort of 54, symptom severity scores were significantly lower using an open clinical interview approach when compared to a guided clinical interview, a computerized symptom inventory, and parent reports (Elbin, et al., 2016).

Return-to-play (RTP) protocol is used to help integrate athletes safely back into his or her sport. Rest was once believed to be the best mechanism of treatment for concussed individuals but research shows that a slow progression both back into normal everyday encounters and physical activity is beneficial. The current RTP protocol endorsed and used by many athletic trainers and clinicians is a graduated five-step progression. Although return-to-play progression is standardized, decisions should be individualized, and progression can vary between athletes (Harmon, et al., 2013). Interestingly, over 27% of athletes who reported being symptom free after RTP exertion did not pass all of the neurocognitive tests (McGrath, et al., 2013). This is yet another reason why CNT is an important tool used in assessing concussion recovery.

Computerized Neurocognitive Testing

Neurocognitive testing has been increasingly useful in assessing concussion for the past 30 years, and is now considered a cornerstone in concussion management. Although the roots of cognitive testing lie in traumatic brain injury research, much of the USA's sport-related concussion testing can be attributed to J. T. Barth. In 1976 Barth began by using tests shown to detect deficits caused by mild head trauma, and complied a relatively brief test battery. This test

consisted of nine different cognitive function tests and took approximately 45 minutes to administer. Barth and colleagues (1983) collected "baseline" data from over 2,300 football players around the nation. In the event of a concussion, the same battery was administered after 24 hours, five days, and ten days post-concussion. This study found significant differences between baseline and concussed players at both 24 hours and five days post-concussion. This study helped create a foundation for the use of neurocognitive and neuropsychological testing in the diagnosis of concussion. It is recommended that all athletes sustaining a concussion have a neurological evaluation during the management of their injury (McCrory, et al., 2013)

With the advancement in technology, researchers developed computerized neurocognitive tests. These tests are an essential component of current concussion management (McCrory, et al., 2013). CNT are more sensitive to fractional reflex delays and also provided a more economical and useful testing method as compared to the paper and pencil tests. CNT has been validated by numerous researchers as a reliable measure of cognitive function (Van Kampen, et al., 2006).Computerized Neurocognitive testing is best administered in a prospective and retrospective method. Prospective baseline tests are administered before an athlete begins contact activities and gives a snapshot of an individual's cognitive function. After a suspected concussion the battery is repeated and the results are compared. Cognitive impairment, slower reaction times, and lower composite scores calculated by the CNT are indications of a concussion.

Factors Effecting Computerized Neurocognitive Testing

There are several factors that affect computerized neurocognitive testing. First, the athlete's motivation has been shown to affect the outcome of the test (Bailey & Arnett, 2006). ImPACT has an internal validity indicator that helps to red-flag scores that may be a result of an athlete not giving their best effort during the test. Even with the validity measure it is difficult to assess the individual's motivation and effort. Other factors such as sex (Covassin, et al., 2012), age (Covassin, et al., 2012), learning disabilities (Elbin et al., 2013), sleep quality (Mihalik et al., 2013), and concussion history (Broglio et al., 2014) can negatively skew the CNT results. Another factor that can negatively affect the results of CNT is exertion. Both cognitive fatigue and physiological fatigue is shown to have significant effects on cognition (Covassin, et al., 2007; Sufrinko, Johnson, & Henry, 2016).

Despite several factors that have shown to affect the accuracy of CNT, these test are useful in objectively assessing the cognitive effects of a concussion. Research shows that when computerized neuropsychological testing is used, in evaluation of HS athletes, individuals are less likely to return to play pre-maturely (Meehan, et al., 2010). Clearly, CNT is a useful tool for concussion diagnosis

Exertion Effects on Cognition

The relationship between exercise and cognitive performance has been studied, and theories used to explain the mechanism responsible for the interaction of exercise and cognition have evolved since the mid 1900's. Unfortunately, research has yielded different conclusions and much controversy exists concerning facilitative or detrimental effects of exercise on cognition.

The theoretical underpinnings of acute exercise and its interaction with cognitive function evolved from an "inverse-U effect" model rooted in cognitive psychology. In 1973, a cognitive psychologist theorized that acute exercise was similar to a psychological stressor and its effect on cognition (Davey, 1973). According to the Yerks and Dodson law (1908) as arousal increases performance also increases until the critical point in which too much arousal causes decreased performance. This relationship when plotted appears as an inverted U. Extreme low and high

arousal/stressful environments facilitates minimal performance, while moderate arousal/stressful environments facilitates maximal performance. This theory was also supported by Cooper in 1973, however the mechanism explaining the inverse-U effect was rooted in neurobiology. Cooper proposed that increasing exercise intensity increases catecholamine concentration in the blood plasma; increased blood plasma concentrations of catecholamines increases dopamine and noradrenalin neurotransmitters; noradrenalin increases arousal in the reticular formation, and arousal facilitates better performance. However, Cooper proposed that high intensity exercise elicited too much arousal, creating "noise" that would interfere with performance (Cooper, 1973).

The inverse-U relationship between exercise intensity and cognition was believed to explain this complex relationship for many years. Many scientists supported Cooper and Davey's inverse-U model. In 1983, scientist reported plasma concentrations of neurotransmitters adrenaline and noradrenalinin rise exponentially with maximal graded exercise tests (Green, Hughson, Orr, & Ranney, 1983). Additionally, other researchers found that a critical threshold for catecholamines occurs near 75% of an individual's VO2max (Podolin, Munger & Mazzeo, 1991). This critical threshold would correlate to the peak of the inverse-U.

However, the relationship between acute exercise and cognition is dynamic, and can differ depending on intensity of exercise, mode of exercise, aspect of cognition assessed, and recovery time following a bout of exercise in 1976, Wrisberg and Herbert found that physical fatigue is a performance variable and that different fatigue mechanisms could result from different types of exercise. Much of the research concerning cognition and exercise is centered on moderate intensity exercise as a mechanism to enhance or "arouse" (Nada et al., 2013). Other studies have shown that physical activity, when completed to exhaustion, results in negative

cognitive effects (McMorris & Hale, 2012). Although all of these studies have important implications, the negative effects of exertion found in literature has a more important hold on concussion neurocognitive testing. High intensity and maximal exercise has been shown to negatively impact neurocognitive function (Covassin, et al., 2007; Whyte, Gibbons, Kerr, & Moran, 2014).

In Covassin's (2007) study baseline CNT was administered to all participants. The treatment group completed a $VO₂$ max treadmill test and then immediately took ImPACT a second time. The control group remained at rest for 15 min (the approximate time it takes for completion of a VO2 max treadmill test) and also took ImPACT a second time. Means and standard deviations were calculated and the level of significance was set $(p=0.05)$. Significant decreases were seen in verbal memory, specifically immediate recall, and delayed recall. Teasing apart neurocognitive deficits caused by concussion or exhaustion is nearly impossible. The results of this study imply that CNT should not be administered immediately after maximal exertion for the most accurate CNT results. Clearly, exertion has effects on cognition and these detriments could negatively impact the validity of CNT baseline scores. Since the early 2000's other theoretical models have been developed to explain the interaction between acute exercise and cognitive function.

Transient Hypofrontality Theory

One theory that researchers have proposed to explain the negative effects of exertion on cognition is the transient hypofrontality theory. The transient hypofrontality theory argues that higher levels of cognitive function are impaired as a result of exercise (Dietrich, 2006). The brain receives a constant supply of nutrients and oxygen despite an increase in cardiac output due to exercise (Ide & Secher, 2000). During exercise, blood carrying oxygen and nutrients is directed

towards the working muscles as a smaller percentage of blood is allocated for the brain (Ide & Secher, 2000). Exercise and movement of large muscle groups requires a significant amount of neural stimulation. This increase in stimulation results in depressed prefrontal cortex functioning, therefore a decrease in higher level cortical functioning. During exercise utilities needed for higher-level executive control are depressed.

The transient hypofrontality hypothesis suggests that higher-level cognitive processing requiring prefrontal cortex activation are temporarily impaired during and immediately after exercise (Dietrich, 2006). This theory has been further supported by several other studies showing that exercise impairs executive functioning and response inhibition (Audiffren, Tomporowski, & Zagrodnik , 2009; Davranche & McMorris, 2009; Mahoney, Hirsch, Hasselquist, Lesher, & Lieberman,2007; Pontifex & Hillman 2007). A study by Del Giorno and colleagues (2010) also proposed that transient hypofrontality might also be responsible for decreases in performance after exercise is terminated. They found that executive control measures remained impaired for a significant amount of time post exercise, potentially until the brain has time to return to homeostasis (Del Giorno, Hall, O'Leary, Bixby, & Miller, 2010).

This theory supports the idea that exercise, causing significant exertion, can potentially depress cortical functioning. Higher level cortical functioning is responsible for several aspects of cognition, including those measured by CNT. This theory helps explain why baseline scores may be depressed after maximal exertion. According to this theory, the appropriate timeline for administering CNT after maximal exertion depends on the amount of time it takes the brain to return to normal function and homeostasis.

VO² Maximal Testing as a Measure of Exhaustion

According to anecdotal reports baseline testing is often worked in and around games, practices, and conditioning, potentially leaving student athletes in an exerted state. In 1923 Hill and Lupton defined $VO₂$ max as the maximal oxygen uptake an individual could elicit during maximal exercise. Maximal exertion can be measured several different exercise types including using a cycle ergometer or treadmill. A maximal $VO₂$ test is confirmed with physiological values. These include: a plateau in oxygen consumption with increasing workload, a heart rate greater than or equal to 10-12 beats below their age-estimated max (220-age), and a respiratory exchange ratio greater than 1.05- 1.15 (Beams & Adams, 2014). Additionally, rating of perceived exertion greater than 18 on a 6-20 scale is a confirmatory factor used (Riebe, Ehrman, Ligouri, & Magal, 2017).

Several protocols for eliciting maximal $VO₂$ exercise intensity have been proposed and are widely used to assess cardio-respiratory performance. It is widely accepted that normal populations perform 5-10% better using treadmill protocols when compared to cycle ergometers. Test duration ranging from 8-12 minutes is supported by several studies to elicit best performance (Buchfuhrer, et. al., 1983; Yoon, Kravaitz, & Robergs 2007). When using graded exercise tests with standardized starting intensities and pre-set incremental stages- variations in aerobic fitness and strength may result in test durations outside of the 8-12 minute window. Additionally, Mauger and Sculthrope (2001) reported self-paced VO² max protocols elicit higher VO² max values.

Methods

Research Design

This was a random cross over, repeated measures design study.

Participants

A convenience sample of healthy, university students (18-26 yrs.) were recruited for the study. All participants were required to be moderately active or vigorously active according to the International Physical Activity Questionnaire-Long Form and deemed healthy enough to complete a maximal oxygen consumption (VO $_2$ max). Any participant with diagnosed learning disability, ADHD, psychological disorder (e.g., clinical depression/anxiety), history of substance abuse, non-English speaking, or reported sustaining a concussion within the last six months was excluded from participating in this study.

Instrumentation/Measures

Pre-participation evaluation measures.

Medical History Questionnaire. As part of the initial screening process, individuals were required to fill out a medical history questionnaire, provided by the University of Arkansas Exercise Research Center. This intake form is comprised of 29 questions. Participants were asked to answer questions concerning their medical history. Answers were reviewed by a certified athletic trainer to ensure that participants did not have any pre-existing conditions or injuries that would make a graded exercise test high risk. Individuals not cleared for participation were referred to a medical doctor to seek clearance before enrolling in the study. Individuals that did not complete the required follow-up were excluded from participation. The medical history questionnaire is provided in Appendix C.

International Physical Activity Questionnaire Long Form. The International Physical Activity Questionnaire Long Form (IPAQ) is a validated 7-day recall assessment. Intended to be used in populations age 18-65, this form quantifies physical activity performed during work, activities of daily living, and leisure (Craig, et al., 2003). Activities are classified by intensity (vigorous, moderate, low) and type (leisure, work-related, activities of daily living, etc.). Responses are quantified and summed using metabolic equivalents (METS), activity duration, and frequency. A data analysis instruction provided by the IPAQ website includes formulas and standards used to classify participants as low, moderate or highly active (Patterson, 2005). The IPAQ-long is provided in Appendix D. Classification and data truncation methods are provided in Appendix E.

Pre-trial compliance assessments/measures.

Hydration status. Urine specific gravity (USG) was accessed via a spot sample. A small amount of urine is analyzed using a refractometer as a proxy of hydration. Urine specific gravity is a convenient and non-invasive method used clinically to assess hydration. A USG >1.025 has been shown to have a specificity of 91%, and sensitivity of 89% in detecting dehydration (Cheuvront, Ely, Kenefick, & Sawka 2010).

24-Hour History Intake Form. In order to ensure pre-test compliance was followed, a 24- Hour Intake Form was used. Participants were asked to report hours of sleep, fluid intake, physical activity, and when they last consumed caffeine, OTC drugs, supplements, or alcohol and their overall rating of how they feel. The 24-Hour History Intake form is provided in Appendix F.

24-Hour Diet Record. Participants were asked to complete a diet record documenting all food ingested within 24 hours of the trial. Participants were encouraged to maintain similar

eating habits prior to each testing session. Each diet log was analyzed using Nutritionist Pro software (Axxya Systems, 2018). A blank diet record is provided in Appendix G.

The maximal **graded VO² max treadmill test and associated measures.**

VO² max protocol. Participants performed a maximal graded exercise test to determine maximal oxygen consumption ($VO₂max$). Participants were asked to run on a treadmill while equipped with headgear used to facilitate breathing in room air and exhaling into a Hans-Rudolph mouthpiece. The mouthpiece was connected via a flexible plastic tube to a calibrated metabolic cart (ParvoMedics, Sandy, UT). To ensure all expired air was collected, participants wore a nose clip. The treadmill speed was initially set at a slow speed with no incline (approximately 60% the participants "all out" mile pace). Intensity of exercise was increased every 2 minutes by increasing the grade of the treadmill by 2%. The protocol used for this study was modified from the Arizona State University protocol (George, 1996). The test was continued until volitional exhaustion was reached. The data intake form used during the maximal graded exercise test is provided in Appendix H. Verbal encouragement cues used during the exercise test are provided in Appendix I. Additionally, intake forms used during recovery are provided in Appendix J.

Heart rate. The participant was fitted with a Blue-tooth equipped heart rate monitor (*Polar*FT1). Heart rate was assessed before changing stages throughout the maximal graded exercise test. Additionally, the participant's heart rate was recorded following 1 minute of recovery, and at the start and end of each CNT.

Rating of perceived exertion (RPE). The Borg (6-20) scale was used in this study (Borg, 1970). During the maximal graded exercise test participants were asked to rate their perceived exertion approximately 15-seconds before each 2 minute stage ended, and immediately following exercise test termination. Since participants were equipped with mouth gear, RPE was indicated by participants by pointing to a number on the Borg RPE scale.

Respiratory exchange ratio (RER). Respiratory exchange ratio is a measure of metabolic function during exercise. This value is calculated by the metabolic cart, and is used as a determinate of maximal exercise test qualification. Respiratory exchange ratio is the proportion of expired carbon dioxide volume to inhaled oxygen.

VO2peak. VO² peak was determined by analyzing metabolic cart output data. The metabolic cart was configured to analyze expired air every 15-seconds. The $VO₂$ peak was defined as the highest 15-second relative $VO₂$ value.

 VO_2 *last-minute average.* VO₂ last-minute average was calculated using the metabolic cart output data. This value was defined as the average of the relative $VO₂$ values during the last minute of the exertion test.

Neurocognitive and Symptom Assessments.

CNT. Computerized neurocognitive performance was measured using The Immediate Post Concussion Assessment and Cognitive Testing (ImPACT). The ImPACT battery takes approximately 20-25 minutes to complete and is comprised of three sections: demographics, post concussion symptom scale, and neurocognitive tasks. ImPACT has five test versions, different test versions were used in each trial to minimize practice effects. After completion of the assessment, ImPACT generates outcome composite scores for the cognitive domains of verbal memory, visual memory, processing speed, and reaction time (Iverson, Lovell, & Collins, 2003). The ImPACT battery has demonstrated acceptable validity and reliability over 8 days across 4 administrations, yielding correlation coefficients ranging from .62 to .88 for outcome scores

(Iverson, et al., 2003). Additionally, the sensitivity and specific of this measure is tool, 81.9% an 82.4%, respectively (Schatz, et al., 2006).

Post-concussion Symptom Scale. The post-concussion symptom scale is composed of 22 symptoms ranked on a 7-point Likert scale (0= "not experiencing a given symptom" and 6= "severe"), reliability and validity of this measure is supported in several studies (Lovell, et al. 2006; Kontos et al., 2012). Total symptom score is the sum of all 22 symptom reports. Additionally, a supplementary analysis of symptom clusters was examined trends in symptom reporting. Symptom clusters group PCSS symptoms into four domains: cognitive-sensory, affective, sleep-arousal, and vestibular-somatic symptoms (Kontos, et al., 2012).

Self-Reported Effort Assessment.

Effort Form. An effort form created by the researchers was used to assess participant's effort during the trial, and confirm physical activity level. The form was given to the participant immediately following the completion of the CNT. The effort form consisted of a 4-point likert scale $(1 = "No Effect"$ and $4 = High Effect"$ and is provided in Appendix K.

Procedure

This study obtained IRB approval (Appendix L) and all participants provided informed consent. Each participant completed four separate experimental trials (baseline, immediate recovery, 10-minute recovery, and 20-minute recovery) order was randomized and counterbalanced. During the baseline visit, the participant did not participate in any type of exercise protocol and only took ImPACT. The remaining trials required participants to complete a maximal graded $VO₂$ max treadmill test followed by a timed rest interval [immediate (<2minute), 10-minute, or 20-minute] before taking ImPACT.

Before beginning an experimental trial, pre-test compliance was confirmed by examining the 24-hour Diet Record, the 24-Hour History Intake form, and analyzing a urine spot sample. In order to participate, individuals needed to be well hydrated (USG \leq 1.025). Additionally, participants were not permitted to consume caffeine or exercise within 12-hours, or intake OTC drugs within 48-hours prior to the start of the trial. During the first $VO₂$ max trial, participants self-select the treadmill belt speed. To encourage equivalent exertion protocols this speed was used for all successive tests. Exercise intensity was increased incrementally by increasing the percent gradient by 2% every 2 minutes. Throughout maximal graded $VO₂$ max treadmill test, researchers assessed heart rate, RPE, RER, and other physiological markers of maximal exertion. Heart rate, RPE, RER, and measured VO2 was recorded at the end of each 2 minute stage. Participants were verbally encouraged during the VO2 max test to elicit best performance. Verbal encouragement cues used throughout the exertion protocols are provided in Appendix I. The $VO₂ max/peak$ was confirmed using 2 of the 4 criteria: as a plateau in oxygen consumption, heart rate \ge estimated maximal heart rate (220-age) +/- 10 bpm, RPE \ge 17, or a respiratory exchange ratio (RER) \geq 1.1. Volitional exhaustion was assumed when the participant could no longer keep up with the treadmill, or the participant indicated they wished to stop.

Immediately following termination of the maximal graded $VO₂$ max treadmill test recovery time was started, and the participant was escorted off the treadmill into the CNT testing room. Heart rate during the recovery period was recorded: one minute after cessation of exercise and at the end of the recovery interval. During the recovery interval participants were asked to sit quietly, and were prohibited from reading, using cellular devices, or walking. Once the maximal graded $VO₂$ max treadmill test, the recovery interval and ImPACT administration was complete, each participant was asked to rate their performance effort.

Data Analysis

All statistical analyses were conducted with SPSS (IBM Corp., 2016).

Inspection of data for accuracy and completeness. The data was inspected by the researcher for outliers, and completeness.

Examination of normality. Normality was examined in outcome variables using a Shapiro-Wilks test, significance was set at $p=0.05$. Additionally, skewness and kurtosis of the results was examined.

Examination of sphericity. Sphericity was examined using Mauchly's test of sphericity, significance was set at $p=.05$.

Describing the sample.

Descriptive statistics (means and standard deviations) were used to describe the sample based on age, height, and weight. Additional descriptive information was analyzed using frequencies (concussion history and IPAQ rating of physical activity).

Examining test condition equivalence.

To examine equivalence of test condition s based on pre-trial compliance assessments/measures a series of repeated measures analysis of variance (ANOVAs) were performed on hydration status (USG), and components of the 24-Hour History Intake Form and 24-Hour Diet Record (i.e. duration of previous night's sleep, rating of overall feeling, and 24 hour total caloric intake). Additional repeated measure ANOVAs were conducted to examine equivalence of the maximal graded VO² max treadmill test outcomes across all trials with an maximal exertion intervention preceding ImPACT administration (i.e. RER, RPE, VO_{2peak} , $VO₂$ last-minute avg., duration of exercise, maximum heart rate, and heart rate 1 minute post exercise). Lastly, a repeated measure ANOVA will be used to examine effort across all conditions based on
likert-scale reports from the Effort Form. Bonferroni-corrected post-hoc analysis was used to control for multiple comparisons for all repeated measures ANOVAs; level of significance was set to ($p < .01$).

Heart rate recorded at the start and end of CNT.

Two repeated measures ANOVAs were performed on heart rates recorded at the start and end of CNT. The independent variables was defined as the recovery interval [baseline, immediate (<2-minute), 10-minute, or 20-minute], and the dependent variable was heart rate (bpm). Bonferroni-corrected post-hoc analysis was used to control for multiple comparisons; level of significance was set to $(p \leq .01)$.

Evaluation of hypotheses 1-5: CNT outcomes and symptom reports.

Data analysis for hypothesis 1: Verbal memory composite scores will be significantly lower than baseline at the immediate recovery interval, but not at the 20-minute recovery interval. A repeated measures ANOVA was performed on verbal memory composite scores. The independent variables was defined as the recovery interval [baseline, immediate (<2 minute), 10-minute, or 20-minute], and the dependent variable was the ImPACT verbal memory composite score. Bonferroni-corrected post-hoc analysis was used to control for multiple comparisons; level of significance was set to $(p \le 0.01)$.

Data analysis for hypothesis 2: Visual memory composite scores will be significantly lower than baseline at the immediate recovery interval, but not at the 20-minute recovery interval. A repeated measures ANOVA was performed on visual memory composite scores. The independent variables was defined as the recovery interval [baseline, immediate \langle -minute), 10minute, or 20-minute], and the dependent variable was the ImPACT visual memory composite

score. Bonferroni-corrected post-hoc analysis was used to control for multiple comparisons; level of significance was set to $(p \leq .01)$.

Data analysis for hypothesis 3: Processing speed composite scores will be significantly lower than baseline at the immediate recovery interval, but not at the 20-minute recovery interval. A repeated measures ANOVA was performed on processing speed composite scores. The independent variables was defined as the recovery interval [baseline, immediate (<2 minute), 10-minute, or 20-minute], and the dependent variable was the ImPACT processing speed composite score. Bonferroni-corrected post-hoc analysis was used to control for multiple comparisons; level of significance was set to $(p \le 0.01)$.

Data analysis for hypothesis 4: Reaction time will be significantly faster than baseline at the immediate recovery interval, but not at the 20-minute recovery interval. A repeated measures ANOVA was performed on reaction time. The independent variables was defined as the recovery interval [baseline, immediate (<2-minute), 10-minute, or 20-minute], and the dependent variable was the reaction time scores. Bonferroni-corrected post-hoc analysis was used to control for multiple comparisons; level of significance was set to $(p \le 0.01)$.

Data analysis for hypothesis 5: Total PCSS scores will be significantly higher than baseline at the immediate recovery interval, but not at the 20-minute recovery interval. A repeated measures ANOVA was performed on total PCSS scores. The independent variables was defined as the recovery interval [baseline, immediate (<2-minute), 10-minute, or 20-minute], and the dependent variable was the total PCSS score. Bonferroni-corrected post-hoc analysis was used to control for multiple comparisons; level of significance was set to $(p \leq .01)$.

Results

Participant Recruitment

A total 55 people responded to the posted flyers (Appendix A) by emailing the researcher. An additional 16 people expressed interest in the study and contacted the researcher directly. The researcher responded using form email to provide individuals a brief synopsis of the protocol and a list of inclusion and exclusion criteria. A copy of the form email is provided in Appendix B. Individuals, who were still interested, were instructed to set up a meeting to discuss specific requirements and fill out additional screening forms. A total of 37 individuals filled out screening forms, two individuals were not cleared to participate without a comprehensive medical exam and opted to not participate. Additionally, five participants did not complete all visits yielding an overall attrition rate of 14% (5/35). These individuals are not included in the analysis.

Participant Demographics

The final sample included 30 college-aged participants, with ages ranging 18 to 26 years $(M=21.87 \pm 2.29)$. The majority (63.3%) of participants were male (19/30), and the remaining 36.7% of the sample were female. Seventy-percent (21/30) of the sample were categorized as highly active according to the IPAQ. Demographics of the final sample, and subgroups based on sex are provided in Table 1.

Table 1.

	Total Sample			Males	Females		
	\boldsymbol{M}	SD	\boldsymbol{M}	SD	M	SD	
Age	21.87	2.29	22.11	2.49	21.45	1.92	
Height (cm)	175.5	9.5	179.8	8.5	167.9	5.9	
Weight (kg)	72.5	12.4		77.6 11.4		9.0	
	Frequencies and Percentages						
IPAQ rating							
Moderate	$(9/30)$ 30.0%			$(5/19)$ 26.3%		$(4/11)$ 36.4%	
High		$(21/30)$ 70.0%		$(14/19)$ 73.7%	$(7/11)$ 63.6%		
History of Concussion							
Yes	$(5/30)$ 16.7%		$(4/19)$ 21.1%		$(1/11)$ 9.1%		
N _o	$(25/30)$ 83.3%		$(17/19)$ 79.9%		$(10/11)$ 90.9%		

Demographics of the total sample (N=30), males (n=19) and females (n=11).

Examining Test Condition Equivalence

A series of one-way repeated measures ANOVAs were conducted to compare pre-trial compliance assessment/measures and Self-reported Effort for each test administration. Hydration status assessed using spot-sample USG was not significantly different between trials, (*p =* .811, *Wilks* $\lambda = .97$ *, F* [3,27] = .32, $\eta^2 = .034$). Sleep duration the night before each trial was not significantly different ($p = .11$, *Wilks* $\lambda = .75$, $F [3,27] = 2.28$, $\eta^2 = .255$). Subjective rating of overall feeling was not significantly different ($p = .188$, *Wilks* $\lambda = .83$, *F* [3,26] = 1.72, $\eta^2 =$.166). Total 24-hour caloric intake was not significantly different between trials (*p =* .274, *Wilks* $\lambda = .87$, *F* [3,27] = 1.37, $\eta^2 = .132$). Self-reported effort assessed after the completion of each test session was not significantly different between trials ($p = .409$, *Wilks* $\lambda = .90$ *, F* [3,27] = 1.0, η^2

= .103). Means and standard deviations for each trial are presented in Table 2.

Table 2.

Mean and standard deviations for Pre-Trial Compliance Assessments/Measures and Selfreported Effort Assessment, (N=30).

	Baseline		Immediate		10-minute		20-minute	
Measure	M	<i>SD</i>	M	<i>SD</i>	M	<i>SD</i>	M	<i>SD</i>
Hydration Status (USG)	1.011	.007	1.012	.006	1.012	.008	1.011	.007
Duration of Previous Night's Sleep (hrs) 7.37			.91 6.96	.96	7.22	1.07	7.52	-1.19
Subjective Rating of Overall Feeling ^a	3.4	1.2	3.5	1.1	3.2	1.0	3.21	-1.2
24-hour Caloric Intake (kcal)	1935	775	1884	869	1877	883	2185	1054
Self-Reported Effort ^b	3.9	.26	3.90	.31	3.90	.31	3.97	.17

Note. ^a Subjective Rating of Overall Feeling was reported by selecting 1 of 9 choices. These choices were organized in descending fashion and coded; $1 =$ "excellent" and $9 =$ "terrible." bSelf-Reported Effort. **p* < .01

The results of a series of one-way repeated measures ANOVAs for graded maximal VO2 treadmill test outcomes were conducted to compare exertional trials. No significant differences were observed between the immediate, 10-minute, and 20-minute test outcomes during the graded maximal $VO₂$ treadmill protocol (RER, RPE, $VO₂$ _{peak}, $VO₂$ _{last minute avg.,} duration of exercise, maximum heart rate, and heart rate 1 minute post-exercise).

The peak respiratory exchange ratio at volitional exhaustion was not significantly different between trials ($p = .462$, *Wilks* $\lambda = .95$, $F [3,27] = .793$, $\eta^2 = .054$). The subjective rating of perceived exertion (RPE) at the end of the treadmill protocol was not significantly different between trials ($p = .655$, *Wilks* $\lambda = .97$, $F [3,27] = .430$, $\eta^2 = .030$).

The VO_{2peak} (ml/kg/min) value based on 15-second interval measurements was not significantly different between trials, ($p = .452$, *Wilks* $\lambda = .95$, $F [3,27] = .818$, $\eta^2 = .055$). The VO₂ last minute average measurements were not significantly different ($p = .889$, *Wilks* $\lambda = .99$ *, F* $[3,27] = .118$, $\eta^2 = .008$). The duration of exercise was not significantly different between exertion trials ($p = .871$, *Wilks* $\lambda = .99$, $F [3,27] = .138$, $\eta^2 = .010$).

Maximum heart rate was not significantly different between exertion trials (*p =* .941, *Wilks* $\lambda = 1.0$ *, <i>F* [3,27] = .061, $\eta^2 = .004$). Heart rate observed 1 minute after stopping the treadmill belt was not significantly different ($p = .208$, *Wilks* $\lambda = .89$, *F* [3,27] = .1.66, $\eta^2 =$.106). Means for each trial are presented in Table 3.

Table 3.

Mean and standard deviations for test outcomes of the graded maximal VO² treadmill protocol, $(N=30)$.

	Immediate		10-minute		20-minute	
Measure	\boldsymbol{M}	<i>SD</i>	\boldsymbol{M}	SD	M	<i>SD</i>
RER	1.11	.04	1.11	.04	1.12	.04
RPE (Borg 6-20 Scale)	19.1	1.1	19.2	1.0	19.3	.8
VO2peak (ml/kg/min)	53.93	9.43	54.68	10.13	53.98	10.13
VO2 last minute avg. (ml/kg/min)	52.30	9.52	51.87	11.56	52.40	9.74
Duration of Exercise (minutes)	13.5	2.1	13.7	2.5	13.6	2.2
Maximum Heart Rate (bpm)	192.33	8.48	192.37	7.90	192.00	7.63
Heart Rate 1 minute post exercise	155.60	20.35	152.60	14.91	156.57	12.29

Note: $* = 01$; $* =$ Different from Baseline; $\frac{b}{c}$ Different from Immediate; $\frac{c}{c}$ Different from 10 min; $\frac{d}{c}$ Different from 20 min

Heart Rate Recorded at the Start and End of CNT

Significant within-subject effect was observed for heart rate taken at the start (*p*=.00, *Wilks* $\lambda = .04$ *, F* [3,27] = 241.4, $\eta^2 = .96$) and finish ($p = .000$, *Wilks* $\lambda = .07$, *F* [3,27] = 116.2, η^2 = .928)of CNT. Post-hoc analysis revealed significant differences between baseline pre-test HR when compared to immediate [t(29)= -20.6, *p=* .00], 10-minutes [t(29)= -23.2, *p=* .00], and 20minute [t(29)= -9.6, *p=* .00] post-exertional recovery trials. Significant differences between pretest heart rate during the immediate trails when compared to 10-minutes [t(29)= 10.5, *p=* .000] and 20-minutes [t(29)= 7.7, *p=* .000]. No significant difference between pre-CNT heart rate taken during the 10-minute trial and 20-minute trial was observed [t(29)= .05, *p=* 1.0]. Post-hoc analyses of heart rate taken after the completion of CNT reveled significant differences between the baseline trial when compared to immediate $[t(29)= -11.3, p=.000]$, 10-minutes $[t(29)= -11.3, p=.000]$ 16.02, *p=* .000], and 20-minute [t(29)= -15.4, *p=* .000] post-exertional recovery trials. Additionally, the immediate recovery trial post-CNT heart rates were significantly different from the 20-minute recovery trial $[t(29)=4.1, p=.002]$. Means for each exertion trial provided in Table 4.

Evaluation of Hypotheses 1-5: CNT Outcomes and Symptom Reports

Normality and sphericity was examined for all CNT composite scores, and symptom scores. Verbal memory scores violated assumptions of normality, however assumptions of sphericity was met (Mauchly's Test of sphericity, $p=74$), skewness values ranged (-2.3–-.66), kurtosis values ranged (-.48–6.0). Visual memory scores violated assumptions of normality at the baseline and 10-minute recovery interval, however assumptions of sphericity was met (Mauchly's Test of sphericity, $p=11$), skewness values ranged (-.92–-.22), kurtosis values ranged (-.73–.46). Processing speed scores violated assumptions of normality at the immediate, 10-minute, and 20-minute recovery interval, assumptions of sphericity was met (Mauchly's Test of sphericity, $p=19$), skewness values ranged (-1.1–-.59), kurtosis values ranged (-.27–1.3). Reaction time violated assumptions of normality at the 10-minute and 20-minute recovery interval, assumptions of sphericity was violated (Mauchly's Test of sphericity, p=.01), skewness values ranged (-.29– 1.1), kurtosis values ranged (-.49–1.7). PCSS total scores violated

assumptions of normality, assumptions of sphericity was also violated (Mauchly's Test of sphericity, $p=.00$), skewness values ranged (1.2–2.3), kurtosis values ranged (-.74–5.7).

The results from a series of repeated measures ANOVA, examining hypothesized differences in CNT composite scores based on the duration of the recovery interval is provided below. There was no significant effect based on recovery interval for verbal memory (*p*= .29, *Wilks* $\lambda = .87$ *, F* [3,27] = 1.31, $\eta^2 = .13$). There was no significant effect based on recovery interval for visual memory ($p = .021$, *Wilks* $\lambda = .70$, $F[3,27] = 3.81$, $\eta^2 = .297$). There was no significant effect based on recovery interval for reaction time ($p=29$, *Wilks* $\lambda = .87$ *, F* [3,27] = 1.32, η^2 = .13). However, a significant effect based on recovery interval processing speed composite scores was observed ($p=0.01$, *Wilks* $\lambda = .66$, $F[3,27] = 4.69$, $\eta^2 = .34$). Post-hoc paired sample t-tests revealed significant improvements in processing speed composite scores between baseline and the 20-minute rest trial $[t(29) = -2.21, p = .006]$. Means for each exertion trial provided in Table 4..

Results from a repeated measures ANOVA revealed significant differences based on recovery interval for total PCSS scores (p =.00, *Wilks* λ = .60, *F* [3,27] = 5.9, η ² = .40) between trials. Post-hoc paired samples t-test revealed significantly higher total symptom scores following all graded maximal $VO₂$ treadmill tests compared to baseline pre-test symptom scores: immediate [t(29)= -4.17, *p*= .002], 10-minute rest [t(29)= -3.24, *p*= .018] and 20-minute rest [t(29) = -3.41 , $p = .011$]. Means for each exertion trial provided in Table 4.

Table 4.

Baseline Immediate 10-minute 20-minute Measure *M SD M SD M SD M SD* HR at Start of CNT test* 62.67^{*b,c,d*} 10.96 142.87^{*a,c,d*} 23.16 102.50^{*a,b*} 11.44 102.33a,b *a,b* 20.08 HR at End of CNT test* 63.60 b,c,d *b*,*d* 103.37^{*a,d*} 19.54 94.33^a 10.77 92.17*a,b* 12.43 Verbal Memory Composite Score 92.87 7.74 90.46 8.00 92.40 7.84 93.53 6.35 Visual Memory Composite Score 82.13 10.47 79.93 11.32 78.40 7.83 75.53 13.11 Processing Speed Composite Score* 44.61 *d* 5.25 45.86 5.50 46.27 5.08 46.61^a 5.68 Reaction Time (sec) 0.61 0.09 0.59 0.09 0.61 0.10 0.60 0.10 Total PCSS Symptom Score* 2.97 *b,c,d* 4.11 11.10 *a,d* 11.56 7.53^a 9.86 $5.70^{a,b}$ 6.42

Mean and standard deviations for CNT scores and physiologic data for all trials, (N=30).

Note: *N*=30; *=.01; ^a= Different from Baseline; ^b= Different from Immediate; ^c=Different from 10 min; $d=$ Different from 20 min

Discussion

General Discussion of Findings

The main purpose of this study was to identify the appropriate recovery interval needed following maximal exertion before administering baseline computerized neurocognitive tests. Baseline CNTs provide clinicians significant information useful for diagnosing and managing concussions. When administered in an appropriate setting, CNT baseline assessments can provide a personalized record of an individual athletes healthy neurocognitive function. However, if baselines CNTs are not administered in settings eliciting best performance, decreased performance may imitate poor neurocognitive capacity. When CNT is administered post-SRC and scores are compared to an artificially decreased baseline, concussions may go undiagnosed, and/or the athletes may be returned to play before fully recovered. It is important to identify best-practice guidelines for administration of baseline CNT used in managing and diagnosing SRC.

Results from this study did not support the necessity of a recovery interval following maximal exercise to elicit accurate neurocognitve composite scores. Hypothesis 1-4 was rejected; no significant deficits in cognitive performance were exhibited following the graded maximal exertion treadmill test. Additionally, processing speed performance was significantly better during the 20 minute rest interval trial compared to baseline. Results from this study did support hypothesis 5; total symptom scores following maximal exercise were significantly higher than symptom scores at baseline.

These results suggest that neurocognitive composite scores are not significantly impaired by maximal exercise and CNT can be administered immediately following maximal exercise- no recovery period is needed. However, symptom reports following maximal exercise are elevated.

Elevated symptom scores may be of important to take into account when interpreting an individual's baseline symptom reports.

Computerized Neurocognitive Outcomes following Maximal Exercise

Computerized neurocognitive test composite scores did not exhibit decreased performance following maximal exercise. These results are in contrast to the findings published by Covassin et al., in 2007. Dr. Covassin and colleagues (2007) reported deficits in verbal memory composite scores immediately following a maximally graded treadmill exercise test. Much of the foundation of this study was aimed to extend the results published by Covassin and colleagues (2007) by proposing a clinically appropriate recovery interval following maximal exercise. However, since results from this study were unable to replicate findings of decreased neurocognitive outcome scores a proposed recovery interval cannot be determined.

As discussed in the literature review, the studies examining the dynamic relationship of acute exercise and neurocognitive function yield inconclusive results. A meta-analysis published by Chang et al., (2012) examined moderators of cognitive performance following acute exercise. This meta-analysis concluded that exercise intensity, sex, and aerobic capacity are significant moderators of performance when assessing cognitive function after a delay of 1-20 minutes. When compared to the cohort used in Covassin's (2007) study our sample had similar average VO₂ peak values (52.2 \pm 9.8 vs 50.3 \pm 6.5 ml/kg/min), and the average age in this study was approximately 1 year older $(21.9\pm 2.3 \text{ vs. } 21.0 \pm 6.45)$. Compared to Covassin's study, the participant sample in this study were predominantly male. These differences, while subtle, could be attributed to differences in main outcomes.

Although results from this study did not align with studies documenting cognitive impairments following exercise (Covassin et al., 2007; Del Giorno, et al., 2010; Lo, et al., 2008),

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other studies have provide mixed support for cognitive changes follow exercise (Chang, et al., 2012; Lambourne, & Tomporowski, 2010). Additionally, a more recent study, examined self reported strenuous exercise within 3 hours of taking CNT, reporting no differences in cognitive performance following exercise (Hall, Cottle, Ketcham, Patel, & Barnes, 2017). Although the current study failed to replicate Covassin's findings of decreased CNT scores, the results from this study are not novel and have been observed in other studies aimed to examine the effects of maximal exercise on cognitive function.

Total Symptom Scores Following Maximal Exertion

On any given day a healthy non-concussed individual may endorse symptoms assessed by the PCSS. The average PCSS total symptom score in healthy college age men and women is 5-9, respectively (Lovell, Iverson, & Collins, 2006). In the current study symptom reports following maximal exercise were significantly higher compared to symptom reports during the baseline (non-exercise) trial. Increased symptom reporting at baseline may reflect prior physical exertion and, in this study, help to distinguish variability amongst the four experimental trials. However, since symptoms had not returned to baseline during the 20 minute recovery trial, a proposed recovery interval for symptom resolution and CNT administration following maximal exercise cannot be recommended. These results suggest that in order for clinicians to collect accurate symptom reports during baseline CNT administration the recovery interval following maximal exercise should be longer than 20 minutes.

Strengths of this Study

The repeated measures design used in this study allows for comparison across all four conditions. This study was carefully designed to minimize the effect of moderating variables of CNT performance such as caffeine intake, hydration status, and OTC drug ingestion. Caffeine

has been shown to significantly influence reaction time and cognitive performance (Haskell, Kennedy, Wesnes, & Scholey, 2005). Hydration status is also been reported to moderate cognitive performance, euhydrated participants perform significantly better than dehydrated individuals (Cian, et al., 2000). Participants that did not comply with pre-trial instructions or whom were unable to meet the hydration status requirements were asked to return on another day. Additionally, much effort was taken to ensure that participants were equally exerted on all trials. Although researchers could not control motivation or effort, self-reports of these variables were assessed during each visit and analyzed in statistical analyses used to assess test condition equivalence. Results from the statistical analysis of maximal exertion test outcomes indicate that individuals were equally exerted between all exercise sessions. Mean metabolic testing data (RER, HR, RPE), presented in Table 4. The mean RER, HR, and RPE would qualify the metabolic data for criteria establishing maximal exercise.

Supplementary Analyses

Supplementary exploratory analyses were conducted to explore data through a moderator lenses. Factors such as sex and aerobic fitness level were used to dichotomize the sample. Although underpowered, repeated measures ANOVAs were conducted with the sample dichotomized.

When the sample was dichotomized by sex, all variables used to determine test condition equivalence remained not significantly different; however, differences between males and females were observed. Compared to baseline measures there were no significant changes in CNT composite scores or symptom totals for females. Males exhibited a significant decline in visual memory composite scores during the 20-minute recovery trial when compared to baseline scores.

When the sample was dichotomized by aerobic fitness, all variables used to determine test condition equivalence remained not significantly different. The sample was dichotomized using percentile rankings of aerobic fitness provided by the ACSM's Guidelines for Exercise Testing and Prescription (Riebe, et al., 2017). The highly fit group was comprised of individuals ranking above the 80th percentile. Compared to baseline, the group of highly fit individuals exhibited significantly better processing speed scores during the 20 minute trial; all other measures were not significantly different from baseline. Additionally, the lower fitness group reported significantly elevated symptoms following exercise when compared to their baseline reports. Analogous with previously published research, results from this supplementary analysis support level of aerobic fitness and sex may moderate the effect of maximal exercise on cognitive function.

Using symptom clusters proposed by Kontos et al., (2012), pre-test total symptom scores were re-coded and compared across all trials using repeated measures ANOVAs. Symptoms associated with the cognitive-sensory and affective symptoms clusters were not significantly different following exertion trials. However significant differences following maximal exercise were observed for the sleep-arousal and vestibular-somatic clusters. Fatigue, trouble falling asleep, sleeping less, and drowsiness are factors within the sleep-arousal cluster (Kontos et al., 2012). These symptoms were significantly elevated during the immediate trial when compared to baseline reports. Headache, nausea, vomiting, balance, and dizziness are factors within the vestibular-somatic symptom cluster (Kontos et al., 2012). These symptoms were significantly elevated during the immediate and 10-minute recovery trial compared to baseline. These results suggest that the majority of symptoms elicited by maximal exercise can be grouped into two

PCSS clusters: sleep-arousal, and vestibular-somatic. If symptom reports are examined using cluster grouping, symptoms may resolve within 20 minutes post maximal exertion.

Implications

Anecdotal evidence suggests baseline CNTs are administered in and around rigorous and demanding sports schedules. Results from a previous study suggested CNT performance might be significantly impaired immediately following maximal exercise. The results from this study suggest sports medicine professionals should wait longer than 20 minutes before administering CNT to allow baseline symptom reports to return to normal; however, CNT composite scores remained stable after maximal exercise. Supplementary analyses suggest better processing speed scores may alter baseline performance when assessed 20 minutes after maximal exercise in certain groups (highly fit) of individuals, and males and females may be provoked differently. The timeline for resolution of these changes is unknown.

Limitations

The configuration of the exercise science laboratory did not allow for "immediate" testing following maximal exertion, the minimum recovery time was 42 seconds. The study conducted by Covassin and colleagues (2007) reported that CNT was administered immediately after test termination. Logistically, immediate test administration was not feasible but effort was taken to minimize the recovery interval during the "immediate" trial. Participants in this study were not allowed a cool down period, headgear used for the VO₂ max test was quickly removed, and the participant was escorted to the CNT testing room. Additional limitations of this study include: using USG for a proxy of hydration, not accounting for menstrual cycles of female participants, reliance on participant's best effort across all trials, and using self reported activity reports to qualify inclusion.

Suggestions for Future Research

Future research should continue to explore the relationship between maximal exercise and cognitive performance. Despite null results of this study, if cognitive deficits do occur following maximal exercise wait times should be determined. This study should be repeated but the wait time following maximal exercise should be extended to allow for symptom resolution. Additionally, a similar study should explore the dynamic relationship of maximal exercise and cognition in younger cohorts. Finally, other types of fatiguing exercise should be used in similar studies to examine sport-specific exertion that may be more applicable in clinical settings.

Conclusion

The results of this study did not support the hypotheses that graded maximal $VO₂$ treadmill testing prior to CNT administration would result in worse composite scores immediately following exercise. Therefore, an appropriate recovery interval following maximal exercise for neurocognitive composite scores was not indicated. The results of the current study did support immediate symptom provocation following maximal exercise. These symptoms still appeared to be significantly elevated following a 20 minute rest interval suggesting symptom resolution following maximal exercise lies beyond a 20 minute recovery interval. In order to administer baseline CNT and obtain accurate representation of normal neurocognitive performance and symptom reports sports medicine professionals should wait longer than 20 minutes.

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Supplementary Analyses

Table 5.

Supplementary Analyses of Results

Table 6.

n caamuu omcomes, and $C(n)$ scores, $(n-1)$).		Baseline	Immediate		10-minute		20-minute			
Measure	\boldsymbol{M}	SD	\boldsymbol{M}	SD	\boldsymbol{M}	SD	\boldsymbol{M}	SD		
Pre-Trial Compliance Assessments/Measures and Self-reported Effort Assessment										
Hydration Status (USG)	1.012	.01	1.013	.01	1.012	.01	1.013	.01		
Previous Night's Sleep (hrs)	7.50	.85	9.57	11.54	7.14	.92	7.36	1.13		
Overall Feeling	3.11	1.41	3.33	1.33	3.00	1.14	3.28	.751		
24-hour Caloric Intake (kcal)	1926	753	2050	1033	1951	1083	2443	1151		
Self-Reported Effort	3.94	.24	3.89	.32	3.89	.32	3.94	.24		
Mean and standard deviations for test outcomes of the graded maximal VO ₂ treadmill protocol										
RER			1.12	.04	1.13	.03	1.12	.05		
RPE (Borg 6-20 Scale)			19.00	1.25	19.05	.97	19.26	.87		
VO ₂ peak (ml/kg/min)			57.10	7.92	58.37	9.17	57.88	8.34		
VO2 last minute avg. (ml/kg/min)			55.45	7.89	56.70	9.06	55.98	8.23		
Duration of Exercise (minutes)			14.17	2.02	14.34	2.70	14.17	2.06		
Maximum Heart Rate (bpm)			189.37	6.95	189.95	6.82	188.79	6.71		
Heart Rate 1 minute post exercise			150.26	20.32	151.95	14.37	154.89	9.46		
CNT scores and physiologic data for all trials.										
HR at Start of CNT test*	60.5 ^{bcd}	10.31	138.74 ^{acd}	23.92	102.26^{ab}	8.56	103.79 ab	23.01		
HR at End of CNT test*	61.6 ^{bcd}	9.35	103.89 ^a	22.23	94.26 bd	8.57	89.89 bc	10.07		
Verbal Memory Composite	92.58	8.36	90.11	8.08	92.16	8.84	92.63	7.01		
Visual Memory Composite *	80.82 ^d	12.41	79.45	12.09	78.18	8.69	77.91	14.31 ^a		
Processing Speed Composite	44.13	5.27	45.63	5.15	46.00	5.05	46.35	5.23		
Reaction Time (sec)	.60	.09	.58	.09	.62	.11	.59	.10		
Total PCSS Symptom Score	3.58	4.86	11.79	12.76	9.47	11.55	6.32	7.18		

Male ONLY means and standard deviations of pre-trial compliance, graded maximal VO² treadmill outcomes, and CNT scores, (n=19).

Note: $*=01$; $*=$ Different from Baseline; $\frac{b}{s}$ Different from Immediate; $\frac{c}{s}$ Different from 10 min; d = Different from 20 min

Results from a series of repeated measures ANOVAs for pre-trial compliance assessments and test outcomes of the graded maximal VO² treadmill protocol show no significant difference across all measures. Results from a series of repeated measures ANOVAs CNT composite scores and PCSS total symptoms show no significant difference across: verbal memory ($p = .57$, *Wilks* $\lambda = .83$, $F [3, 16] = .70$, $\eta^2 = .12$), , processing speed ($p = .02$, *Wilks* $\lambda = .54$, *F* [3,16] = 4.72, η^2 = .46), reaction time (*p*=.12, *Wilks* λ = .70, *F* [3,16] = 2.25, η^2 = .30), PCSS total score (*p*=.03, *Wilks* $\lambda = .58$, *F* [3,16] = 3.81, $\eta^2 = .42$). Heart rate taken at the start (*p*=.00, *Wilks* $\lambda = .04$ *, F* [3,16] = 143.94, $\eta^2 = .96$) and after (*p*=.00, *Wilks* $\lambda = .07$, *F* [3,16] = 76.90, $\eta^2 =$.93) CNT was significantly different across trails. Visual memory ($p=0.0$, *Wilks* $\lambda = 0.25$, *F* [3,16] $= 16.35, \eta^2 = .75$), Scores during the 20 minute time interval were significantly lower than baseline $[t(11)=-6.61, p=.00]$.

Table 7.

	Baseline			Immediate		10-minute		20-minute
Measure	M	SD	\boldsymbol{M}	SD	\boldsymbol{M}	SD	M	SD
Pre-Trial Compliance Assessments/Measures and Self-reported Effort Assessment								
Hydration Status (USG)	1.009	.01	1.010	.01	1.011	.01	1.009	.01
Previous Night's Sleep (hrs)	7.17	1.00	10.44	10.58	7.33	1.32	7.78	1.30
Overall Feeling	3.82	.75	3.64	.67	3.64	.67	3.09	.54
24-hour Caloric Intake (kcal)	1952	754	1599	849	1750	347	1739	700
Self-Reported Effort	3.91	.30	3.91	.30	3.91	.30	4.00	.00
Mean and standard deviations for test outcomes of the graded maximal VO ₂ treadmill protocol								
RER			1.10	.04	1.09	.04	1.12	.04
RPE (Borg 6-20 Scale)			19.18	.98	19.36	.92	19.27	.79
VO ₂ peak (ml/kg/min)			48.46	9.95	48.3	8.70	47.24	9.65
VO2 last minute avg. (ml/kg/min)			46.84	9.96	43.54	10.92	46.22	9.32
Duration of Exercise (minutes)			12.39	1.76	12.76	1.65	12.48	2.06
Maximum Heart Rate (bpm)			197.46	8.72	196.55	8.18	197.55	5.87
Heart Rate 1 minute post exercise			164.82	17.61	153.73	16.47	159.45	16.21
CNT scores and physiologic data for all trials.								
HR at Start of CNT test*	66.45 ^{bcd}	11.48	150.00 ^{acd}	20.91	102.9^{ab}	15.73	99.82 ^{ab}	14.32
HR at End of CNT test*	67.00 _{bcd}	10.48	102.45^{a}	14.71	94.45 ^a	14.28	96.09 ^a	15.46
Verbal Memory Composite	93.36	6.92	91.09	8.22	92.82	6.10	95.09	4.93
Visual Memory Composite	82.90	9.46	80.21	11.18	78.53	7.53	74.16	12.55
Processing Speed Composite	45.44	5.39	46.27	6.30	46.73	5.34	47.05	6.64
Reaction Time (sec)	.61	.09	.60	.08	.59	.07	.61	.11
Total PCSS Symptom Score	1.91	2.17	9.91	9.61	4.18	4.71	4.64	5.01

*Female ONLY means and standard deviations of pre-trial compliance, graded maximal VO*₂ *treadmill outcomes, and CNT scores (n=11).*

Note: $*=01$; $*=$ Different from Baseline; $\frac{b}{s}$ Different from Immediate; $\frac{c}{s}$ Different from 10 min; $d=$ Different from 20 min

Results from a series of repeated measures ANOVAs for pre-trial compliance assessments and test outcomes of the graded maximal VO₂ treadmill protocol show no significant difference across all measures. Results from a series of repeated measures ANOVAs CNT composite scores and PCSS total symptoms show no significant difference across: verbal memory (*p*=.16, *Wilks* $\lambda = .54$, *F* [3,8] = 2.25, $\eta^2 = .46$), visual memory (*p*=.90, *Wilks* $\lambda = .25$, *F* $[3,8] = 16.35$, $\eta^2 = .75$), processing speed ($p = .34$, *Wilks* $\lambda = .67$, *F* $[3,8] = 1.31$, $\eta^2 = .33$), reaction time (*p*=.51, *Wilks* $λ = .76$, $F [3,8] = .84$, $η² = .24$), PCSS total score (*p*=.18, *Wilks* $λ =$.44, *F* [3,8] = 2.1, η^2 = .44. Heart rate taken at the start (*p*=.00, *Wilks* λ = .03, *F* [3,8] = 94.10, η^2 = .97) and after (*p*=.00, *Wilks* $λ = .06$, $F [3,8] = 38.72$, $η² = .94$) CNT was significantly different across trails.

Table 8.

						20-minute	
M	SD	\boldsymbol{M}	SD	\boldsymbol{M}	SD	\boldsymbol{M}	SD
1.010	.00.	1.011	.01	1.011	.01	1.010	.01
7.27	.79	11.55	13.5	7.23	1.17	7.74	1.28
3.23	1.24	3.46	1.33	3.08	1.19	3.31	1.32
2178	621	2069	889	1869	892	2454	1184
4.0	.00	3.93	.27	3.93	.27	3.93	.27
		1.11	.05	1.11	.04	1.11	.05
		19.0	1.04	18.93	1.0	19.36	.84
		61.5	6.4	62.9	7.7	62.2	6.8
		60.0	6.59	61.3	7.46	60.45	6.34
		13.8	1.76	14.4	2.46	13.7	2.06
		191.5	6.0	192.9	5.7	190.7	3.97
57.50 ^{bcd}	8.06	138.79 ^{acd}	20.31	101.2^{ab}	12.64	95.14^{ab}	11.7
58.36bcd	7.74	96.00 ^{ad}	9.27	92.86 ^a	11.23	87.57ab	10.55
93.86	4.67	90.07	8.71	91.43	9.25	92.64	8.18
85.86	8.59	81.00	12.23	78.71	9.24	78.07	12.51
45.96 ^d	5.35	46.43	4.83	48.46	3.3	49.12	$4.24^{\rm a}$
.59	.09	.56	.09	.58	.08	.58	.08
3.64	3.93 h	14.71	14.39	9.29	9.09	6.29	5.92
	VO2 last minute avg. (ml/kg/min)	Baseline CNT scores and physiologic data for all trials.	$\mathbf{v} \cup \mathbf{z}$ in caumin bancomes, and $\mathbf{c}_1 \mathbf{v}_1$ scores, $(n-1\tau)$.	Immediate		10-minute Pre-Trial Compliance Assessments/Measures and Self-reported Effort Assessment	Mean and standard deviations for test outcomes of the graded maximal VO ₂ treadmill protocol

"High Fitness" ONLY means and standard deviations of pre-trial compliance, graded maximal VO² treadmill outcomes, and CNT scores, (n=14).

Note: $*=01$; $*=$ Different from Baseline; $\frac{b}{s}$ Different from Immediate; $\frac{c}{s}$ Different from 10 min; $d=$ Different from 20 min

Results from a series of repeated measures ANOVAs for pre-trial compliance assessments and test outcomes of the graded maximal VO² treadmill protocol show no significant difference across all measures. Results from a series of repeated measures ANOVAs CNT composite scores and PCSS total symptoms show no significant difference across: verbal memory (*p*=.53, *Wilks* $\lambda = .83$, *F* [3,11] = 78, $\eta^2 = .18$), visual memory (*p*=.02, *Wilks* $\lambda = .41$, *F* $[3,11] = 5.26$, $\eta^2 = .59$), reaction time ($p = .40$, *Wilks* $\lambda = .78$, $F[3,11] = 1.06$, $\eta^2 = .23$), PCSS total score ($p = .08$, *Wilks* $\lambda = .56$, $F [3, 11] = 2.9$, $\eta^2 = .44$). Processing speed was significantly different ($p=0.01$, *Wilks* $\lambda = .36$, $F[3,11] = 6.44$, $\eta^2 = .64$), post- hoc analysis revealed significantly higher scores during the 20 minute recovery interval when compared to baseline, [t(11)=-4.6, $p = .003$]. Additionally heart rate taken at the start ($p = .00$, *Wilks* $\lambda = .02$, *F* [3,11] = 165.22, $\eta^2 = .98$) and after (*p*=.00, *Wilks* $\lambda = .03$, *F* [3,11] = 117.1, $\eta^2 = .97$) CNT was significantly different across trails.

Table 9.

	Baseline		Immediate			20-minute 10-minute				
Measure	\boldsymbol{M}	SD	\boldsymbol{M}	SD	\boldsymbol{M}	SD	\boldsymbol{M}	SD		
Pre-Trial Compliance Assessments/Measures and Self-reported Effort Assessment										
Hydration Status (USG)	1.012	.01	1.012	.01	1.013	.01	1.013	.01		
Previous Night's Sleep (hrs)	7.46	1.03	8.42	8.27	7.21	1.03	7.63	1.15		
Overall Feeling	3.5	1.27	3.44	.96	3.38	.89	3.13	1.09		
24-hour Caloric Intake (kcal)	1724.1	852.0	1722.2	844.8	1884.9	904.1	1948.9	897.6		
Self-Reported Effort	3.87	.35	3.87	.35	3.87	.35	4.0	.00		
Mean and standard deviations for test outcomes of the graded maximal VO ₂ treadmill protocol										
RER			1.11	.03	1.11	.04	1.13	.04		
RPE (Borg 6-20 Scale)			19.13	1.26	19.38	.89	19.19	.83		
VO ₂ peak (ml/kg/min)			47.27	5.87	47.51	5.46	46.76	6.23		
VO2 last minute avg. (ml/kg/min)			45.57	5.84	43.61	7.36	45.36	5.94		
Duration of Exercise (minutes)			13.30	2.37	13.22	2.41	13.41	2.36		
Maximum Heart Rate (bpm)			193.06	10.33	191.94	9.57	193.12	9.80		
CNT scores and physiologic data for all trials.										
HR at Start of CNT test*	67.19 ^{bcd}	11.36	146.44 ^{acd}	25.51	103.63^{ab}	10.56	108.62^{sb}	23.88		
HR at End of CNT test*	68.19bcd	9.56	109.81 ^{acd}	23.85	95.63^{ab}	10.53	96.19ab	12.85		
Verbal Memory Composite	92.00	9.76	90.81	7.59	93.25	6.55	94.31	4.32		
Visual Memory Composite	78.88	11.13	79.00	10.76	78.13	6.66	73.31	13.61		
Processing Speed Composite	43.44	5.05	45.37	6.15	44.34	5.65	44.40	5.98		
Reaction Time (sec)	.92	.09	.61	.08	.63	.11	.62	.11		
Total PCSS Symptom Score	2.38	4.30	7.94	7.49	6.00	10.53	5.19	7.0		
Note: $*=01$; $*=$ Different from Baseline; \overline{b} = Different from Immediate; \overline{c} = Different from 10 min;										

"Lower fitness" ONLY means and standard deviations of pre-trial compliance, graded maximal VO² treadmill outcomes, and CNT scores, (n=16).

 $d=$ Different from 20 min

Results from a series of repeated measures ANOVAs for pre-trial compliance assessments and test outcomes of the graded maximal $VO₂$ treadmill protocol show no significant difference across all measures. Results from a series of repeated measures ANOVAs CNT composite scores and PCSS total symptoms show no significant difference across: verbal memory ($p = 0.23$, *Wilks* $\lambda = 0.73$, $F[3,13] = 1.63$, $\eta^2 = 0.27$), visual memory ($p = 0.52$, *Wilks* $\lambda = 0.85$, $F[3,13] = 0.63$, $\eta^2 = 0.27$ $[3,13] = .80, \eta^2 = .16$, processing speed ($p = .27$, *Wilks* $\lambda = .75$, *F* $[3,13] = 1.48, \eta^2 = .25$), reaction time (*p*=.08, *Wilks* $λ = .92$, $F [3,13] = .38$, $η² = .08$), PCSS total score (*p*=.02, *Wilks* $λ =$.47, *F* [3,13] = 4.81, η^2 = .53). Significant differences were observed for heart rate at the start (*p*=.00, *Wilks* λ = .04, *F* [3,13] = 103.19, η ² = .96), and end (*p*=.27, *Wilks* λ = .75, *F* [3,13] = 1.48, $\eta^2 = .25$) of CNT. Additionally heart rate taken at the start ($p=.00$, *Wilks* $\lambda = .04$, *F* [3,13] = 103.19, $\eta^2 = .96$) and after ($p = .00$, *Wilks* $\lambda = .09$, *F* [3,13] = 46.7, $\eta^2 = .92$) CNT was significantly different across trails

Table 10.

Note: $*=0.01$; $*=$ Different from Baseline; $\frac{b}{c}$ Different from Immediate; $\frac{c}{c}$ Different from 10 min; $d=$ Different from 20 min

Results from a repeated measures ANOVA revealed significant differences based on recovery interval for PCSS sleep/arousal and vestibular/somatic cluster scores ($p=0.01$, *Wilks* λ *=* .65, *F* [3,27] = 4.8, η^2 = .40) and (p =.00, *Wilks* λ = .60, *F* [3,27] = 6.0, η^2 = .40), respectively. Results from a repeated measures ANOVA revealed no significant differences based on recovery interval for PCSS cognitive/sensory and affective cluster scores ($p=0.04$, *Wilks* $\lambda = .73$ *, F* [3,27] = 3.3, $\eta^2 = .27$) and ($p = .03$, *Wilks* $\lambda = .73$ *, F* [3,27] = 3.4, $\eta^2 = .27$), respectively. Post-hoc paired samples t-test revealed significantly higher sleep/arousal cluster scores following an immediate recovery interval compared to baseline pre-test symptom scores interval $[t(29)=3.61, p=.01]$. No significant differences were observed for 10-minute rest interval [t(29)= -2.47, *p*= .12] or 20 minute rest interval $[t(29)= -3.02, p=.03]$. Post-hoc paired samples t-test revealed significantly higher vestibular/somatic cluster scores following an immediate recovery interval compared to baseline pre-test symptom scores $[t(29)= -4.04, p=.00]$ and the 10-minute recovery interval [t(29)= -3.36, *p*= .01] No significant differences were observed for the 20-minute rest interval $[t(29)= -2.34, p=.26].$

References

Axxya Systems. Released 2018. Nutritionist Pro, Version 7.3.0. Redmond, WA.

- Audiffren, M., Tomporowski, P., Zagrodnik, J. (2009). Acute aerobic exercise and informational processing: modulation of executive control in a random number generation task. *Acta Psychologica*, *132*, 85-95. Doi: 10.1016/j.actpsy.2009.06.008
- Bailey, C., & Arnett, P. A. (2006). Motivation and the assessment of sports-related concussion. In *Foundation of sport-related brain injuries* (pp. 171-193). Springer, Boston, MA.
- Barkhoudarian, G., Hovda, D., & Giza, C. (2011). The molecular pathophysiology of concussive brain injury. *Clinics of Sports Medicine*, *30*(1), 33-48.
- Barth, J. T., Macciocchi, S. N., Giordani, B., Rimel, R., Jane, J. A., & Boll, T. J. (1983). Neuropsychological sequelae of minor head injury. *Neurosurgery*, *13*(5), 529-533.
- Beam, W. C., & Adams, G. M. (2014). *Exercise physiology laboratory manual* (7th ed.). New York, NY: McGraw-Hill.
- Borg, G. (1970). Perceived exertion as an indicator of somatic stress. *Scandinavian Journal of Rehabilitation Medicine, 2*, 92-98.
- Brisswalter, J., Collardeau, M., & Rene, A. (2002). Effects of acute physical exercise characteristics on cognitive performance*. Sports Medicine, 32(9)*, 555-566.
- Broglio, S. P., Cantu, R. C., Gioia, G. A., Guskiewicz, K. M., Kutcher, J., Palm, M., & Valovich, T. C. (2014). National Athletic Trainers' Association position statement: Management of sport concussion. *Journal of Athletic Training*, *49(2)*, 245-265.
- Broglio, S. P., Schnebel, B., Sosnoff, J. J., Shin, S., Feng, X., He, X., & Zimmerman, J. (2010). Biomechanical properties of concussion in high school football. *Medicine and Science in Sports and Exercise*, *42(11),* 2064-2071. Doi: 10.1249/MSS.0b013r3181dd9156
- Bryan, M., Rowhani-Rahbar, A., Comstock, R., & Rivara, F. (2016). Sports- and recreationrelated concussions in US youth. *Pediatrics, 138(1).* Doi: 10.1542/peds.2015-4635.
- Buchfuhrer, M.J., Hansen, J.E., Robinson, T.E., Sue, D.Y., Wasserman, K. & Whipp, B.J. (1983). Optimizing the exercise protocol for cardiopulmonary assessment. *Journal of Applied Physiology Respiratory, Environmental and Exercise Physiology,55*(5), 1558- 1564. Doi: 10.1152/jappl.1983.55.5.1558
- Chang,Y.K., Labban, J.D., Gapin, J.I., Etnier, J.L. (2012). The effects of acute exercise on cognitive performance: A meta-analysis. *Brain Research,1453,* 87-101. Doi: 10.1016/j.brainres.2012.02.068
- Cheuvront, S. N., Ely, B. R., Kenefick, R. W., & Sawka, M. N. (2010). Biological variation and diagnositic accuracy of dehydration assessment markers. *American Journal of Clinical Nutrition, 92*(3), 565-573. Doi: 10.3945/ajcn.2010.29490
- Cian, C., Koulmann, N., Barraud, P. A., Ralpehel, C., Jimenez, C., & Melin, B. (2000). Influence of variation of body hydration on cognitive function: Effect of hyperhydration, heat stress and exercise induced dehydration. *Journal of Psychophyiology, 14,* 29-36.
- Coles, K., & Tomporowski, P. (2008). Effects of acute exercise on executive processing, shortterm and long-term memory. *Journal of Sports Science, 26(3)*, 333-344. Doi: 10.1080/0240410701591417
- Covassin, T., Elbin, R.J., Bleeker, A., Lipchik, A., & Kontos, A.P. (2013). Are there differences in neurocognitive function and symptoms between male and female soccer players after concussion? *Journal of Sports Medicne, 41*(12), 2890-2895. Doi: 10.11777/0363546513509962
- Collins, M. W., Iverson, G., Lovell, M.R., McKeag, D., Norwig, J., & Maroon, J. (2003). Onfield predictors of neuropsychological and symptom deficit following sports-related concussion. *Clinical Journal of Sport Medicine, 13,* 222-229.
- Collins, M. W., Kontos, A. P., Reynolds, E., Murawski, C. D., & Fu, F. H. (2014). A comprehensive, targeted approach to the clinical care of athletes following sport-related concussion. *Knee Surgery, Sports Traumatology, Arthroscopy*, *22(2)*, 235-246. Doi: 10.1007/s00167-013-2791-6
- Cooper, C. J. (1973). Anatomical and physiological mechanisms of arousal, with special reference to the effects of exercise. *Ergonomics, 16*(5), 601-609. Doi: 10.1080/00140137308924551
- Covassin, T., Elbin, R., Harris, W., Parker, T., & Kontos, A. (2012). The role of age and sex in symptoms, neurocognitive performance, and postural stability in athletes after concussion. *The American Journal of Sports Medicine*, *20*(10), 1302-1312. Doi: 10.1177/0363546512444554
- Covassin, T., Elbin, R., Stiller-Ostrowski, J., & Kontos, A. (2009). Immediate Post-concussion assessment and cognitive (ImPACT) practices of sports medicine professionals. *Journal of Athletic Training*, *44*(6), 639-644.
- Covassin, T., Weiss, L., Powell, J., & Womack, C. (2007). Effects of a maximal exercise test on neurocognitive function. *British Journal of Sports Medicine*, *41(6)*, 370-374. Doi: 10.1136/bjsm.2006.032334
- Craig, C. L., Marshall, A. L., Sjostrom, M., Bauman, A., Booth, M. L., Ainsworth, B. E., ... Oja, P. (2003). International Physical Activity Questionaire: 12-country reliablity and validity. *Medicine and Science in Sports and Exercise, 35*, 1381-1395. Doi: 10.1249/01.MSS.0000078924.61453.FB
- Davey, C. P. (1973). Physical exertion and mental performance. *Ergonomics, 16*(5), 595-599. Doi: 10.180/00140137308924550
- Davranche, K., & McMorris, T. (2009). Specific effects of acute moderate exercise on cognitive control. *Brain & Cognition, 69*(3), 565-570. Doi: 10.1006/j.bandc.2008.12.0001
- Del Giorno, J. M., Hall, E. E., O'Leary, K. C., Bixby, W. R., & Miller, P. C. (2010). Cognitive Function During Acute Exercise: A test of the transient Hypofrontality theory. *Journal of Sport & Exercise Psychology*, *32*, 312-323.
- Dietrich, A. (2006). Transient hypofrontality as a mechanism for the psychological effects of exercise. *Psychiatry Research*, *145*(1), 79-83. Doi: 10.016/j.psychres.2005.07.033
- Dominguez, D.C., & Raparla, M. (2014). Neurometabolic aspects of sports-related concussion. *Seminars in Speech and Language, 35*(3), 159-165. Doi: 10.1055/s-0034-1384677
- Elbin, R.J., Knox, J., Kegal, N., Schatz, P., Lowder, H. B., French, J., ... Kontos, A.P. (2016). Assessing symptoms in adolescents following sport-related concussion: A comparison of four different approaches. *Applied Neuropsychology: Child, 5*(4), 294-302. Doi: 10.1080/21622965.2015.1077334
- Elbin, R.J., Kontos, A. P., Kegel, N., Johnson, E., Burkhart, S., & Schatz, P. (2013). Individual and combined effects of LD and ADHD on computerized neurocognitive concussion test performance: Evidence for separate norms. *Archive of Clinical Neuropsychology, 28(5)*, 476-484. Doi: 10.1093/arclin/act024
- Elbin, R.J., Sufrinko, A., Schatz, P., French, J., Henry, L., Burkhart, S., ... Kontos, A.P. (2016). Removal from play after concussion and recovery time. *Pediatrics, 138*(3). Doi: 10.1542/peds.2016-0910
- Frommer, L. J., Gurka, K.K., Cross, K. M., Ingersoll, C. D., Comstock, R. D., & Saliba, S. A. (2011). Sex differences in concussion symptoms of high school athletes. *Journal of Athletic Training, 46*(1), 76-84. Doi: 10.4085/1062-6050-46.1.76
- Gessel, L. M., Fields, S. K., Collins, C. l., Dick, R. W., & Comstock, R. D. (2007). Concussions among United States high school and collegiate athletes. *Journal of Athletic Training*, *42 (4)*, 495-503.
- George, J. D. (1996). Alternative approach to maximal exercise testing and VO2max prediction in college students. *Research Quarterly for Exercise and Sport, 67*(4), 452-457. Doi: 10.1080/02701367.1996.10607977
- Giza, C. C., & Hovda, D. A. (2001). The neurometabolic cascade of concussion. *Journal of Athletic Training, 36*(3), 228-235.
- Giza, C. C., & Hovda, D. A. (2014). The new neurometabolic cascade of concussion. *Neurosurgery,* 7*5*(4), S24-S33. Doi: 10.1227/NEU.0000000000000505
- Green, H. J., Hughson, G. W., & Ranney, D. A. (1983). Anaerobic threshold, blood lactate, and muscle metabolites in progressive exercise. *Journal of Applied Physiology, 54*(4), 1032- 1038. Doi: 10.1152/jappl.1983.54.4.1032
- Greenwald, R., Gwin, J., Chu, J., & Crisco, J. (2008). Head impact severity measures for evaluating mild traumatic brain injury risk exposure. *Neurosurgery, 62(4),* 789-798. Doi: 10.1227/01.neu.0000318162.67472.ad
- Gurjian, E. S. (1972). Recent advances in the study of the mechanism of impact injury of the head-a summary. *Clinical Neurosurgery, 19,* 1-42.
- Guskiewicz K. M., Weaver N. L., Padua D. A., & Garett W. E. (2000). Epidemiology of concussion in collegiate and high school football players. *American Journal of Sports Medicine*, *28*(5), 643-650. Doi: 10.1177/036355465000280050401
- Guskiewicz, K. M., & Mihalik, J. P. (2011). Biomechanics of sport concussion: Quest for the elusive injury threshold. *Exercise and Sport Science Reviews*, *39(1),* 4-11. Doi: 10.1097/JES.0b013e318201f53e
- Hall, E., Cottle, J., Ketcham, C., Patel, K., & Barnes, K. P. (2017). Concussion baseline testing preexisting factors, symptoms, and neurocognitive performance. *Journal of Athletic Training, 52*(2), 77-81. Doi: 10.4085/1062-6050-51.12.21
- Halstead, M. E., & Walter, K. D. (2010). Clinical Report- sport related concussion in children and adolescents. *Pediatrics*, *126*(3), 597-615.
- Harmon, K. G., Drenzer, J. A., Gammons, M., Guskiewicz, K. M., Halstead, M., Herring, S. A…Roberts, W. (2013). American Medical Society for Sports Medicine position statement: concussion in sport. *British Journal of Sports Medicine*, *47*(1), 15-26. Doi: 10.1136/bjsports-2012-091941
- Haskell, C. F., Kennedy, D.O., Wesnes, K. A., & Scholey, A. B. (2005). Cognitive and mood improvemnts of caffeine in habitual consumers and habitual non-consumers of caffeine. *Psychopharmacology, 179*(4), 813-825. Doi: 10.1007/s00213-004-2104-3
- Hill, A. V. & Lupton, H. (1923). Muscular exercise, lactic acid and the supply and utilization of oxygen. *QJM: An International Journal of Medicine, 16*(62), 135-171. Doi: 10.1093/qjmmed/os-16.62.135
- Hillman, C. H., Snook, E. M., & Jerome, G. J. (2003) Acute cardiovascular exercise and executive control function. *International Journal of psychophysiology*, *48*, 307-314.
- Holbourne, A. (1943). Mechanics of head injuries. *Lancet 2,* 438-441.
- Ide, K., & Secher, N. H. (2000). Cerebral blood flow and metabolism during exercise. *Progress in Neurobiology*, *61*(4), 397-414.
- IBM Corp. Released 2016. IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY: IBM Corp.
- Iverson, G. L., Lovell, M. R., & Collins, M. W. (2003). Interpreting change on ImPACT following sport concussion. *Clinical Neuropsychology*, *17*(4), 460-467.
- Johnson, E. W., Kegel, N. E., & Collins, M. W. (2011). Neuropsychological assessment of sportrelated concussion. *Clinical Sports Medicine*, *30*, 73-88. Doi: 10.1016/j.csm.2010.08.007
- Kalimo, H., Rehncrona, S., Soderfeld, B., Olsson, Y., & Siesjo, BK. (1981). Brain lactic acidosis and ischemic cell damage: 2. Histopathology. *Journal of Cerebral Blood Flow Metabolism, 1*(3), 313-327. Doi: 10.1038/jcbfm.1981.35
- Kontos, A. P., Elbin, R., Schatz, P., Covassin, T., Henry, L., Pardini, J., & Collins, M. W. (2012). A revised factor structure for the post-concussion symptom scale: baseline and post-concussion factors. *The American Journal of Sports Medicine*, *40*(10), 2375-2384. Doi: 10.1177/0363546512455400
- Lambourne, K., & Tomporowski, P. (2010). The effect of exercise-induced arousal on cognitive task performance: A meta-regression analysis. *Brain Research, 1341,*12-24. Doi: 10.1016/j.brainres.2010.03.091
- Langlois, J., Rutland-Brown, W., & Wald, M. (2006). The epidemiology and impact of traumatic brain injury: a brief overview. *Journal of Head Trauma and Rehabilitation*, *21(5)*, 375- 378.
- Lau, B. C., Kontos, A. P., Collins, M. W., Mucha, A., & Lovell, M. R. (2011). Which on-field signs/symptoms predict protracted recovery from sport-related concussion among high school football players? *American Journal of Sports Medicine*, *39*(11), 2311-2318. Doi: 10.177/0363546511410655
- Lau, B., Lovell, M. R., Collins, M. W., Pardini, J. (2009). Neurocognitive and symptom predictors of recovery in high school athletes. *Clinical Journal of Sports Medicine, 19*(3), 216-21. Doi: 10.1097/JSM.0b013e31819d6edb
- Laurer, H. L., Bareyre, F. M., Lee, V. M., Trojanowski, J. Q., Longhi, L., Hoover, R., ... McIntosh, T. K. (2001). Mild head injury increasing the brain's vulnerability to a second concussive impact. *Journal of Neurosurgery*, *95*, 859-870.
- Le, T. H. & Gean, A. D. (2009). Neuroimaging of traumatic brain injury. *The Mount Sinai Journal of Medicine, 76*(2), 145-162. Doi: 10.1002/msj.20102
- Lo, B. E., Willer, B., Burton, H., Leddy, J. J., Wilding, G. E., Horvath, P.J. (2008). Short-term exercise to exhaustion and its effects on cogntive funciton in young women. *Perceptual and Motor Skills,107*(3), 933-945. Doi: 10.2466/pms.107.3.933-945
- Lovell, M. R., & Collins, M. W. (1998). Neuropsychological assessment of the college football player. *Journal of Head Trauma Rehabilitation. 13,* 9-26.
- Lovell, M. R., Iverson, G. L., Collins, M. W., McKeag, D., & Maroon, J. C. (1999). Does Loss of Consciousness Predict Neurophysiological Decrements after concussion. *Journal of Sports Medicine*, *9*(4), 193-198.
- Lovell, M. R., Iverson, G. L., Collins, M. W., Podell, K., Johnston, K. M., Pardini, D., ... Maroon, J. C. (2006). Measurement of Symptoms Following Sports-Related Concussion: Reliability and Normative Data for the Post-Concussion Scale. *Applied Neuropsychology*, 13(3), 166-174.
- Mahoney, C.R., Hirsch, E., Hasselquist, L., Lesher, L. L., Lieberman, H. R. (2007). The effects of movement and physical exertion on soldier vigilance. *Aviation, Space and Environmental Medicine, 78*(5)*,* B51-57.
- Marar, M., Mcllvain, N., Fields, S., & Comstock, R. (2012). Epidemiology of concussions among United States high school athletes in 20 sports. *American Journal of Sports Medicine, 40(4),* 747-755. Doi: 10.1177/036354626
- Mauger, A. R., & Sculthorpe, N. (2012). A new VO2 max protocol allowing self-pacing in maximal incremental exercise. *British Journal of Sports Medicine, 46*(1), 59-63. Doi: 10.1136/bjsports-2011-090006
- McCrea, M., Guskiewicz, K. M., Marshall, S. W., Barr, W., Randolph, C., Cantu, R. C., ... Kelly, J.P. (2003). Acute effects and recovery time following concussion in collegiate football players: the NCAA concussion study. *JAMA*, *290*(19), 2556-2563. Doi: 10.1001/jama.290.19.2556
- McCrea, M., Hammeke, T., Olsen, G., Leo, P., & Guskiewicz, K. (2004). Unreported concussion in high school football players: Implications for preventions. *Clinical Journal of Sport Medicine*, *14(1)*, 13-17.
- McCrory, P. (2001). Does second impact syndrome exist? *Clinical Journal of Sport, 11*(3), 144- 149.
- McCrory, P., Meeuwisse, W., Aubry, M., Cantu, B., Dvorak, J., Echemendia, R., …Tator, C. (2013). Consensus statement on concussion in sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012. *Clinical Journal of Sport Medicine*, 23(2), 89-117. Doi: 10.1097/JSM.0b013e31828b67cf
- McCrory, P., Meeuwisse, W., Dvorak, J., Aubry, M., Bailes, J., Broglio, S., …Vos, P. (2017). Consensus statement on concussion in sport: The 5th International Conference on Concussion in Sport held in Zurich, October 2016. *British Journal of Sports Medicine* Advanced online publication, 1-10. Doi: 10.1136/bjsports-2017-097699
- McGrath, N., Dinn, W. M., Collins, M. W., Lovell, M. R., Elbin, R., & Kontos, A. P. (2013). Post-exertion neurocognitive test failure among student-athletes following concussion. *Brian Injury*, *27*(1), 103-113. Doi: 10.3109/02699052.2012.729282
- McIntosh, T.K., Faden, A.I., Yamakami, I., & Vink, R. (1998). Magnesium deficency exacerbates and pretreatment improves outcome following traumatic brain injury rates in rats: 31P magnetic resonance spectroscopy and behavioral studies. *Journal of Neurotrauma, 5*(1), 17-31. Doi: 10.1089/neu.1988.5.17.
- McMorris, T., & Hale, B. J. (2012). Differential effects of differing intensities of acute exercise on speed and accuracy of cognition: A meta-analytical investigation. *Brain and Cognition*, *80*(3), 338-351. Doi: 10.1016/j.bandc.2012.09.001
- Meehan III, W. P., Pierre, D., & Comstock, R. D. (2010). High School Concussions in the 2008-2009 Academic Year. *The American Journal of Sports Medicine*, *38*(12), 2405-2409. Doi: 10.1177/0363546510376737
- Mihalik, J., Lengas, E.,Register-Mihalik, J., Oyama, S., Begalle, R. & Guskiewicz, K. (2013). The effect of sleep quality and sleep quantity on concussion baseline assessment. *Clinical Journal of Sport Medicine, 23*(5), 343-348. Doi: 10.1097/JSM.0b013e318295a834
- Moser, R. S., Schatz, P., Neidzwski, K., & Ott, S. D. (2011). Group verses individual administration affects baseline neurocognitive test performance. *American Journal of Sports Medicine, 39(11)*, 2325-2330. Doi: 10.1177/0363546511417114
- Nada, B., Balde, J., & Manjunatha, S. (2013). The acute effects of a single bout of moderateintensity aerobic exercise on cognitive functions in healthy adult males. *Journal of Clinical and Diagnostic Research, 7(9)*, 1883-1885. Doi: 10.7860/JCDR/2013/5855.3341
- Noble, J., & Hesderffer, D. (2013). Sport-related concussions: A review of epidemiology, challenges in diagnosis, and potential risk factors. *Neuropsycholgy Review, 23(4),* 273- 284. Doi: 10.1007/s11065-013-9239-0
- Ommaya, A. K., & Gennarelli, T. (1974). Cerebral concussion and traumatic unconsciousness. *Brain*, *97*, 633-654.
- Patterson, E. (2005). Guidelines for data processing and analysis of the International Physical Activity Questionaire (IPAQ). Retrieved from https://sites.google.com/site/theipaq/scoring-protocol
- Pellman, E., Viano, D., Tucker, A., Casson, I., Waeckerle, J. (2003). Concussion in professional football: Reconstruction of game impacts and injuries. *Neurosurgery, 53(4),* 799-812.
- Pescatello, L. S. (Ed.). (2014). ASCM's Guidelines for Exercise Testing and Prescription (9th ed.). Baltimore, MD: ACSM.
- Podolin, D. A., Munger, P. A., & Mazzeo, R. S. (1985). Plasma catecholamine and lactate response during graded exercise with varied glycogen conditions. *Journal of Applied Physiology, 71*(4), 1427-1433. Doi: 10.1152/jappl.1991.71.4.1427
- 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 & Science in Sports & Exercise*, *41(4)*, 927-934. Doi: 10.1249/MSS.0v013e3181907d69
- Post, A., & Hoshizaki, T. B. (2015). Rotational acceleration, brain tissue strain, and the relationship to concussion. *Journal of Biomechanical Engineering, 137*(3). Doi: 10.1115/1.4028983
- Reilly, P. (2001). Brain injury: The pathophysiology of the first hours. *Journal of Clinical Neuroscience, 8*(5), 398-403. Doi: 10.1054/jocn.2001.0916
- Riebe, D., Ehrman, J. K., Ligouri, G., & Magal, M. (Eds.). (2017). *ACSM's guidelines for exercise testing and prescription (10th ed.).* Philadelphia, PA: Wolters Kluwer/ Lippincott Williams and Wilkins.
- Rosenthal, M., LaManna, J., Yamada, S., & Somjen, G. (1979). Oxidative metabolism, extracellular potassium and sustained potential shifts in cat spinal cord in situ. *Brain Research, 162,* 113-127.
- Schatz, P., Pardini, J., Lovell, M., Collins, M. W., Podell, K. (2006). Sensitivity and specificity of the ImPACT battery for concussion in athletes. *Archives of Clinical Neuropsychology, 21*(1), 91-99. Doi: 10.1016/j.acn.2005.08.001
- Shaw, N. A. (2002). The neurophysiology of concussion*. Progress in Neurobiology*, *67*, 281- 344.
- Stone, J. L., Patel, V., & Bailes, J. E. (2014). The history of neurosurgical treatment of sports concussion. *Neurosurgery*, 75 (4), 53-523.
- Sufrinko, A., Johnson, E. W., Henry, L.C. (2016). The influence of sleep duration and sleeprelated symptoms on baseline neurocognitive performance among male and female high school athletes. *Neuropsychology, 30*(4), 484-491. Doi: 10.1037/neu0000250
- Van Kampen, D. A., Lovell, M. R., Pardini, J. E., Collins, M. W., & Fu, F. H. (2006). The "value added" of neurocognitive testing after sports-related concussion. *American Journal of Sports Medicine*, *34*(10), 1630-1635.
- Whyte, E., Gibbons, N., Kerr, G., & Moran, K. (2014). The effect of a high intensity, intermittent exercise protocol on neurocognitive function in healthy adults: implications for return to play management following sport related concussion. *Journal of Sport Rehabilitation,* 1-5. Doi: 10.1123/jsr.2014-0201
- Wiese-Bjornstal, D. M., White, A. C., Russsel, H. C., & Smith, A. M. (2015). Psychology of sport concussions. *Human Kinetics*, *4*(2), 169-189. Doi: 10.1123/kr.2015-0012
- Wrisberg, C., & Herbert, W. (1976). Fatigue effects on the timing performance of well practiced subjects. *The Research Quarterly*, *47*(4), 839-844.
- Yang, C.C., Tu, Y. K., Hua, M. S., Huang, S. J. (2007). The association between post-concussion symptoms and clinical outcomes for patients with mild traumatic brain injury. *Journal of Trauma, 62*(3), 657-663. Doi: 10.1097/01.ta.0000203577.668764b8
- Yerkes, R. M., & Dodson, J. D. (1908). The relation of the strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology, 18,* 459-482. Doi: 10.1002/cne.920180503
- Yoon, B., Kravitz, L., & Robergs, R. (2007). VO2 max, protocol duration and the VO2 plateau. *Medince & Science in Sports & Exercise,39*(7), 1186-1192. Doi: 10.1249/mss.0b13e318054e304
- Yoshino, A., Hovda, D., Tatsuro, K., Yoichi, K., & Becker, D. (1991). Dynamic changes in local cerebral glucose utilization following cerebral concussion in rats: Evidence of a hyperand subsequent hypometablolic state. *Brain Research, 561*(1), 106-119. Doi: 10.1016/0006-8993(91)90755-K
- Zuckerman, S.L., Kerr, Z. Y., Yengo-Kahn, A., Wasserman, E., Covassin, T., Solomon, G. S. (2015). Epidemiology of sport-related concussion in NCAA athletes from 2009-2010 to 2013-2014: Incidence, recurrence, and mechanisms. *American Journal of Sports Medicine, 43(11)*, 2654-2662. Doi: 10.1177/0363546515599634

Earn \$40 **CASH**

We are recruiting moderate to highly active individuals ages $18 - 24$ to participate in our study. Email us today to learn more information and see if you qualify to participate.

This study is conducted under the direction of R.J. Elbin, Ph.D. The protocol has been approved by the University of Arkansas Institutional Review Board (IRB) for human subjects in research.

For More Information Contact:

Samantha Mohler

Appendix B- Recruitment Materials (Form Email to individuals responding to flyer)

Hi **FIRST NAME**,

Thanks for responding to the flyer!

To participate in this study individuals (Age 18-26) must:

- Qualify as at least a moderate rating of physical activity based on the International Physical Activity Questionnaire
- Be healthy enough to complete a VO2max assessment

Individuals will not be eligible for participation if they have:

- Diagnosed learning disability
- ADHD
- Psychological disorder (e.g., clinical depression/anxiety)
- History of substance abuse
- Migraine history
- Non-English speaking
- History of concussion (within the last six months) will be

If you agree to participate in the study you will be asked to visit the lab once a week for four consecutive weeks. During these visits you will be asked to complete a maximal exertion treadmill test followed by a standardized computer concussion test. The max exertion test will be a progressive run on a treadmill; it usually takes individuals about 10-15 minutes to reach their maximum effort. After the completion of the fourth visit to the lab you will be given **\$40.00.**

If you are still interested and qualify we would be more than happy to include you in the study! The next step is filling out screening forms. Do you have time tomorrow to swing by my office (HPER 219) and fill them out?

--- Thanks,

Sam

Appendix C- University of Arkansas Medical History Questionnaire

Appendix C- University of Arkansas Medical History Questionnaire, continued

INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE (October 2002)

LONG LAST 7 DAYS SELF-ADMINISTERED FORMAT

FOR USE WITH YOUNG AND MIDDLE-AGED ADULTS (15-69 years)

The International Physical Activity Questionnaires (IPAQ) comprises a set of 4 questionnaires. Long (5 activity domains asked independently) and short (4 generic items) versions for use by either telephone or self-administered methods are available. The purpose of the questionnaires is to provide common instruments that can be used to obtain internationally comparable data on health-related physical activity.

Background on IPAQ

The development of an international measure for physical activity commenced in Geneva in 1998 and was followed by extensive reliability and validity testing undertaken across 12 countries (14 sites) during 2000. The final results suggest that these measures have acceptable measurement properties for use in many settings and in different languages, and are suitable for national population-based prevalence studies of participation in physical activity.

Usina IPAQ

Use of the IPAQ instruments for monitoring and research purposes is encouraged. It is recommended that no changes be made to the order or wording of the questions as this will affect the psychometric properties of the instruments.

Translation from English and Cultural Adaptation

Translation from English is encouraged to facilitate worldwide use of IPAQ. Information on the availability of IPAQ in different languages can be obtained at www.ipaq.ki.se. If a new translation is undertaken we highly recommend using the prescribed back translation methods available on the IPAQ website. If possible please consider making your translated version of IPAQ available to others by contributing it to the IPAQ website. Further details on translation and cultural adaptation can be downloaded from the website.

Further Developments of IPAQ

International collaboration on IPAQ is on-going and an International Physical Activity Prevalence Study is in progress. For further information see the IPAQ website.

More Information

More detailed information on the IPAQ process and the research methods used in the development of IPAQ instruments is available at www.ipaq.ki.se and Booth, M.L. (2000). Assessment of Physical Activity: An International Perspective. Research Quarterly for Exercise and Sport, 71 (2): s114-20. Other scientific publications and presentations on the use of IPAQ are summarized on the website.

INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the last 7 days. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the vigorous and moderate activities that you did in the last 7 days. Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal.

PART 1: JOB-RELATED PHYSICAL ACTIVITY

The first section is about your work. This includes paid jobs, farming, volunteer work, course work, and any other unpaid work that you did outside your home. Do not include unpaid work you might do around your home. like housework, yard work, general maintenance, and caring for your family. These are asked in Part 3.

 $1.$ Do you currently have a job or do any unpaid work outside your home?

The next questions are about all the physical activity you did in the last 7 days as part of your paid or unpaid work. This does not include traveling to and from work.

During the last 7 days, on how many days did you do vigorous physical activities like $\overline{2}$ heavy lifting, digging, heavy construction, or climbing up stairs as part of your work? Think about only those physical activities that you did for at least 10 minutes at a time.

How much time did you usually spend on one of those days doing moderate physical 5. activities as part of your work?

 $6.$ During the last 7 days, on how many days did you walk for at least 10 minutes at a time as part of your work? Please do not count any walking you did to travel to or from work.

7.

PART 2: TRANSPORTATION PHYSICAL ACTIVITY

These questions are about how you traveled from place to place, including to places like work, stores, movies, and so on.

8. During the last 7 days, on how many days did you travel in a motor vehicle like a train, bus, car, or tram?

No bicycling from place to place

Skip to question 12

 $11.$ How much time did you usually spend on one of those days to bicycle from place to place?

 $12.$ During the last 7 days, on how many days did you walk for at least 10 minutes at a time to go from place to place?

 $13.$ How much time did you usually spend on one of those days walking from place to place?

PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

This section is about some of the physical activities you might have done in the last 7 days in and around your home, like housework, gardening, yard work, general maintenance work, and caring for your family.

14. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, chopping wood, shoveling snow, or digging **in the garden or yard**?

17. How much time did you usually spend on one of those days doing moderate physical activities in the garden or yard?

19. How much time did you usually spend on one of those days doing moderate physical activities inside your home?

1

PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVITY

This section is about all the physical activities that you did in the last 7 days solely for recreation, sport, exercise or leisure. Please do not include any activities you have already mentioned.

20. Not counting any walking you have already mentioned, during the last 7 days, on how many days did you walk for at least 10 minutes at a time in your leisure time?

23. How much time did you usually spend on one of those days doing vigorous physical activities in your leisure time?

PART 5: TIME SPENT SITTING

The last questions are about the time you spend sitting while at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading or sitting or lying down to watch television. Do not include any time spent sitting in a motor vehicle that you have already told me about.

26. During the last 7 days, how much time did you usually spend sitting on a weekday?

hours per day minutes per day

During the last 7 days, how much time did you usually spend sitting on a weekend 27. day?

hours per day $-$ minutes per day

This is the end of the questionnaire, thank you for participating.

Appendix E- International Physical Activity Questionnaire Long Form Scoring Protocol

Scoring of the IPAQ

Microsoft Excel was used to calculate total metabolic expenditure, metabolic calculations

and categorization of activity level are based off of guidelines provided by IPAQ on their

website (https://sites.google.com/site/theipaq/home). Table 8 outlines the qualifications for each

activity level.

Table 8

Note: Adapted from Patterson, 2005.

Appendix F- 24-Hour History Intake Form

24-Hour History

Appendix G - 24 -Hour Diet Record

Human Performance Laboratory

Maximal Graded Exercise Test Results

Determining the Appropriate Timing of Administration for Baseline Computerized Neurocognitive

Testing (CNT) Following Maximal Exertion

Pre-test compliance: Pres

Estimated Mile Time:

Total Exercise Time:

Tech Initials:

VO₂ Max:

NOTES:

Appendix I-Verbal encouragement used during graded exercise maximal exertion test.

In order to elicit best performance, verbal encouragement was used throughout the physical exertion trial. However, participants were not allowed to listen to music and the verbal encouragement was limited to the list provided below in Table 10.

Table 10

Human Performance Laboratory

Recovery and CNT Information

Determining the Appropriate Timing of Administration for Baseline Computerized Neurocognitive

Testing (CNT) Following Maximal Exertion

Subject #

Date:

ImPACT test: P1 P2 P3 P4

RECOVERY INTERVAL: IMMEDIATE 10 MINUTES 20 MINUTES BASELINE

Tech Signature: The Contract of the Contract o

Office for Sport Concussion Research

Date: $\qquad \qquad$

Please check your current activity level:

□ Sedentary (little or no exercise)

□ Lightly active (light exercise/sports 1-3 days/week)

 \square Moderately active (moderate exercise/sports 3-5 days/week)

 \Box Very active (hard exercise/sports 6-7 days a week)

 \Box Extra active (very hard exercise/sports & physical job or 2x training)

AFTER YOU COMPLETE THE TEST SESSION

Please CIRCLE your effort (i.e., how hard did you try) while taking this test:

Appendix L- Institutional Review Board Approval Letters

Office of Research Compliance **Institutional Review Board**

March 21, 2016

Your protocol has been approved by the IRB. Protocols are approved for a maximum period of one year. If you wish to continue the project past the approved project period (see above), you must submit a request, using the form Continuing Review for IRB Approved Projects, prior to the expiration date. This form is available from the IRB Coordinator or on the Research Compliance website (https://vpred.uark.edu/units/rscp/index.php). As a courtesy, you will be sent a reminder two months in advance of that date. However, failure to receive a reminder does not negate your obligation to make the request in sufficient time for review and approval. Federal regulations prohibit retroactive approval of continuation. Failure to receive approval to continue the project prior to the expiration date will result in Termination of the protocol approval. The IRB Coordinator can give you guidance on submission times.

This protocol has been approved for 200 participants. If you wish to make any modifications in the approved protocol, including enrolling more than this number, you must seek approval prior to implementing those changes. All modifications should be requested in writing (email is acceptable) and must provide sufficient detail to assess the impact of the change.

If you have questions or need any assistance from the IRB, please contact me at 109 MLKG Building, 5-2208, or irb@uark.edu.

Appendix L- Institutional Review Board Approval Letters Continued

Office of Research Compliance **Institutional Review Board**

February 14, 2017

MEMORANDUM

Your request to extend the referenced protocol has been approved by the IRB. If at the end of this period you wish to continue the project, you must submit a request using the form Continuing Review for IRB Approved Projects, prior to the expiration date. Failure to obtain approval for a continuation on or prior to this new expiration date will result in termination of the protocol and you will be required to submit a new protocol to the IRB before continuing the project. Data collected past the protocol expiration date may need to be eliminated from the dataset should you wish to publish. Only data collected under a currently approved protocol can be certified by the IRB for any purpose.

This protocol has been approved for 200 total participants. If you wish to make any modifications in the approved protocol, including enrolling more than this number, you must seek approval prior to implementing those changes. All modifications should be requested in writing (email is acceptable) and must provide sufficient detail to assess the impact of the change.

If you have questions or need any assistance from the IRB, please contact me at 109 MLKG Building, 5-2208, or irb@uark.edu.