

1-1-2016

# Neurocognitive Implications of Sport-Related Concussion in High School Athletes Over-Time

Evan Lee Smith

*Nova Southeastern University*, [evan.smith1105@gmail.com](mailto:evan.smith1105@gmail.com)

This document is a product of extensive research conducted at the Nova Southeastern University [College of Psychology](#). For more information on research and degree programs at the NSU College of Psychology, please [click here](#).

Follow this and additional works at: [http://nsuworks.nova.edu/cps\\_stueta](http://nsuworks.nova.edu/cps_stueta)



Part of the [Psychology Commons](#)

## Share Feedback About This Item

---

### NSUWorks Citation

Smith, E. L. (2016). Neurocognitive Implications of Sport-Related Concussion in High School Athletes Over-Time. .  
Available at: [http://nsuworks.nova.edu/cps\\_stueta/79](http://nsuworks.nova.edu/cps_stueta/79)

This Dissertation is brought to you by the College of Psychology at NSUWorks. It has been accepted for inclusion in College of Psychology Theses and Dissertations by an authorized administrator of NSUWorks. For more information, please contact [nsuworks@nova.edu](mailto:nsuworks@nova.edu).

**Neurocognitive Implications of Sport-Related Concussion in  
High School Athletes Over-Time**

**By**

**Evan L. Smith, M.S.**

A Dissertation Presented to the College of Psychology of Nova Southeastern University in  
Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy

NOVA SOUTHEASTERN UNIVERSITY

2016

**DISSERTATION APPROVAL SHEET**

This dissertation was submitted by Evan L. Smith under the direction of the Chairperson of the dissertation committee listed below. It was submitted to the College of Psychology and approved in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Clinical Psychology at Nova Southeastern University.

Approved:

\_\_\_\_\_  
Date of Defense

\_\_\_\_\_  
Stephen Russo, Ph.D., Chairperson

\_\_\_\_\_  
Barry Nierenberg, Ph.D., ABPP (Rp)

\_\_\_\_\_  
Ryan Black, Ph.D.

\_\_\_\_\_  
Date of Final Approval

\_\_\_\_\_  
Stephen Russo, Ph.D.

## ACKNOWLEDGEMENTS

I have been truly fortunate to have been afforded several opportunities in my life thus far, and am overwhelmed with the support I've received from my family, friends, faculty and others during my pursuit of a doctoral degree in clinical psychology. Without the encouragement and guidance of those closest to me, my accomplishments, including the completion of my dissertation, would not be possible. It is with those who have shared in my journey that I gain the deepest satisfaction in sharing my achievements. My gratitude for several individuals extends well past the limits of this book. I am humbled to acknowledge many of them and will attempt to express how deeply influenced I've been by their contributions to my life.

Unequivocally, without the unconditional love and support from my family that I have received for the past 29 years, I would not be the human being I am today. My mom, Bonnie, has been, continues to be, and will always be the engine that drives my success. In moments that I have confronted challenges, her unwavering love, intelligence and persistence has refused to let me flounder. I have profound gratitude for my dad, Gary's, unwavering support for me to pursue what drives my passion and helped me realize that failure is a building block for success. I have learned an immeasurable number of lessons from him that cannot be learned in any classroom or textbook. Their generosity cannot be overstated. To my older sister, Shauna, her and Doug's support is always a phone call away, no matter the circumstance or time. I am lucky to have learned from her what bravery looks like from an early age and use those lessons to persevere when times are "tough" for me. Being my oldest sister, Michelle has led by example and she has always been there to show me the ropes on the road to a career in psychology, as well as my sounding board to decompress and keep me sane. Whether it be a 5-minute FaceTime call, holiday cards or check-ins, I'm grateful for the efforts she's made to keep me involved with her,

Chris, and Olivia's lives. As evidence that it truly takes a village, I've also been molded by being raised in-part by my other family members, including Grandma Maye & Rita (the "5-foot-tall giants" of our family), Grandpa Bernie, Aunts April & Cindy, Godfather Gary and Uncle Donny. Although I never met Grandpa Simon, from the stories I've heard, he instilled a backbone into our family that was imparted to me, and for that I am thankful.

Academically, I am pleased to recognize Dr. Stephen Russo for his generosity, taking me on as a Ph.D. candidate and subsequently engaging me to challenge myself clinically, through research, and in completing my dissertation. His unique appreciation for my background as a student-athlete, "down-to-earth" personality, and vast knowledge of psychology assisted me to develop both professionally and personally throughout my graduate school career. My expert procrastination skills were met and exceeded by his honesty and support in guiding me through the completion of my doctoral degree. My introduction Dr. Barry Nierenberg during my second year profoundly changed my career trajectory, and he is solely responsible for my introduction to rehabilitation psychology. I have benefited from his tremendous enthusiasm for training and positive outlook, frequently convincing me of how competent I am, in spite of how much I may overlook. My knowledge of statistics and completion of this dissertation can be attributed to the deft training I received from Dr. Ryan Black. His excitement for research is unmatched in my experience and something I have admired in working with him during my academic career. As Dr. Nierenberg likes to reference, "If I have seen further it is by standing on the shoulders of giants." Standing a 5'7" I've been privileged by the opportunity to stand on such sturdy shoulders of these wonderful and caring mentors.

I've found that it is not where you are, but who you are with that makes the experience worth-while. I would never have learned a balance of professional life and self-care without the

connections I've made during my graduate career. Sean Coad, Dee Nwosu, Caroline Spearman, Doug Polster and my group of intern peers (among several others) have been my surrogate family members and I'm honored to have made such close and meaningful friendships over the years. While home and abroad, Vincent Maltese and Miguel Roxas, are lifelong friends who can never be replaced. From elementary school through graduate school and beyond, I am grateful to the people who have chosen to join me on this ride.

**TABLE OF CONTENTS**

Acknowledgements.....	iii
List of Tables and Figures.....	x
Abstract.....	1
Chapter I: Statement of the Problem.....	2
Chapter II: Review of the Literature.....	6
Concussion from a Psychobiological Perspective.....	6
Neuro-mechanical etiology of concussion.....	6
mTBI and the “energy crisis”.....	7
Diagnostic Evaluation of Concussion.....	9
Adolescent Neurocognitive Development and Impairment.....	10
ImPACT® studies with high school athletes.....	11
Risk Factors Associated with Repeated Blows to the Head.....	13
Second-Impact Syndrome (SIS).....	14
Chronic Traumatic Encephalopathy (CTE).....	15
Future Research.....	19
Purpose of the Study.....	19
Chapter III: Methods.....	23
Participants.....	23
Materials and Procedure.....	24
Neurocognitive testing.....	24
Neurocognitive evaluations.....	24
Sections.....	24

Post-Concussion Symptom Scale (PCSS).....	25
Modules.....	25
Composite Subscale Scores.....	27
Proposed Analyses.....	30
Effect Size Considerations.....	30
Hypothesis I.....	31
Hypothesis II.....	31
Hypothesis III.....	32
Hypothesis IV.....	33
Hypothesis V.....	34
Chapter IV: Results.....	36
Statistical Plan.....	36
Selection Criteria.....	36
Demographic Information.....	37
Analyses Employed.....	39
Hypothesis I.....	39
Verbal Memory (VeM).....	40
Visual Memory (ViM).....	40
Visual Motor Speed (VMS).....	40
Reaction Time (RT).....	41
Impulse Control (IC).....	41
Post-Concussion Symptom Scale (PCSS).....	41
Hypothesis II.....	41



Verbal Memory (VeM).....	42
Visual Memory (ViM).....	43
Visual Motor Speed (VMS).....	43
Reaction Time (RT).....	44
Impulse Control (IC).....	44
Post-Concussion Symptom Scale.....	45
Hypothesis III.....	46
Verbal Memory (VeM).....	47
Visual Memory (ViM).....	48
Visual Motor Speed (VMS).....	49
Reaction Time (RT).....	50
Impulse Control (IC).....	51
Post-Concussion Symptom Scale.....	52
Hypothesis IV & V.....	54
Chapter V: Discussion.....	55
Overall Findings.....	55
Uniqueness of Sample.....	55
Hypothesis I.....	56
Hypothesis II.....	57
Hypothesis III, IV & V.....	58
Implications.....	58
Chronic Disease Model Interpretation.....	58
Clinical Implications.....	59

Limitations.....	61
Method Biases.....	62
Future Directions.....	63
References.....	64

**LIST OF TABLES AND FIGURES**

## Tables

Table 1: Frequency of Age in Sample.....	38
Table 2: Frequency of Concussion in Sample.....	38
Table 3: Demographics and Other Characteristics of Sample.....	39
Table 4: Descriptive Statistics and RM ANOVA for Non-Concussed Sample.....	40
Table 5: Descriptive and BS ANOVA Statistics Amongst Concussion Groups.....	40
Table 6: Descriptive and MM ANOVA Statistics Amongst Concussion Groups....	47

## Figures

Figure 1: Neuro-metabolic Cascade Following Experimental Concussion.....	8
Figure 2: Neuropathological Findings in a Brain with Stage IV CTE.....	18
Figure 3: Path Model of Moderation.....	33
Figure 4: Path Model of Indirect Effects.....	35
Figure 5: Selection Criteria Flow Chart.....	37
Figure 6: Verbal Memory by Concussion Group Over-Time.....	48
Figure 7: Visual Memory by Concussion Group Over-Time.....	49
Figure 8: Visual Motor Speed by Concussion Group Over-Time.....	50
Figure 9: Reaction Time by Concussion Group Over-Time.....	51
Figure 10: Impulse Control by Concussion Group Over-Time.....	52
Figure 11: PCSS by Concussion Group Over-Time.....	53

**Neurocognitive Implications of Sport-Related Concussion in  
High School Athletes Over-Time**

**By**

**Evan L. Smith, M.S.**

Nova Southeastern University

The identification of sport-related concussion (mild traumatic brain injury [mTBI]), its neurocognitive sequelae, and subsequent management have become a top priority within a spectrum of research disciplines at the intersection of psychology and sports medicine. To properly understand the complex neurocognitive changes associated with sport-related concussion in high school age individuals, multiple aspects of the injury were explored including the psychobiological nature of the injury, diagnostic concerns, normative adolescent neurocognitive development and abnormal changes as a result of the injury, and risk for further injury. While a wealth of literature exists in these areas, one aspect in particular, neurocognitive changes associated with sport-related mTBI in adolescents, is the focus of this research study. A review of the current research reveals a lack of exploration into neurocognitive deficits over-time as early as adolescence. To advance the understanding of how sport-related concussions may influence neurocognitive performance during this vulnerable age for brain development, multiple group comparisons were conducted to determine differences based upon reported concussion history. Results suggest that adolescents who experience sport-related concussion demonstrate significantly reduced levels of neurocognitive performance in several domains on initial baseline testing. Furthermore, these findings generally persist upon follow-up neurocognitive testing during adolescence. Thus, persistent neurocognitive deficits found during adolescence may have profound implications for brain development and concussion management.

## Chapter I

### Statement of the Problem

An estimated 38 million children and adolescents participate annually in organized sports across the United States (US) (Daneshvar, Nowinski, McKee & Cantu, 2011). While participation in sport is associated with several psychobiological benefits such as improved cardiovascular and metabolic disease risk profiles, reduced body fat, increased cardiovascular fitness and improved psychological health (Eime et al., 2013), over the past two decades there has been increasing awareness that sport-related concussions, or mild traumatic brain injuries (mTBI), are a common consequence of sport participation. In the US alone, the estimated rates of sport-related concussion range as high as 3.8 million incidents (Barkhoudarian, Hovda, & Giza, 2011). Of even greater concern is the increasing scientific evidence which suggests that concussions can result in long-term neurological problems and may induce persistent reductions in neurocognitive functioning (Daneshvar et al., 2011).

Concern for player safety has remained at the forefront of high-collision sports such as football, boxing, rugby, and ice hockey. In the early 20<sup>th</sup> Century, the National Collegiate Athletic Association (NCAA) was convened to implement stricter player safety regulations after 19 athletes were killed or paralyzed playing football (Dunn, Dunn & Day, 2006). However, it wasn't until recently that formal concussion policies and procedures were implemented by collegiate and professional governing bodies, including the National Hockey League (NHL) in 1995, National Football League (NFL) in 2008 (revised in 2010) and NCAA in 2010 (Tomei et al., 2012).

Despite the emphasis on sports such as football and ice hockey, sport-related brain injury (i.e., concussion) is also evident in limited-contact and non-contact sports such as soccer,

basketball, baseball and tennis. Nonfatal sport-related brain injuries pose a serious public health issue, as the Center for Disease Control (CDC) reported more than 207,000 emergency room visits related to these injuries from 2001 to 2005 (CDC, 2009). Historically, definitions of concussion and guidelines for concussion management have widely varied. Typically, these definitions utilized loss of consciousness (LOC) to determine “grade”, or severity, of concussive injury. However, these systems lacked consistency and empirical support (Collins & Hawn, 2002). The inadequacy of these concussion management guidelines were re-examined and defined in 2001 when the International Ice Hockey Federation, Federation Internationale de Football Association (FIFA) and the International Olympic Committee, held an international conference in Vienna, Austria (Aubry et al., 2001). Understanding of concussive brain injury continues to evolve. To better understand the distinction between concussive and sub-concussive brain injury, we will explore the contemporary definition of “concussion”. For the purposes of this dissertation, mTBI and concussion will be used interchangeably.

The American Medical Society for Sports Medicine (AMSSM) currently defines a concussion as a “traumatically induced transient disturbance of brain function and involves a complex pathophysiological process. Concussion is a subset of mild traumatic brain injury (mTBI) which is generally self-limited and at the less-severe end of the brain injury spectrum,” (Harmon et al., 2013). This definition is derived from a growing literature supporting a complex neurobiological cascade of events in the brain triggered by the occurrence of the injury. Evolving from previous descriptions, LOC is no longer a prerequisite for the incidence of brain injury and, based on the most recent international summit on concussion, McCrory et al. (2013) defines a concussion as a “complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces”. More comprehensively, the authors identify several common features that

incorporate the clinical, pathologic and biomechanical injury constructs that are utilized in defining the nature of a concussive brain injury:

1. Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head.
2. Concussion typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously. However, in some cases, symptoms and signs may evolve over a number of minutes to hours.
3. Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging studies.
4. Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course. However, it is important to note that, in a small percentage of cases, symptoms may be prolonged. (p. 250-251)

Each facet of the definition provides a contributory perspective to the overall complexity of our current understanding of sport-related mTBI. When considering the breadth of the current definition, it becomes crucial to understand the implications of brain injury amongst developing athletes.

To better understand the implications of brain injury within a high school population a variety of aspects will be discussed, including the psychobiological consequences of the injury and the unique set of protective and risk factors that exist for youth and adolescents who incur sport-related concussions. Risk factors such as repetitive blows to the brain have been documented in former professional athletes, but have not been fully considered within the

context of the neurocognitive development of youth and adolescent athletes. For example, Brooks et al. (2013) examined the lingering effects of concussion history in adolescent hockey players and found that those players who report two or more concussions report more symptoms of concussion at future baseline testing. These results suggest that high school athletes are likely suffering the cumulative effects of multiple concussive injuries earlier than what has been reported in the post-concussion syndrome (PCS) and chronic traumatic encephalopathy (CTE) literature. Additionally, a review of the current research reveals a lack of exploration into these deficits over-time as early as adolescence. Given the lack of literature examining potential risk for ongoing neurocognitive dysfunction in those athletes who incur multiple concussions at an early age, empirical exploration of diagnostic concerns and concussive management guidelines as well as the implications of repeated mTBI in high school athletes will be examined in this dissertation.



## Chapter 2

### Review of the Literature

#### Concussion from a Psychobiological Perspective

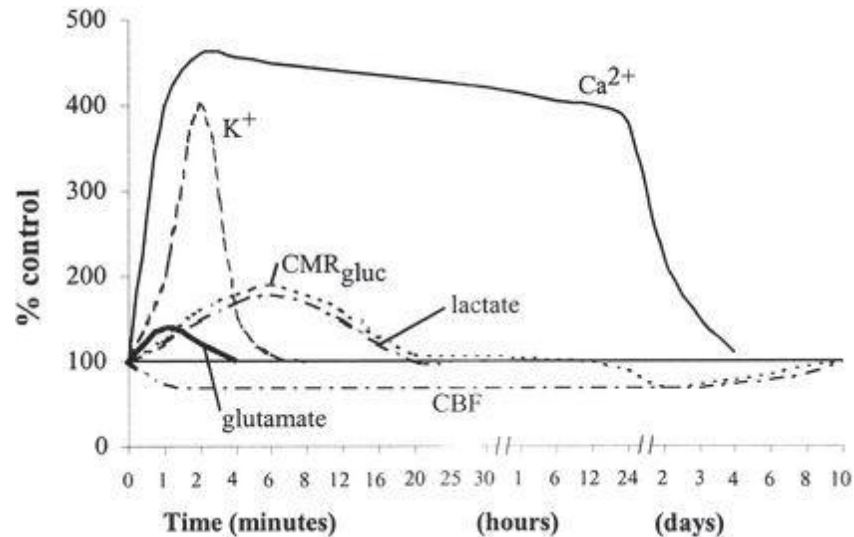
The research on sport-related concussion has provided both quantitative and qualitative data regarding the clinical presentation of the injury. The subsequent advancement has allowed clinicians and researchers to gain a better understanding of the influence of brain dysfunction on return-to-play (RTP) guidelines for athletes. Critical to the advancement of clinical research was the establishment of a comprehensive understanding of the biological, chemical and metabolic “cascade” that follows a concussive injury (Giza & Hovda, 2001). As such, the neurological repercussions of mTBI are thoroughly discussed in the literature using animal and human subjects. First, research will be explored to provide a description of the biomechanical processes resulting from concussion and mTBI. Next, the cascade of neurological events, known as the “energy crisis” will be examined to understand the signs and symptoms of sport-related concussion.

**Neuro-mechanical etiology of concussion.** A concussion is a form of mTBI which occurs when the brain is subjected to rapid acceleration and deceleration forces causing displacement of the cerebral spinal fluid (CSF) that surrounds the brain and may result in the brain making impact with the skull. Injury sustained to the brain at the site of impact is known as a coup injury, while injury occurring on the opposite side is referred to as a contra-coup or whiplash injury. Additionally, rotational forces on the brain may cause the tissue (i.e., axons and dendritic connections) to elongate, deform, or shear within the neurological tracts of the cranium. As a result of the traumatic injury, damage occurs to neuronal cell bodies, axons, dendrites, blood vessels and glial cells, disrupting neurological communication within the brain

(Povlishock, 1992). The literature concerning the neurobiological etiology of concussion through axonal injury and the subsequent metabolic cascade of dysfunction is detailed below.

**mTBI and the “energy crisis”.** A distinction is made in the literature between moderate-severe traumatic brain injury (TBI) and mTBI, as structural damage or injury are not common with mTBI. Neurons in the brain do not typically die as they do in TBI, but rather, they become stretched and torqued, causing a complex cascade of ionic, metabolic and physiologic events (Giza & Hovda, 2001). In discussing the metabolic changes that occur immediately after a biomechanical injury to the brain, the authors detail an abrupt shift in ionic fluxes that cause depolarization, with the unchecked efflux of potassium ( $K^+$ ) and influx of calcium ( $Ca^{2+}$ ). The resulting changes in cellular physiology cause the sodium-potassium ( $Na^+-K^+$ ) pump within the cell to trigger a massive increase in glucose metabolism, as it is starved for adenosine triphosphate (ATP). Because vasospasming of the cerebral arteries also occurs following a concussive injury, an “energy crisis” is created within the cell as the disparity between glucose supply and demand increases. The concussed brain experiences a period of depressed metabolism immediately following the “hyper-metabolism”, contributing further to the energy crisis as  $Ca^{2+}$  may impair mitochondrial oxidative metabolism. This unchecked  $Ca^{2+}$  accumulation may even lead to cell death. Below is a figure illustrating the abrupt metabolic cascade of events in a concussed brain. It is likely that during this period, the brain becomes vulnerable to additional impacts (Giza & Hovda, 2001).

Figure 1: Neuro-metabolic Cascade Following Experimental Concussion



*Figure 1.* Neurometabolic cascade following experimental concussion. K<sup>+</sup>, potassium; Ca<sup>2+</sup>, calcium; CMR<sub>gluc</sub>, oxidative glucose metabolism; CBF, cerebral blood flow. (Reprinted with permission. Giza CC, Hovda DA. Ionic and metabolic consequences of concussion. In: Cantu, R. C. & Cantu, R. I., (2000)). Neurologic Athletic and Spine Injuries. St Louis, MO: WB Saunders Co; p.80–100.).

Typically, mTBI symptoms resolve within a few weeks. McRea et al. (2009) suggests that 85% of athletes average 17.49 (SD = 1.6) years of age evidence full neurocognitive and symptomatic recovery from concussive symptoms within 1 week. Fewer than 3% of subjects reported concussive symptomatology one month post-injury. Should the signs and symptoms of concussion persist for up to six months, this could indicate more diffuse damage within the brain and is known as Post-Concussion Syndrome (PCS). Impairments are persistent and more severe in most cases, including dizziness, hearing loss, sleep disorders, loss of taste or smell, attention deficits, and other difficulties with memory (Kushner, 1998). Given an understanding of the biopsychological etiology of brain dysfunction following a concussion, a review of detection methods and protocols is outlined below.

### **Diagnostic Evaluation of Concussion**

The neurological underpinnings of head trauma reveal a myriad of signs and symptoms that have been established as important in the diagnosis of concussion. Aubry et al. (2002) refers to the First International Conference on Concussion in Sport (ICCS) as establishing the first guidelines for systematic concussion evaluation, including ten protocols: clinical history, evaluation, neuropsychological testing, imaging procedures, research methods, management and rehabilitation, prevention, education, future directions and medico-legal considerations. Now in its fourth iteration, the 2012 ICCS in Zurich, Germany, supplemented the findings of the previous conferences and refinement of the definition of concussion. Recommendations for diagnosis and ongoing evaluation include sideline testing of athletes suspected of being injured, restrictions on RTP times, assessment of psychological comorbidities, and more stringent regulations for athletes under the age of 18. As outlined by the 2012 ICCS, diagnosis of concussion will include one or more of the following: cognitive impairments, sleep disturbance, behavioral changes or somatic, cognitive and/or emotional symptoms (McCrory et al., 2013).

For high school athletes, the evidence of brain dysfunction and prognosis for recovery informs the current rules and regulations for return-to-play (RTP) decisions. RTP guidelines established at the 2012 ICCS denote a six step process that injured high school athletes must achieve before final RTP clearance. McCrory et al (2013) outlined the updated RTP as such: (1) The athlete must rest to alleviate all cognitive, emotional and physical symptoms. This is achieved by refraining from physical and cognitive stress (e.g. limiting interactions with friends, homework, or television). (2) Return to light aerobic exercise (e.g. walking or swimming) to assess whether symptoms return as heart rate increases. (3) Engage in sport-specific exercise (e.g. light lifting or jogging). (4) Engage in non-contact training and complex drills (e.g. heavy

lifting or pitching drills) to determine if symptoms return under strenuous exercise and cognitive load. (5) Full contact return to practice, pending medical clearance to expose the athlete to game-like conditions under supervision. (6) Athlete is cleared to RTP pending symptom-free completion of steps 1-5. To begin RTP protocol, the high school athlete must present as non-symptomatic and meet baseline neurocognitive functioning as outlined below. Moreover, an athlete must be symptom-free for 24 hours following each step of the RTP progression.

### **Adolescent Neurocognitive Development and Impairment**

To better understand the neurocognitive risk incurred by adolescents with a history of concussion, normative neurocognitive development in adolescents is examined next. In particular, Connors et al. (2003) provides an examination of normative reaction time (RT) and impulse control performance amongst a normative epidemiological sample (N=816) of 9-17-year-old children. Utilizing the continuous performance test (CPT), the authors demonstrate main effects of improved performance in RT and impulse control inhibition as age increases amongst this healthy sample. Additionally, gender main effects reveal males evidenced faster RT and more impulsive errors when compared to female counterparts. Additionally, this finding is corroborated by an electroencephalography (EEG) study (Johnstone et al., 2005) demonstrating improved RT with age and identifies event related potentials (ERP) utilized in activation and inhibition associated with these findings. These findings suggest that additional research is warranted into exploring the effects of neurocognitive functioning amongst several demographic variables (e.g., age, gender, and ethnicity).

Given the developmental changes that occur in high school-age athletes, it is important to recognize the unique set of risk and protective factors that mediate the relationship between normative development and potential dysfunction caused by mTBI. Brain development in this

age group is accentuated by the growth of the executive function system. Proliferations of neurons in the prefrontal cortex and limbic system during adolescence influence the development of cognitive functioning, planning and behaviors. A higher-order control system, executive functioning (EF) regulates lower-order brain functions (e.g. short-term memory, sensory perceptions, language and motor skills) and organizes future/goal-oriented thinking and behaviors (Pokhrel et al., 2013). Additionally, this increased aptitude for sequencing of actions towards a goal is an outward expression of acquiring abstract reasoning skills at this age. Differential acquisition of these skills is common in this age group as individuals mature at varying rates (Spear, 2000). Injury to the brain during this critical period in neurocognitive development could be uniquely detrimental and is explored below.

**ImPACT® studies with high school athletes.** Not fully developed, adolescents may exhibit poorer cognitive performance under stress, both environmental and biological (Spear, 2000). Orthopedic injuries may differ from neurological injuries in diagnosis, prognosis and symptomology. Additionally, coping strategies and recovery from concussion may vary within this population. For these reasons, it is crucial to examine the dysfunction that occurs in high school-age athletes when recovering from a concussion.

Exploration of the neurocognitive effects of concussion in youth athletes delineates between concussion (mTBI) and orthopedic injury (OI) (Reiger et al., 2013). Utilizing a sample of 69 parent-child pairs (39 mTBI, 30 OI), injured adolescents participated in initial ImPACT® testing (child) and the Behavior Rating Inventory of Executive Function (BRIEF) (parent) in the emergency room. 18 participants withdrew from the study and the remaining 51 participated in 3-month follow up ImPACT® testing and the BRIEF. As expected, mTBI youth endorsed more post-concussion symptoms and performed poorer on initial ImPACT® testing. However, BRIEF

results did not distinguish groups. Neurocognitive measures did not differentiate between OI and mTBI groups at 3-month follow up, except for visual memory scores, which remained impaired in the mTBI group comparatively. Results suggest that although neurocognitive differences existed between groups, parents may not accurately assess child functioning during recovery from mTBI.

Covassin et al. (2013) examined the relationship between neurocognitive performance and reported symptoms with coping responses. A sample of 104 concussed athletes ( $M = 16.41$ ,  $SD = 2.19$  years) was comprised of 76% high school ( $n = 79$ ) and 24% collegiate ( $n = 25$ ) athletes. Each participant completed a baseline neurocognitive functioning measure (ImPACT®) and post-concussion ImPACT® with a measure of coping responses during recovery from concussion (Brief COPE) approximately 3 and 8 days post-injury. Results were compared using a regression model and revealed that concussed athletes reported higher levels of avoidance coping behaviors (e.g. denial, venting, behavioral disengagement) with lower cognitive functioning scores (Visual Memory) on the ImPACT®. In addition, athlete self-report of self-blame, self-distraction and religion resources was higher at 3 days compared to 8 days post-concussion. Implications for these findings suggest that high school and collegiate athletes may use maladaptive avoidant coping strategies when recovering neurocognitive function due to concussion.

During high-collision sports, athletes accept the risk of acute injury as a risk of gameplay. Research with moderate and mild-collision sports is unclear regarding the inherent risk of participation in sport. For example, Lovell & Solomon (2013) found that 61% of cheerleaders examined for concussion ( $n = 138$ ) reported an increase in symptoms compared with baseline ImPACT® testing. As a group, neurocognitive performance 7 days post-concussion remained

significantly declined relative to baseline testing ( $F = 6.5$ ,  $p < .001$ ). Kontos et al. (2011) investigated the relationship between soccer heading and neurocognitive functioning and symptoms in youth soccer players. A sample consisted of 63 athletes (27 females, 36 males) aged 13 to 18 ( $M = 15.89$ ,  $SD = 1.17$ ) who were observed during practice and game and grouped into heading exposure groups (low, moderate, high) based on average number of headers taken. Participants completed ImpACT® testing and results revealed no differences between groups. While each group outperformed the 10<sup>th</sup> percentile normative group, high frequency males demonstrated slower processing speed scores than females. Authors suggest that the effects of heading in youth soccer may be subtle and further research is warranted at higher levels of play.

The literature suggests that youth participation in sport demonstrates inherent risks for injury. Acute mild brain injury (mTBI) symptoms may take longer to resolve in a high-school age population and require different intervention and prevention protocol from OI. As symptoms and cognitive dysfunction appear to have more deleterious effect during recovery from mTBI in this population, additional examination of literature regarding repetitive blows to the head is described below.

### **Risk Factors Associated with Repeated Blows to the Head**

As discussed, the signs, symptoms and neurophysiology of impairment resulting from mTBI are usually self-limiting and resolve spontaneously over a period of several weeks. In high school athletics, return to play (RTP) protocols have been established to minimize the risk of prolonged neurocognitive and somatic deficits resulting from sport-related concussion. However, impact injuries less severe than concussion may occur during participation in sport that do not produce overt neurological symptoms. These injuries to the brain are associated with subtle neuropsychiatric deficits or changes in functional magnetic resonance imaging (fMRI) and are



referred to as “sub-concussive” hits (Gysland et al., 2012). In addition, many sub-concussive symptoms and signs may be fleeting and go undetected following the immediate neurological insult. For this reason, players, coaches, parents and medical staff, may overlook mild sport-related head injury, as they do not produce immediate observable concerns. Consequently, the athlete may put him or herself at greater risk for long-term dysfunction if repeated exposure to mTBI, concussion and sub-concussive hits are undiagnosed (McCrorry et al., 2009). The risk factors associated with repetitive blows to the head, including the development of Second Impact Syndrome (SIS) and Chronic Traumatic Encephalopathy will be explored.

**Second-Impact Syndrome (SIS).** Essential to the treatment of sport-related concussion is the accuracy, sensitivity and enforcement of the RTP protocols established above. Adherence to this measure should mitigate the risk of dangerous neurogenic dysfunction resulting of repeated blows to the head. Should the athlete suffer a second head injury before the symptoms associated with the “energy crisis” from first impact have resolved, a synergistic and potentially fatal neurobiological cascade of events may trigger, known as Second Impact Syndrome (SIS). While the second impact to the brain may be sub-concussive in nature, the athlete can suffer an acute episode of severe cerebrovascular engorgement, diffuse cerebral swelling and brain herniation, resulting in coma and even death (Cantu & Gean, 2010).

The literature on the evidence for SIS (McCrorry, 2001) is mixed. Presumably, SIS results from the brain’s inability to regulate cerebral blood flow coupled with catecholamine release which abruptly increases intracranial blood volume (Lam, Hsiang & Poon, 1997). Young athletes appear to be at increased risk to experience SIS, with all reported cases occurring in athletes from the age of 10 to 24. The majority of deaths occurred in the high school athletic population ( $\mu=17.9$  years of age) and those participating in high-collision sports appear to be uniquely

susceptible, as most SIS instances resulted from American football and boxing injuries (71% and 14% respectively) (Mori, Katayama & Kawamata, 2006).

High school athletes in particular may be particularly susceptible to the dangers of SIS. Several unique risk factors exist which create increased vulnerability for high school-age athletes, including age, type of sport, and prior history of concussion. Proctor and Cantu (2000) discovered that beginning at approximately age 12, head injuries increase as a function of increasing age. Additionally, high school athletes evidence slower recovery of neurocognitive functioning domains when compared to college athletes (Collins, Stump & Lovell, 2004). Engagement in high-collision sport-related activities may be especially risky, as Cantu (2003) notes, 69% of all football-related fatalities from 1945 to 1999 were due to brain injury. Prior concussion history was also found in many of the athletes in this study. A history of concussive events may expose the athlete to magnified risk of incurring additional and more symptomatic concussions (Cobb & Battin, 2004).

**Chronic Traumatic Encephalopathy (CTE).** Chronic traumatic encephalopathy (CTE) is a neurodegenerative condition that is characterized by confusion, slowing of speech, tremors, Parkinsonian symptoms and overall mental deterioration (Saffary & Chin, 2012). A paucity of research exists in the area of CTE research. Once referred to as “dementia pugilistica,” CTE was thought to be a disease state specific to boxers who had experienced repeated blows to the head. Recently becoming an attention-grabbing issue in sports, it has been confirmed not only former boxers but also retired football players from the National Football League (NFL) as well as professional soccer and rugby players (Omalu et al., 2005). While CTE can only be confirmed via autopsy, it is thought to be the result of repeated concussive or subconcussive injuries. Rising concern over the physiological and psychological effects of concussions and their long-term

sequelae has prompted the medical and sports community to more closely examine the literature on concussions.

While the commonly reported symptoms of concussions (e.g. headaches, dizziness, fatigue, difficulty concentrating, feeling slowed down, foggiess and memory dysfunction) are typically acute, those accounted for by the long-term effects of CTE are insidious. Initially, individuals with CTE have been reported to experience deficits in attention, memory and concentration and may become confused or disoriented at times. As the disease state progresses, symptoms include poor judgment, changes in personality, dementia, lack of insight, depression, irrational behavior and Parkinsonianism (Omalu et al., 2005).

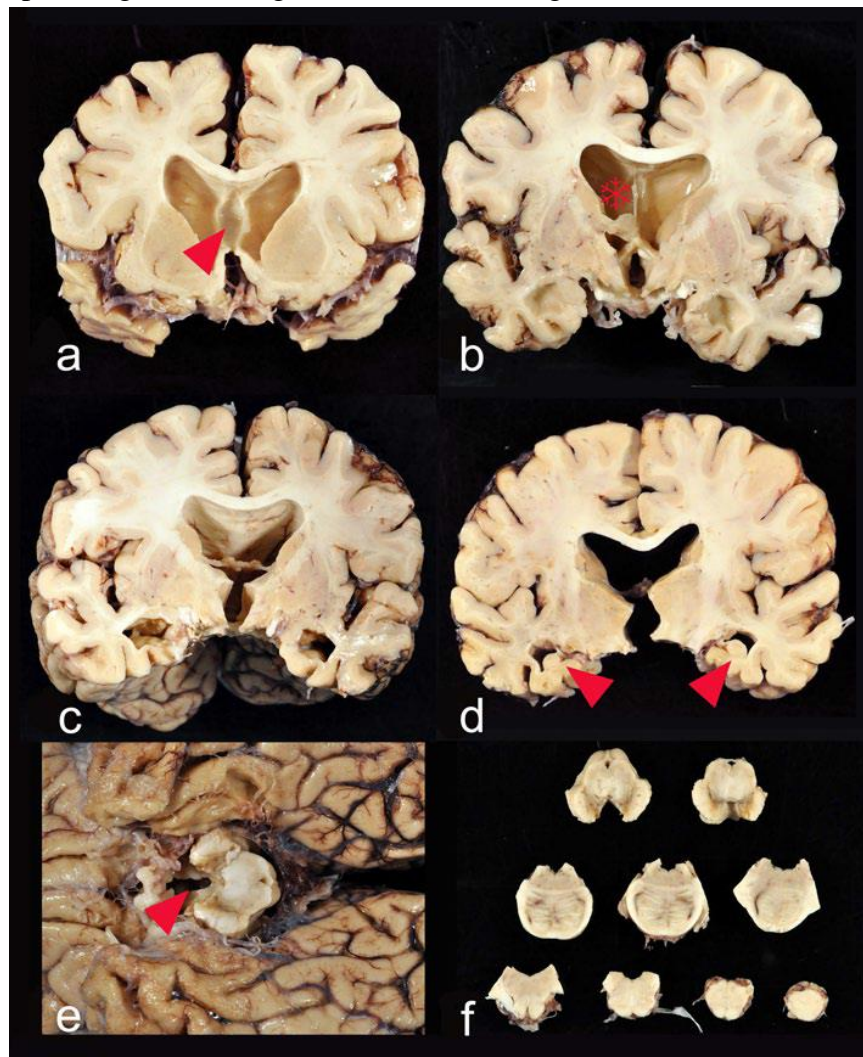
Evidence for the existence of biomarkers for CTE remains limited in the emerging literature. However, in 46 of the 51 confirmed cases of CTE as of 2009, McKee et al. (2009) identified that changes in tau proteins and the creation of plaques which lead to neurofibrillary tangles were evident. An increase in tangles appears to interfere with neuronal communication, eventually weakening the connections to the point of ceased neurological interconnectedness. As the progression of the disease occurs, symptomology expressed by the individual becomes more chronic and severe.

Retrospectively, CTE is found to present in 4 progressively deteriorated stages (McKee et al., 2012). Stage 1 is marked by headache and loss of attention. It may also include short-term memory problems, depression, and aggressive tendencies. Executive function limitations and explosivity is exhibited by only a few individuals at this stage. Stage 2 individuals were more likely to have experienced headache, attention and concentration problems, mood swings, short-term memory loss, and impulsivity. Less commonly they may have also experienced suicidal thoughts and language problems. In Stage 3, the symptoms of the previous stages are

exacerbated, with the possible addition of visuospatial difficulties, more extensive cognitive and memory problems, and apathy. Lastly, Stage 4 is commonly associated with more significant cognition problems and memory loss including profound loss of attention and concentration, executive dysfunction, language difficulties, explosivity, aggressive tendencies, paranoia, depression, gait and visuospatial difficulties. Few individuals may also experience problems with physical movement including Parkinsonism (McKee et al., 2012).

In addition to neurofibrillary tangles, McKee et al. (2009) suggested that brains examined for CTE have produced similar findings to those patients inflicted with Alzheimer's disease. Structural changes due to the disease progression include overall brain atrophy leading to loss of brain volume, namely, cell loss in the hippocampus, substantia nigra, and cerebral cortex. In addition, cell loss in frontal, temporal, subcallosal and insular cortices affect the size of ventricles within the brain. CSF traveling through the ventricles may pool in these areas, placing the brain at greater risk for neuronal damage. Evidence in brain structure between Alzheimer's disease and CTE may be distinguished by the shortened tauopathy found in CTE cases in the form of neuropil threads (NT) (McKee et al, 2009). McKee et al. (2013) illustrates this point in the Figure 2, below.

Figure 2: Neuropathological Findings in a Brain with Stage IV CTE



*Figure 2.* Gross neuropathological findings in a 77-year-old former Australian Rules rugby player who died with severe dementia and Stage IV CTE. Cognitive problems, memory loss, attention difficulties, and executive dysfunction were first noted in his mid-50s, followed by depression and anxiety, worsening explosivity and impulsivity. By his mid-60s, he was physically and verbally abusive, paranoid, and severely demented. He began playing rugby at age 13, and played for 19 years in U21 and senior leagues. **a** At autopsy, the brain weighed 1,030 g and showed severe atrophy and ventricular enlargement with a prominent cavum septum pellucidum (*arrowhead*). **b–c** The mid-portion of the septum pellucidum (*asterisk*) is reduced to a thin filament with severe atrophy of the fornix, thalamus, hypothalamus, mammillary bodies, amygdala, anterior hippocampus, and entorhinal cortex. **d** There is bilateral hippocampal atrophy (*arrowheads*). **e** The floor of the hypothalamus is severely thinned and the mammillary bodies are severely atrophic (*arrowhead*). **f** Brainstem sections show pallor of the pars compacta of the substantia nigra and locus coeruleus, with discoloration of the frontal tracts of the cerebral peduncle (McKee et al., 2013).

High school athletes who participate in high-collision sports and activities are typically aware of the acute risks associated with the sport itself, however, little is known about long-term damage that may be occurring within the brains of these individuals. Current literature reveals some insight into the biomarkers associated with CTE, however, developmental changes that may occur in brain injured athletes remain relatively unknown. Neuropsychological factors associated with the current theory of CTE may distinguish those who experience repeated blows to the head from those who do not. Given the retrospective nature in diagnosing CTE and long-term cognitive dysfunction, future directions in research and the early detection and intervention of preventative measures from an early-age are indicated.

### **Future Research**

Research involving sport-related concussion, mTBI and subconcussive events, provides initial findings regarding the unique dysfunction in high-school age athletes recovering from injury. Concussions are demonstrated to be an injury not only structural, but associated with a cascade of metabolic dysfunction within the brain that causes cognitive and clinical symptoms. This effect may be more prominent in athletes who are less mature in biological brain development. Ongoing studies examining the prevention, diagnosis, evaluation, intervention and recovery involved in RTP protocol will be essential to providing appropriate care to high school-age athletes and may determine is athlete are at risk for the development of long-term difficulties associated with repeated blows to the head, namely PCS and CTE.

### **Purpose of the Study**

This study proposed to explore the relationships between neurocognitive functioning in high school athletes as a function of reported concussion history. The examination of these relationships were cross-sectional, based on archival data. The neurocognitive measures utilized

assess functioning in various cognitive areas including visual memory, verbal memory, visual-motor speed, reaction time and impulse control. The data examined were collected from 34 Broward County, Florida high schools as part of a Florida regulation for all high school athletes to be measured biannually on baseline neurocognitive functioning prior to beginning their sport. This mandate was instituted by the School Board of Broward County, Florida and the Broward County Athletic Association (BCAA) to help diagnose and effectively manage a concussive injury. This data has been collected longitudinally over a span of 3 years (2011-2014). The study attempted to contribute to the existing literature on the risks associated with multiple concussive brain injuries, as well as provide further exploration on neurocognitive functioning in adolescents. Specifically, this study aimed to answer the following research questions:

1. Does neurocognitive functioning tend to remain stable over-time in healthy adolescents? If so, what changes occur in healthy high school athletes?
  - a.  $H_0$ : High school athletes' neurocognitive functioning remains stable over-time.
  - b.  $H_1$ : High school athletes' neurocognitive functioning varies over-time.
2. At baseline, do high school athletes differ in neurocognitive functioning profiles as a function of reported concussion history?
  - a.  $H_0$ : At baseline, high school athletes reporting prior concussion(s) do not differ in neurocognitive functioning from those who report no prior concussions.
  - b.  $H_1$ : At baseline, high school athletes reporting prior concussion(s) differ in neurocognitive functioning from those who report no prior concussions.

3. Are there differences in neurocognitive functioning in high school athletes' over-time due to a history of reported concussions?
  - a.  $H_0$ : High school athletes reporting prior concussion(s) do not differ in changes in neurocognitive functioning from those who report no prior concussions over-time.
  - b.  $H_1$ : High school athletes reporting prior concussion(s) differ in changes in neurocognitive functioning from those who report no prior concussions over-time.
4. If a significant difference is found between change in neurocognitive functioning among those who reported concussion history and those that did not, is this difference moderated by any variables (e.g., demographic variables, injury variables)?
  - a.  $H_0$ : If a significant difference is found between changes in neurocognitive functioning among those who reported concussion history and those that did not, this relationship will not be modified by an additional variable(s).
  - b.  $H_1$ : If a significant difference is found between changes in neurocognitive functioning among those who reported concussion history and those that did not, this relationship is modified by an additional variable(s).
5. Furthermore, if neurocognitive function is found to be different between groups of athletes with varying concussion history, is this change explained, even partially, by a component specific to concussion?
  - a.  $H_0$ : If neurocognitive function is found to be different between groups of athletes with varying concussion history, this change is fully explained by reported concussion history.



- b.  $H_1$ : If neurocognitive function is found to be different between groups of athletes with varying concussion history, this change is explained, even partially, by a component specific to concussion (e.g., baseline PCSS score, time since last concussion).

## **Chapter III**

### **Methods**

#### **Participants**

The current study is based on archival de-identified data provided by the Nova Southeastern University Sports Medicine Clinic (NSU-SMC). The sample consisted of approximately 23,000 Broward County, Florida, high school athletes who have completed at least one baseline neurocognitive functioning screening prior to beginning their sport in a given year. The subject ages ranged from 12-19 years of age. All subjects submitted to a baseline computerized neurocognitive functioning assessment as part of a county school board resolution requiring all high school athletes to complete baseline neurocognitive functioning measures to inform RTP protocol, should they be suspected of suffering a concussive brain injury. As this mandate encourages biannual testing, longitudinal data for several athletes in the archival database were provided.

Administration of the computerized assessment was delivered by licensed medical professionals trained to provide this form of testing. Training included instructions regarding login requirements, software requirements, test set-up, group and individual guidelines for administration. Typically, tests are expected to be administered in a controlled environment. In a group setting, an external mouse is required, along with empty terminals between examinees, 10-15 athletes per administration and in a quiet room. Computerized testing access is provided to the BCAA through the NSU-SMC, thus allowing access to the data collected.

## **Materials and Procedure**

### ***Neurocognitive testing.***

Computerized neurocognitive assessments are administered to each athlete biannually by licensed medical staff including the high school Certified Athletic Trainer (ATC) or the licensed psychologist at the NSU-SMC. Athletes received an explanation of RTP protocol and the importance of giving their full effort on the baseline neurocognitive function assessment. Additionally, administrators provided instructions regarding the assessment including what the test seeks to measure, length of time expected and format of the assessment. Players were expected to complete the computerized assessment in a quiet room individually or in group format. Once completed, athletes were given the option to send confirmation to a pre-registered email address or are given permission to leave.

### ***Neurocognitive evaluations.***

The Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT®) computerized test is the most widely used and most empirically validated computerized concussion evaluation system (Iverson, Lovell, & Collins, 2006). It was developed to assist practitioners in making informed decisions regarding RTP following concussions by comparing and athlete's post-concussion neuropsychological performance with baseline results. The test is divided into 3 sections which provide information to aid in the neurocognitive evaluation of concussion and are described below.

***Sections.*** The ImPACT® is comprised of 3 sections. Section 1 (Demographics Information and Health History Questionnaire) requires the athlete to input basic demographic and descriptive information using a computer keyboard and mouse to navigate/select responses on the screen. Additionally, this section asks athletes the answer questions regarding height,

weight, sport, position, concussion history and other historical health questions. Section 2 (Current Concussion Symptoms and Conditions) asks questions about the most recent date, hours slept last night and current medications. The athlete then rates their current severity of 22 concussion symptoms using the Post-Concussion Symptom Scale (PCSS) which relies on a 7-point Likert scale (a score of 0 representing no severity and a score of 6 representing the most severe rating). The PCSS calculates a total symptom score for each individual and is further explained below. In Section 3, athletes complete six modules that test neuropsychological functioning. These modules (Word Discrimination, Design Memory, X's and O's, Symbol Matching, Color Match and Three Letter Memory) are combined into 5 neuropsychological functioning scores that are also described below (ImPACT®, 2014).

***Post-Concussion Symptom Scale (PCSS).*** As outlined by the ICCS, RTP protocol suggests that athletes not return to play until they are asymptomatic (McCrory et al., 2013). Thus, the PCSS is utilized in the management of concussion by assessing and documenting clinical symptomology in athletes. The PCSS is presented to the athlete in Section 2 of the ImPACT® and identifies 22 commonly reported concussion symptoms. Respondents are required to endorse each of the 22 symptoms on a 7-point, Likert-type scale ranging in severity from “None” to “Severe” experience of the symptom within 24 hours of testing. A total symptom score is derived by adding the sum of all points endorsed for the 22 items (see Appendix for PCSS table).

***Modules.*** Each ImPACT® module is displayed in order to the athlete with the ability to pause between modules. In Module 1 (Word Discrimination), athletes are presented with twelve target words for 750 milliseconds. They are then given a second presentation of the same word set. After the second presentation, the athlete is presented with a randomized list of 12 target and

12 non-target words that have been chosen from the same semantic category. Athletes are asked to discriminate whether the word was a target word using the mouse to click “yes” or “no” responses on the computer screen. A delay condition (approximately 15 minutes) is administered after all other test modules using the same method (ImPACT®, 2014).

Module 2 (Design Memory) is similar to the method of Module 1, however, it uses target designs rather than target words. Additionally, the non-target designs are comprised of target designs that have been rotated. A delay condition (approximately 15 minutes) is assessed for this module as well. Individual and total percent scores are provided for correct “yes” and “no” responses for each module (ImPACT®, 2014).

In Module 3 (X’s and O’s) the athlete practices a distracter task (clicking the “Q” key if a blue square appears on the screen or the “P” key if a red circle is presented) and is then presented with a memory task. During the memory task, the athlete is presented with a randomized assortment of X’s and O’s for 1.5 seconds and asked to remember which three X’s or O’s were illuminated in yellow in their respective locations on the screen. After each presentation, the athlete completes the distracter task. The memory task then reappears and the athlete is asked to click the X’s or O’s that were previously illuminated in their location on the screen. Each athlete undergoes four trials of Module 3 and three scores are provided: correct identification, reaction time during the distracter task, and number of errors during the distracter task (ImPACT®, 2014).

In Module 4 (Symbol Matching) the athlete is initially presented with a 2x9 grid with 9 common symbols (i.e., square, circle, triangle, etc.) that are paired with a number from 1 to 9 underneath. Below this grid, a symbol is presented and the athlete is asked to click the corresponding number on the grid as quickly and accurately as possible. Correct performances

result in a green illumination of the number and incorrect performances result in a red illumination. After the athlete completes 27 trials, the symbols disappear from the top grid and the athlete is asked to recall the correct pairing by clicking the corresponding number in the grid to the symbol that appears below. Average reaction time scores are provided along with correct memory recognition scores (ImPACT®, 2014).

Module 5 (Color Match) is a Stroop-type challenge that begins with a task to ensure the athlete can discriminate color. The athlete is required to click red, blue or green buttons on the screen as a demonstration of visual acuity. Next, color words (i.e., RED, BLUE, GREEN) are displayed in the same color ink (i.e., the word RED displayed in red ink) or in a different color ink (i.e., the word GREEN displayed in red ink). The athlete is asked to click the box only if the word is displayed in the same ink as quickly as possible. Scores are provided for reaction time and errors (ImPACT®, 2014).

Lastly, the athlete is presented with Module 6, known as Three Letter Memory, where three randomized consonant letters are displayed on the screen and are immediately followed by randomized distractor task. In the 18-second distractor task, the athlete uses the computer mouse to click in backward order, as quickly as possible, on a 5x5 grid that contains randomized numbers from 1 to 25. The athlete is then presented with a memory task, where they are asked to recall the three consonants by typing them on the keyboard. After five trials, scores are provided for correctly identified letters and the average number of correctly clicked numbers during the distractor task (ImPACT®, 2014).

***Composite Subscale Scores.*** The results of the 6 neurocognitive modules on the ImPACT® test are combined and five composite subscale scores are calculated: Verbal Memory, Visual Memory, Processing Speed, Reaction Time and Impulse Control.

Verbal Memory Composite is a score ranging from 1 to 100, with higher scores indicating better performance. It is comprised of the average of the following scores: total memory percent correct, total correct hidden symbols matched from the Symbol Matching module (Module 4) and total percent letters correct from the Three Letter Memory module (Module 6).

Visual Memory Composite is a score ranging from 1 to 100, with higher scores indicating better performance. It is comprised of an average of the following scores: total percent correct from the Design Memory module (Module 2) and total correct memory score from the X's and O's module (Module 3).

Processing Speed Composite is a score ranging from 1 to 100, with higher scores indicating better performance. It is comprised of an average of the following scores: total number correct on the distractor task divided by 4 (number correct/4) from the X's and O's module (Module 3) and average numbers counted correctly times 3 (number correct x 3) from the Three Letter Memory module (Module 6).

Reaction Time Composite is a score ranging from 0 to 1, with lower scores indicating better performance. The value is measured in seconds. It is comprised of an average of the following scores: average correct reaction time (RT) from the X's and O's module (Module 3), average correct RT divided by 3 (avg. correct/3) from the Symbol Matching module (Module 4) and average correct RT from the Color Match module (Module 5).

Impulse Control Composite is a total score ranging from 0 to 132 with lower scores indicating better performance. Each point represents an individual error. It is comprised of the following scores: total incorrect (interference) during the distractor task from the X's and O's

module (Module 3) and total commissions from the Color Match module (Module 5) (ImPACT®, 2014).

Once the composite scores are calculated, a percentile rank is produced for all composite scales except the Impulse Control composite to inform the clinician's interpretation of the athlete's scores compared within the normative sample. This is valuable during both baseline and post-injury testing to establish an expected range of neurocognitive functioning norms for each athlete. If an athlete is suspected of sustaining a concussion, composite scores or percentile ranks may be used to compare post-concussion tests to that athlete's baseline functioning norms. If a current baseline test is not available, scores may be compared to age-appropriate norms. The athlete may be cleared to begin RTP protocol once baseline or normative scores are met (McCrary, et al., 2013).

Iverson, Lovell, & Collins (2003) examined the psychometric properties of the ImPACT® test. Primarily, the study found no test-retest practice effects after a two-week interval. This is necessary to validate when using a concussed sample, but is also important when discussing baseline year-to-year results. Additionally, there were no significant differences in test-retest for any of the composite scores (Verbal Memory:  $t(55) = -0.17, p < .87$ , Visual Memory:  $t(55) = 0.85, p < .40$ , Reaction Time:  $t(55) = 0.97, p < .34$  and Total Symptom Score:  $t(55) = -0.54, p < .60$ ) except for Processing Speed ( $t(55) = -3.26, p < .003$ ) in which 68% did it faster at re-test. More contemporary findings (Nakayama, et al., 2014) revealed stronger evidence to suggest the ImPACT is a reliable neurocognitive battery. Intraclass correlation coefficients (ICCs) were calculated for baseline to day 45, day 45 to day 50, baseline to day 50, and overall. Results indicated all ICCs exceeded the threshold value of 0.60 (Verbal Memory: 0.76, 0.69, 0.65, and 0.78; Visual Memory: 0.72, 0.66, 0.60, and 0.74; Visual Motor Speed: 0.87,



0.88, 0.85, and 0.91; Reaction Time: 0.67, 0.81, 0.71, and 0.80). At baseline testing, sections inquiring about current concussion are omitted, although concussion history is requested.

### **Proposed Analyses**

The analyses below were performed using the IBM SPSS 23. A description of the proposed analyses is provided next.

#### ***Effect Size Considerations.***

Initially outlined in 2009 and updated in 2016, Ferguson (2016) provides detailed guidelines on the selection and interpretation of reported effect sizes. Four general categories of effect size are provided, including (1) Group difference indices, (2) Strength of association indices, (3) Corrected estimates, and (4) Risk estimates. Group difference indices usually note the magnitude of difference between two or more groups. Strength of association indices typically examine the magnitude of shared variance between two or more variables. Corrected estimates, or squared associations, including partial eta-squared ( $\eta^2$ ) is most commonly used for factorial ANOVA designs. Risk estimates are generally used in medical research and estimate the difference in risk for a particular outcome between two or more groups of individuals.

To determine selection of an appropriate effect size, Ferguson (2016) provides general guidelines: (1) Rigid adherence to arbitrary guidelines is not recommended. That is, study limitations must be considered when interpreting effect size, as the guidelines suggested are minimal cutoffs and not guarantees that the observed effect sizes are meaningful, (2) Corrected effect sizes are preferable to uncorrected effect sizes, (3) For correlational designs, partial  $r$  and standardized regression coefficients are preferred to bivariate  $r$  as they estimate the unique variance attributable to a predictor controlling for other variables, (4) When the data are binomial, only  $r_h$  should be used as an effect size estimate, (5) For ordinal data, Somer's  $d$  or

Kendall's  $\tau$  should be used, (6) It is recommended that effect size  $C_i$  be reported along with effect size estimates, and (7) Effect size estimates can be influenced by sampling and measurement errors which may reduce the representation of the "true" effect in the population.

In an effort to adhere to these guidelines and for the purpose of the proposed analyses in this study (described below), the partial eta-squared ( $\eta^2$ ) index was employed to interpret the magnitude of effect observed, with  $r^2$  utilized for post-hoc testing. The following threshold interpretation of these effects are utilized: .04 (RMPE = recommended minimum effect size), .25 (moderate effect), and .64 (strong effect).

### ***Hypothesis I.***

Utilizing a longitudinal design, we attempted to determine what, if any, changes in neurocognitive functioning can be expected over time in high school athletes. Athletes who report no prior concussions to baseline and have at least two valid baselines were included in these analyses. A paired, or "repeated measures", t-test was used to evaluate if there is a change in neurocognitive functioning over-time in these "healthy" athletes. Utilizing a paired t-test approach allows for comparison of the same group of individuals at multiple time points, reducing variation and increasing statistical power.

### ***Hypothesis II.***

In order to choose an appropriate analysis to perform in *Hypothesis III*, we must compare the neurocognitive functioning in high school athletes who report varying concussion histories at baseline. A one-factor between-subjects ANOVA was performed with reported concussion history (e.g., Group 1[0 concussions]; Group 2[1 concussion]; Group 3[2+ concussions]) entered as the IV and each of the four neurocognitive functioning composite scores (visual memory, verbal memory, visual-motor speed and reaction time) were treated as DVs.

Assumptions associated with ANOVA include (1) independence of observations, (2) normality, and (3) homogeneity of variances. Independence of observations denotes that the occurrence of one event does not affect the probability of the other event, thus independently observed. The assumption of normality states that the deviations of an observed value in a sample are normally distributed within the population from which the sample was chosen. The tenability of these assumptions were evaluated before conducted the ANOVA's a priori (e.g., testing equality of variances using Levene's test). Level of significance was set to  $\alpha = .05$  to yield a 95% confidence interval for interpretation of results. If the omnibus, or overall, was found to be significant, the Tukey (honest significant difference) *HSD* post hoc procedure was conducted using a family-wise error rate of  $\alpha_{FWE} = .05$  to further explore the relationships between groups. Tukey's *HSD* test compares the means of each level of the group to the means of every other level and is applied simultaneously to the set of all pairwise comparisons.

### ***Hypothesis III.***

Once the "healthy" trend of neurocognitive change over time was established (*Hypothesis II*), neurocognitive functioning over-time as a function of prior reported concussion history was evaluated utilizing Analysis of Covariance (ANCOVA). Athletes included in this statistical analysis must have had at least 2 baseline tests of neurocognitive functioning.

Additional considerations in ANCOVA include (1) independence of the covariate and treatment effect, and (2) homogeneity of regression slopes. The independence of the covariate and treatment effect refers to the assumption that the IV and covariate are not significantly associated to each other. Homogeneity of regression slopes refers to the assumption that the relationship (i.e., positive, negative and with relative strength) between the covariate and the DV is homogenous across the groups being tested (IV). If the analysis conducted in *Hypothesis III*

revealed differences in baseline neurocognitive functioning between athletes with varying concussion histories, alternative analyses were considered (e.g., 3x2 Mixed Model ANOVA).

If the assumptions are met, the proposed statistical procedure utilized groups of athletes separated based upon reported prior concussion history (G1: 0 concussions; G2: 1 concussion; G3: 2+ concussions) as the levels of IV. The covariate in the ANCOVA model was the baseline neurocognitive functioning composite scores. The DV was the second baseline of the high school athletes included in the procedure.

***Hypothesis IV.***

If a statistical difference was found when comparing groups of high school athletes based on reported prior concussion history, we would explore the possibility of alternative components which may moderate the strength of the relationship between concussion history and neurocognitive functioning. Exploration of moderation tests the extent to which the prediction of a DV from an IV differs across levels of a third variable. This third variable affects the strength and/or direction of the relationship between the IV and DV, depending on the levels of the IV and third variable. The model below offers a path diagram for mediation where the IV, DV and moderator variable are represented by X, Y and Z, respectively.

Figure 3: Path Model of Moderation

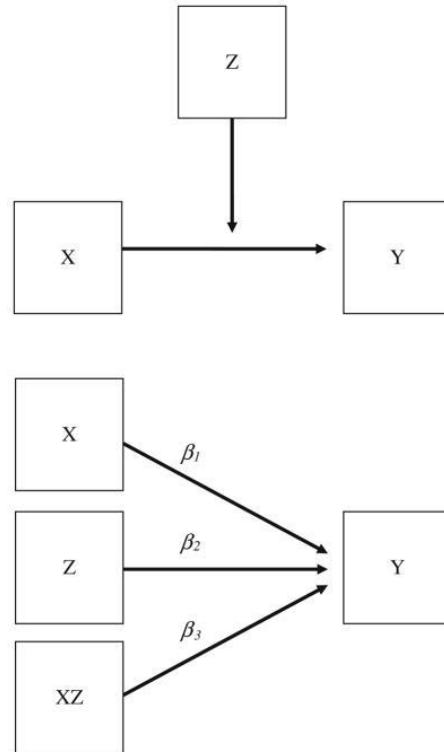


Figure 3. Note:  $XZ$  = the product of  $X$  and the moderator variable,  $\beta_1$  = the effect of  $X$  on  $Y$ ,  $\beta_2$  = the effect of  $Z$  on  $Y$ , and  $\beta_3$  = the effect of  $XZ$  on  $Y$  (Fairchild and MacKinnon, 2009).

### ***Hypothesis V.***

If a significant statistical difference was found when comparing groups of high school athletes based on reported prior concussion history, we would explore the possibility of alternative components which may mediate the relationship between concussion history and neurocognitive functioning. In mediation, the relationship between the IV and the DV is hypothesized to be an indirect effect that exists due to the influence of a third variable, the mediator. When the mediator variable is included in a regression analysis model with the IV, the effect of the IV is reduced and the effect of the mediator remains significant. Sobel's test of Significance of the Indirect Effect ( $ab$ ) is used in simple mediation models to test the significance of a mediation effect. Thus, it provides a method to determine whether the reduction

in effect of the IV, after including the mediator variable, is a significant reduction and therefore whether the indirect effect is statistically significant.

A Path model of indirect effects was utilized to conceptually understand the influence of a mediating variable. Here, the relationship between the IV and mediator (Path *a*) is established if variations in the level of the IV significantly account for variations in the mediator. This relationship is explored by performing an ANOVA. Next, the relationship between the mediator and DV (Path *b*) is established if variations in the level of the mediator significantly account for variations in the DV. Lastly, mediation is confirmed if the relationship between the IV and DV (Path *c*) are no longer significant when Paths *a* and *b* are controlled. Partial mediation is confirmed if Path *c* does not result in a value of zero.

Figure 4: Path Model of Indirect Effects

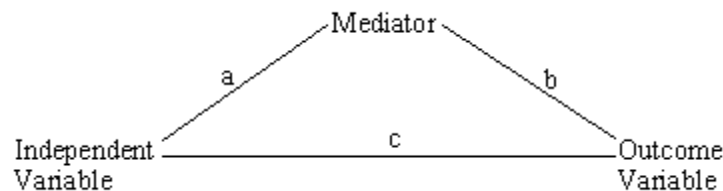


Figure 4. The path model of indirect effects (Preacher & Hayes, 2008).

## **Chapter IV**

### **Results**

#### **Statistical Plan**

Results provided below were analyzed using IBM SPSS 23. Several considerations were made during the statistical implementation of the proposed analyses to ensure the data was utilized in an efficient manner, consistent with the proposed statistical plan. Considerations and findings are provided below.

#### **Selection Criteria**

Participants were all initially selected for entry into the current study if they were Broward County, Florida, high school athletes who had completed at least one baseline neurocognitive functioning screening prior to beginning their sport in a given year from 2009 – 2014. This initial sample consisted of 23,376 unique individuals who completed 1, 2, 3, or 4 baseline screenings during this timeframe. Based on the current best research practice in the literature (Brooks et al., 2013; Covassin et al., 2011; Kontos et al., 2011) and consultation with committee members, including an expert sport psychologist, the decision was made to eliminate neurocognitive profiles to reduce confounding variables and ensure the results were reflective of best research practices. Additionally, given the target sample (e.g., high school athletes), selection criteria were evaluated based on factors deemed directly influential to adolescent development. Selection criteria eliminated profiles of athletes who: (1) were younger than age 12 or older than age 19; (2) had a self-reported history of attention deficit hyperactivity disorder (ADHD); and (3) self-reported history of a learning disorder (LD). Results are illustrated in the figure below.

Figure 5: Selection Criteria Flow Chart

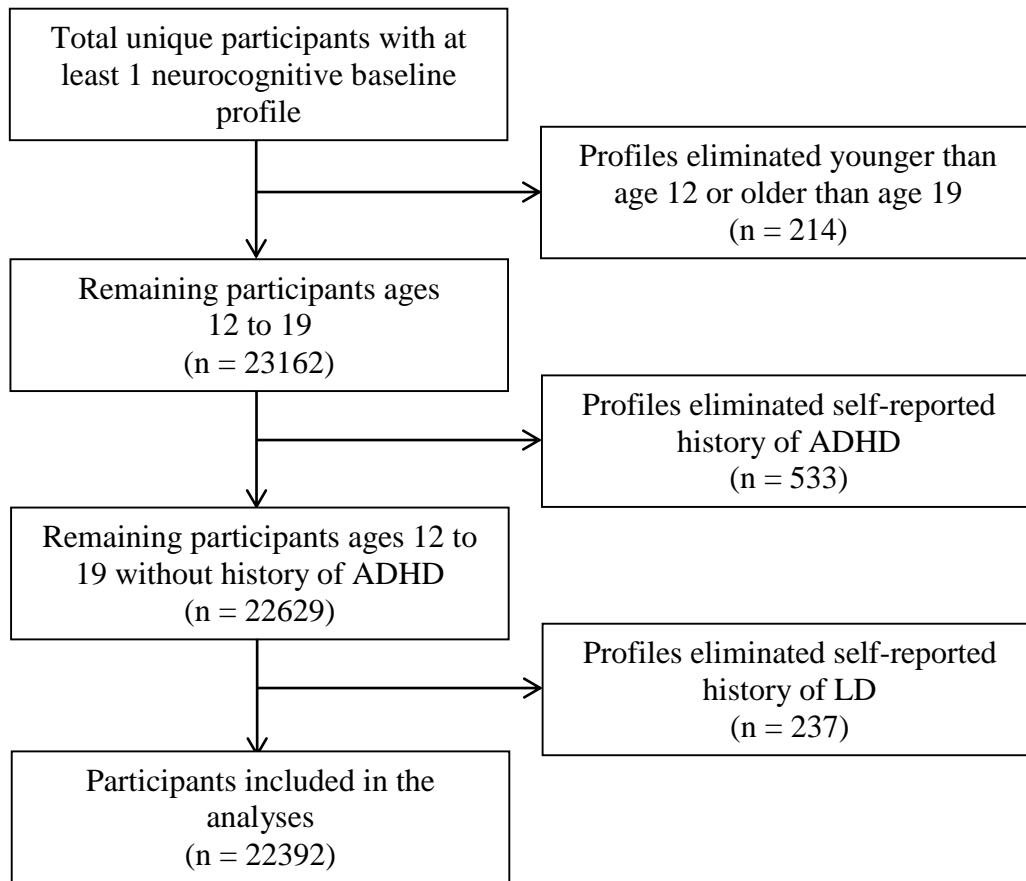


Figure 5. Selection criteria flow chart.

### Demographic Information

Of the remaining sample (n = 22392) several groups with multiple levels were identified. To ensure clarity for the reader, demographic frequencies at initial baseline pertinent to the current study are illustrated in the tables below.



Table 1

*Frequency of Age in Sample*

Age	<i>n</i>	%
12	113	0.50
13	405	1.81
14	5521	24.66
15	5918	26.43
16	5142	22.96
17	4189	18.71
18	1015	4.53
19	89	0.40

Table 2

*Frequency of Concussions in Sample*

# of Concussions	<i>n</i>	%
0	21123	94.33
1	1002	4.47
2	189	.85
3	47	.21
4	11	.05
5	14	.06
6	1	.005
7	1	.005
9	4	.02

Table 3

*Demographics and Other Characteristics of Sample*

Characteristic	<i>n</i>	%
Gender		
Male	13820	61.72
Female	8572	38.28
Ethnicity		
White	8177	36.52
Black or African American	7889	35.23
Hispanic or Latino	3800	16.97
Asian	260	1.16
American Indian or Alaska Native	93	0.42
Native Hawaiian or Other Pacific Islander	71	.32
Missing Data	2102	9.38
Country of origin		
United States	20773	92.77
Other	1619	7.23
Language of origin (n=25957)		
English	20823	93.00
Spanish	1053	4.70
Other	516	2.30

**Analyses Employed***Hypothesis I.*

It was hypothesized that athletes who did not endorse a history of concussion would not demonstrate significant changes in neurocognitive functioning when tested at separate time points. To test this hypothesis, four composite scores related to neurocognitive functioning along with impulse control and total symptom score as produced by the ImPACT® test (e.g., Verbal Memory [VeM], Visual Memory [ViM], Visual Motor Speed [VMS], Reaction Time [RT], Impulse Control [IC], and Post-Concussion Symptom Scale [PCSS]) were measured at two time points: initial baseline and follow-up testing. Six Repeated Measures ANOVA's were employed

to determine if neurocognitive changes occur from initial baseline profile to follow-up baseline profile in non-concussed high school athletes.

Table 4

*Descriptive and Repeated Measures ANOVA Statistics in Non-Concussed Sample (n = 2141)*

Variable	Initial Mean (SD)	Follow-Up Mean (SD)	F	Partial Eta <sup>2</sup>
Verbal Memory	79.40 (12.46)	82.46 (11.62)	116.77*	.052
Visual Memory	67.81 (14.78)	72.17 (14.22)	209.30*	.089
Visual Motor Speed	32.96 (7.36)	35.39 (7.32)	394.33*	.156
Reaction Time	0.65 (0.13)	0.64 (0.11)	22.56*	.010
Impulse Control	8.17 (9.80)	6.66 (6.28)	53.85*	.025
Post-Concussion Symptom Scale	4.23 (7.55)	3.64 (7.56)	12.61*	.006

\*Significant at the  $< .01$  level

**Verbal Memory (VeM).** Descriptive statistics for the non-concussed group at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2141$ ,  $\mu = 79.40$ ,  $\sigma = 12.46$ ; Follow-Up:  $n = 2141$ ,  $\mu = 82.46$ ,  $\sigma = 11.62$ ). Repeated Measures ANOVA revealed a significant difference within the non-concussed group over-time ( $F = 116.768$ ,  $p < .001$ , partial  $\eta^2 = .052$ ) with a small effect.

**Visual Memory (ViM).** Descriptive statistics for the non-concussed group at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2141$ ,  $\mu = 67.81$ ,  $\sigma = 14.78$ ; Follow-Up:  $n = 2141$ ,  $\mu = 72.17$ ,  $\sigma = 14.22$ ). Repeated Measures ANOVA revealed a significant difference within the non-concussed group over-time ( $F = 209.298$ ,  $p < .001$ , partial  $\eta^2 = .089$ ) with a small effect.

**Visual Motor Speed (VMS).** Descriptive statistics for the non-concussed group at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2141$ ,  $\mu = 32.96$ ,  $\sigma = 7.36$ ; Follow-Up:  $n = 2141$ ,  $\mu = 35.39$ ,  $\sigma = 7.32$ ). Repeated Measures ANOVA revealed a

significant difference within the non-concussed group over-time ( $F = 394.334, p < .001$ , partial  $\eta^2 = .156$ ) with a small effect.

**Reaction Time (RT).** Descriptive statistics for the non-concussed group at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2141, \mu = .651, \sigma = .129$ ; Follow-Up:  $n = 2141, \mu = .637, \sigma = .114$ ). Repeated Measures ANOVA revealed a significant difference within the non-concussed group over-time ( $F = 22.561, p < .001$ , partial  $\eta^2 = .010$ ) with a small effect.

**Impulse Control (IC).** Descriptive statistics for the non-concussed group at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2141, \mu = 8.17, \sigma = 9.80$ ; Follow-Up:  $n = 2141, \mu = 6.66, \sigma = 6.28$ ). Repeated Measures ANOVA revealed a significant difference within the non-concussed group over-time ( $F = 53.852, p < .001$ , partial  $\eta^2 = .025$ ) with a small effect.

**Post-Concussion Symptom Scale (PCSS).** Descriptive statistics for the non-concussed group at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2141, \mu = 4.23, \sigma = 7.55$ ; Follow-Up:  $n = 2141, \mu = 3.64, \sigma = 7.56$ ). Repeated Measures ANOVA revealed a significant difference within the non-concussed group over-time ( $F = 12.606, p < .001$ , partial  $\eta^2 = .006$ ) with a small effect.

### **Hypothesis II.**

It was hypothesized that at initial baseline testing, athletes would demonstrate significantly different neurocognitive performances based on reported concussion history. To test this hypothesis, 6 separate one-factor between-subjects ANOVA's were performed with reported concussion history (e.g., Group 1[0 concussions]; Group 2[1 concussion]; Group 3[2+ concussions]) at initial baseline entered as the independent variables and each of the four

neurocognitive functioning composite scores along with impulse control and total symptom score as produced by the ImPACT® test (e.g., Verbal Memory [VeM], Visual Memory [ViM], Visual Motor Speed [VMS], Reaction Time [RT], Impulse Control [IC], and Post-Concussion Symptom Scale [PCSS]) were treated as dependent variables.

Table 5

*Descriptive and Between-Subjects ANOVA Statistics Amongst Concussion Groups*

Variable	0 Con. M (SD) <sup>a</sup>	1 Con. M (SD) <sup>b</sup>	2+ Con. M (SD) <sup>c</sup>	F	Partial Eta <sup>2</sup>
Verbal Memory	81.14 (11.19)	79.78 (11.96)	78.99 (12.40)	11.54*	.001
Visual Memory	69.41 (13.86)	68.40 (14.46)	67.00 (16.61)	6.35*	.001
Visual Motor Speed	33.47 (7.12)	33.61 (7.88)	32.94 (9.16)	.921	< .001
Reaction Time	0.65 (0.12)	0.64 (0.11)	0.67 (0.21)	8.50*	.001
Impulse Control	6.67 (6.58)	7.87 (8.24)	7.41 (7.22)	16.77*	.001
Post-Concussion Symptom Scale	4.32 (7.85)	6.36 (9.71)	8.23 (12.77)	60.94*	.005

<sup>a</sup> Denotes  $n = 21123$ ; <sup>b</sup> Denotes  $n = 1002$ ; <sup>c</sup> Denotes  $n = 267$

\*Significant at the  $< .01$  level

**Verbal Memory (VeM).** Descriptive statistics for the 3 groups are as follows: 0

Concussions ( $n = 21123$ ,  $\mu = 81.13$ ,  $\sigma = 11.19$ ), 1 Concussion ( $n = 1002$ ,  $\mu = 79.78$ ,  $\sigma = 11.96$ ), 2+ Concussions ( $n = 267$ ,  $\mu = 78.99$ ,  $\sigma = 12.40$ ). Between-subjects ANOVA revealed significant differences between groups ( $F = 11.537$ ,  $p < .001$ , partial  $\eta^2 = .001$ ) with a small effect, however, a violation of Levene's Test of Equality of Variances ( $F(2, 22389) = 3.944$ ,  $p = .019$ ) is also found. Although Levene's Test of Homogeneity of Variance is significant, and thus violated, the standard deviation between-groups are extremely similar and likely due to large sample size. Subsequent Welch's test was performed to account for unequal variances (Welch's  $F(2, 578.722) = 9.976$ ,  $p < .001$ ) and also revealed significant between-group differences. Post-hoc Independent T-tests using Welch's test account for the probability of inflation of Type I error. Bonferroni's correction is also applied, comparing each p-value to  $.05/3$  for interpretation. Post-

hoc Independent Samples T-Test revealed significant between group differences for 0 Concussions vs. 1 Concussion ( $t(1085.867) = 3.522, p < .001, r^2 = .011$ ), 0 Concussions vs. 2+ Concussions ( $t(271.507) = 2.822, p = .005, r^2 = .028$ ), however, the 1 Concussion vs. 2+ Concussions comparison was not significant ( $t(407.484) = .937, p = .349, r^2 = .002$ ).

**Visual Memory (ViM).** Descriptive statistics for the 3 groups are as follows: 0 Concussions ( $n = 21123, \mu = 69.41, \sigma = 13.86$ ), 1 Concussion ( $n = 1002, \mu = 68.40, \sigma = 14.46$ ), 2+ Concussions ( $n = 267, \mu = 67.00, \sigma = 16.61$ ). Between-subjects ANOVA revealed significant differences between groups ( $F = 6.349, p = .002, partial \eta^2 = .001$ ) with a small effect, however, a violation of Levene's Test of Equality of Variances ( $F(2, 22389) = 12.070, p < .001$ ) is also found. Although Levene's Test of Homogeneity of Variance is significant, and thus violated, the standard deviation between-groups are extremely similar and likely due to large sample size. Subsequent Welch's test was performed to account for unequal variances (Welch's  $F(2, 577.842) = 5.051, p = .007$ ) and also revealed significant between-group differences. Post-hoc Independent T-tests using Welch's test account for the probability of inflation of Type I error. Bonferroni's correction is also applied, comparing each p-value to  $.05/3$  for interpretation. Post-hoc Independent Samples T-Test revealed significant between group differences for 0 Concussions vs. 1 Concussion ( $t(1090.028) = 2.171, p = .03, r^2 = .004$ ), 0 Concussions vs. 2+ Concussions ( $t(270.704) = 2.365, p = .019, r^2 = .020$ ), however, the 1 Concussion vs. 2+ Concussions comparison was not significant ( $t(380.233) = 1.257, p = .209, r^2 = .004$ ).

**Visual Motor Speed (VMS).** Descriptive statistics for the 3 groups are as follows: 0 Concussions ( $n = 21123, \mu = 33.47, \sigma = 7.12$ ), 1 Concussion ( $n = 1002, \mu = 33.61, \sigma = 7.88$ ), 2+ Concussions ( $n = 267, \mu = 32.94, \sigma = 9.16$ ). Between-subjects ANOVA revealed no significant differences between groups ( $F = .921, p = .398, partial \eta^2 < .001$ ) with no effect. In addition, a

violation of Levene's Test of Equality of Variances ( $F(2, 22389) = 16.455, p < .001$ ) is also found. Although Levene's Test of Homogeneity of Variance is significant, and thus violated, the standard deviation between-groups are extremely similar and likely due to large sample size. Subsequent Welch's test was performed to account for unequal variances (Welch's  $F(2,575.809) = .610, p = .544$ ) and remained non-significant. Post-hoc Independent T-tests using Welch's test are not indicated.

**Reaction Time (RT).** Descriptive statistics for the 3 groups are as follows: 0 Concussions ( $n = 21123, \mu = .648, \sigma = .115$ ), 1 Concussion ( $n = 1002, \mu = .640, \sigma = .107$ ), 2+ Concussions ( $n = 267, \mu = .673, \sigma = .206$ ). Between-subjects ANOVA revealed significant differences between groups ( $F = 8.504, p < .001, partial \eta^2 = .001$ ) with a small effect, however, a violation of Levene's Test of Equality of Variances ( $F(2, 22389) = 17.416, p < .001$ ) is also found. Although Levene's Test of Homogeneity of Variance is significant, and thus violated, the standard deviation between-groups are extremely similar and likely due to large sample size. Subsequent Welch's test was performed to account for unequal variances (Welch's  $F(2,576.195) = 4.408, p = .013$ ) and also revealed significant between-group differences. Post-hoc Independent T-tests using Welch's test account for the probability of inflation of Type I error. Bonferoni's correction is also applied, comparing each p-value to  $.05/3$  for interpretation. Post-hoc Independent Samples T-Test revealed significant between group differences for 0 Concussions vs. 1 Concussion ( $t(1115.458) = 2.130, p = .033, r^2 = .004$ ), 0 Concussions vs. 2+ Concussions ( $t(268.124) = -2.040, p = .042, r^2 = .015$ ), and the 1 Concussion vs. 2+ Concussions comparison ( $t(305.004) = -2.540, p = .012, r^2 = .021$ ).

**Impulse Control (IC).** Descriptive statistics for the 3 groups are as follows: 0 Concussions ( $n = 21123, \mu = 6.67, \sigma = 6.583$ ), 1 Concussion ( $n = 1002, \mu = 7.87, \sigma = 8.242$ ), 2+

Concussions ( $n = 267$ ,  $\mu = 7.41$ ,  $\sigma = 7.218$ ). Between-subjects ANOVA revealed significant differences between groups ( $F = 16.771$ ,  $p < .001$ ,  $partial \eta^2 = .001$ ) with a small effect, however, a violation of Levene's Test of Equality of Variances ( $F(2, 22389) = 14.208$ ,  $p < .001$ ) is also found. Although Levene's Test of Homogeneity of Variance is significant, and thus violated, the standard deviation between-groups are extremely similar and likely due to large sample size. Subsequent Welch's test was performed to account for unequal variances (Welch's  $F(2,576.496) = 11.499$ ,  $p < .001$ ) and also revealed significant between-group differences. Post-hoc Independent T-tests using Welch's test account for the probability of inflation of Type I error. Bonferoni's correction is also applied, comparing each p-value to  $.05/3$  for interpretation. Post-hoc Independent Samples T-Test revealed significant between group differences for 0 Concussions vs. 1 Concussion ( $t(1062.455) = -4.527$ ,  $p < .001$ ,  $r^2 = .019$ ), however, the 0 Concussions vs. 2+ Concussions was not significant ( $t(271.623) = -1.671$ ,  $p = .096$ ,  $r^2 = .010$ ), and the 1 Concussion vs. 2+ Concussions comparison was not significant ( $t(467.956) = .886$ ,  $p = .376$ ,  $r^2 = .002$ ).

***Post-Concussion Symptom Scale (PCSS)***. Descriptive statistics for the 3 groups are as follows: 0 Concussions ( $n = 21123$ ,  $\mu = 4.32$ ,  $\sigma = 7.848$ ), 1 Concussion ( $n = 1002$ ,  $\mu = 6.36$ ,  $\sigma = 9.712$ ), 2+ Concussions ( $n = 267$ ,  $\mu = 8.23$ ,  $\sigma = 12.768$ ). Between-subjects ANOVA revealed significant differences between groups ( $F = 60.942$ ,  $p < .001$ ,  $partial \eta^2 = .005$ ) with a small effect, however, a violation of Levene's Test of Equality of Variances ( $F(2, 22389) = 77.552$ ,  $p < .001$ ) is also found. Although Levene's Test of Homogeneity of Variance is significant, and thus violated, the standard deviation between-groups are extremely similar and likely due to large sample size. Subsequent Welch's test was performed to account for unequal variances (Welch's  $F(2,571.631) = 33.522$ ,  $p < .001$ ) and also revealed significant between-group



differences. Post-hoc Independent T-tests using Welch's test account for the probability of inflation of Type I error. Bonferoni's correction is also applied, comparing each p-value to  $.05/3$  for interpretation. Post-hoc Independent Samples T-Test revealed significant between group differences for 0 Concussions vs. 1 Concussion ( $t(1063.922) = -6.558, p < .001, r^2 = .039$ ), 0 Concussions vs. 2+ Concussions ( $t(268.547) = -4.989, p < .001, r^2 = .085$ ), and the 1 Concussion vs. 2+ Concussions comparison ( $t(352.124) = -2.221, p = .027, r^2 = .014$ ).

### ***Hypothesis III.***

It was hypothesized that high school athletes reporting prior concussion(s) would differ in changes in neurocognitive functioning from those who report no prior concussions, over-time. Six 2x2 Mixed Model ANOVA's were employed rather than ANCOVA, given the differences in neurocognitive functioning discovered at initial baseline between concussion groups. Participants were divided into two groups; 0 Concussions and 1+ Concussions. Time was divided into two groups; Initial Baseline and Follow-Up Baseline. These operations were performed to capture the maximum amount of participants who completed more than one baseline test. Descriptive statistics revealed that, on average, all athletes in the sample tested at least twice were tested approximately 328 days between initial and follow-up ( $n = 2235, \mu = 328.04, \sigma = 219.07$ ). The interaction term (Time x Group) was analyzed for each of the four neurocognitive functioning composite scores along with impulse control and total symptom score as produced by the ImPACT® test (e.g., Verbal Memory [VeM], Visual Memory [ViM], Visual Motor Speed [VMS], Reaction Time [RT], Impulse Control [IC], and Post-Concussion Symptom Scale [PCSS]) were treated as dependent variables.

Table 6

*Descriptive and Mixed Model ANOVA Statistics Amongst Concussion Groups*

Variable	Time	0 Con. M (SD) <sup>a</sup>	1+ Con. M (SD) <sup>b</sup>	F-within (Partial Eta <sup>2</sup> )	F-between (Partial Eta <sup>2</sup> )
Verbal Memory	I	79.56 (12.38)	75.34 (13.56)	2.61 (.001)	14.72** (.007)
	FU	83.09 (11.26)	80.74 (11.53)		
Visual Memory	I	67.89 (14.76)	65.80 (15.13)	0.001 (< .001)	3.67 (.002)
	FU	72.66 (14.08)	70.53 (14.46)		
Visual Motor Speed	I	32.98 (7.32)	32.33 (7.76)	0.91 (< .001)	0.51 (< .001)
	FU	35.56 (7.25)	35.39 (7.22)		
Reaction Time	I	0.65 (0.13)	0.66 (0.12)	0.58 (< .001)	0.15 (< .001)
	FU	0.64 (0.11)	0.64 (0.11)		
Impulse Control	I	8.11 (9.79)	9.71 (10.76)	0.60 (< .001)	4.76* (.002)
	FU	6.53 (6.09)	7.47 (8.83)		
Post-Concussion Symptom Scale	I	4.21 (7.55)	5.93 (8.10)	0.64 (< .001)	7.71** (.003)
	FU	3.56 (7.48)	4.96 (8.40)		

Note. I = Initial Testing; FU = Follow-Up Testing

<sup>a</sup> Denotes  $n = 2094$ ; <sup>b</sup> Denotes  $n = 141$

\*Significant at the  $< .05$  level

\*\*Significant at the  $< .01$  level

**Verbal Memory (VeM).** Descriptive statistics for the 2 groups at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2094$ ,  $\mu = 79.56$ ,  $\sigma = 12.38$ ; Follow-Up:  $n = 2094$ ,  $\mu = 83.09$ ,  $\sigma = 11.26$ ), 1+ Concussions (Initial:  $n = 141$ ,  $\mu = 75.34$ ,  $\sigma = 12.50$ ; Follow-Up:  $n = 141$ ,  $\mu = 80.74$ ,  $\sigma = 11.53$ ). Mixed-Model ANOVA revealed no significant difference within groups as a function of rate of change over time ( $F = 2.609$ ,  $p = .106$ , *partial*  $\eta^2 = .001$ ). Independent Samples T-Test was employed also yielded non-significant findings ( $t(2233) = 1.615$ ,  $p = .106$ ,  $r^2 = .001$ ). Mixed-Model ANOVA revealed a significant difference between groups ( $F = 14.721$ ,  $p < .001$ , *partial*  $\eta^2 = .007$ ). This significant finding suggests that although groups do not differ in the rate at which they change over the two time points, the significant difference at initial baseline persists over-time. This finding is illustrated in Figure 6, below.

Figure 6: Verbal Memory by Concussion Group Over-Time

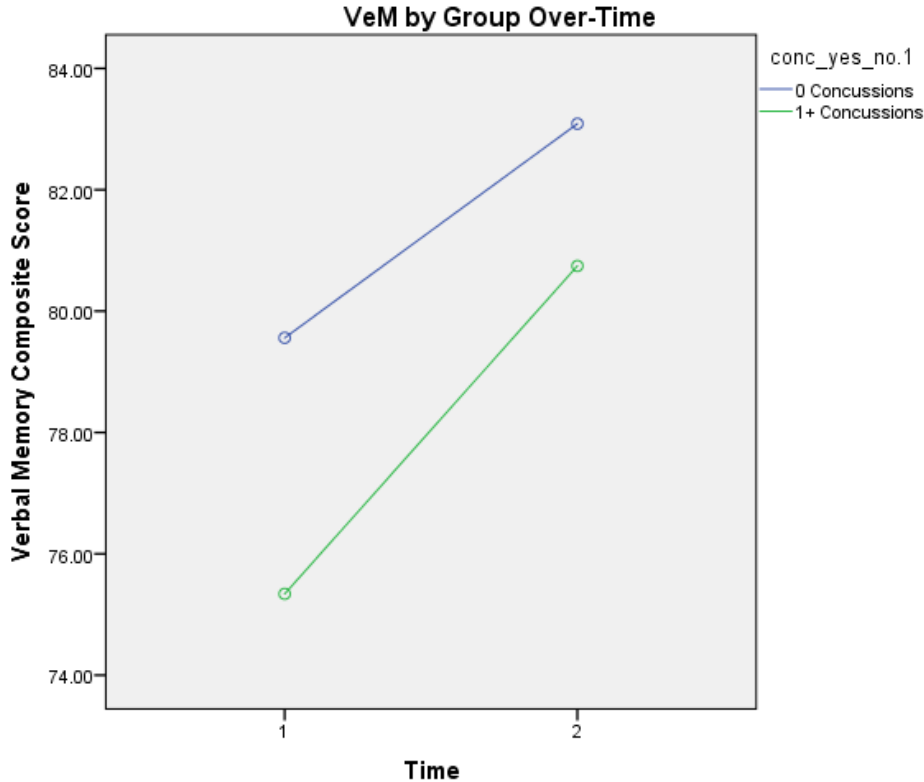


Figure 6. Verbal Memory Composite Score by concussion group over-time.

**Visual Memory (ViM).** Descriptive statistics for the 2 groups at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2094$ ,  $\mu = 67.89$ ,  $\sigma = 14.76$ ; Follow-Up:  $n = 2094$ ,  $\mu = 72.66$ ,  $\sigma = 14.08$ ), 1+ Concussions (Initial:  $n = 141$ ,  $\mu = 65.80$ ,  $\sigma = 15.13$ ; Follow-Up:  $n = 141$ ,  $\mu = 70.53$ ,  $\sigma = 14.46$ ). Mixed-Model ANOVA revealed no significant difference within groups as a function of rate of change over time ( $F = .001$ ,  $p = .976$ ,  $partial \eta^2 < .001$ ). Independent Samples T-Test was employed also yielded non-significant findings ( $t(2233) = .976$ ,  $p = .976$ ,  $r^2 < .001$ ). Although approaching significance, Mixed-Model ANOVA revealed a non-significant difference between groups ( $F = 3.668$ ,  $p = .056$ ,  $partial \eta^2 = .002$ ). This finding is illustrated in Figure 7, below.

Figure 7: Visual Memory by Concussion Group Over-Time

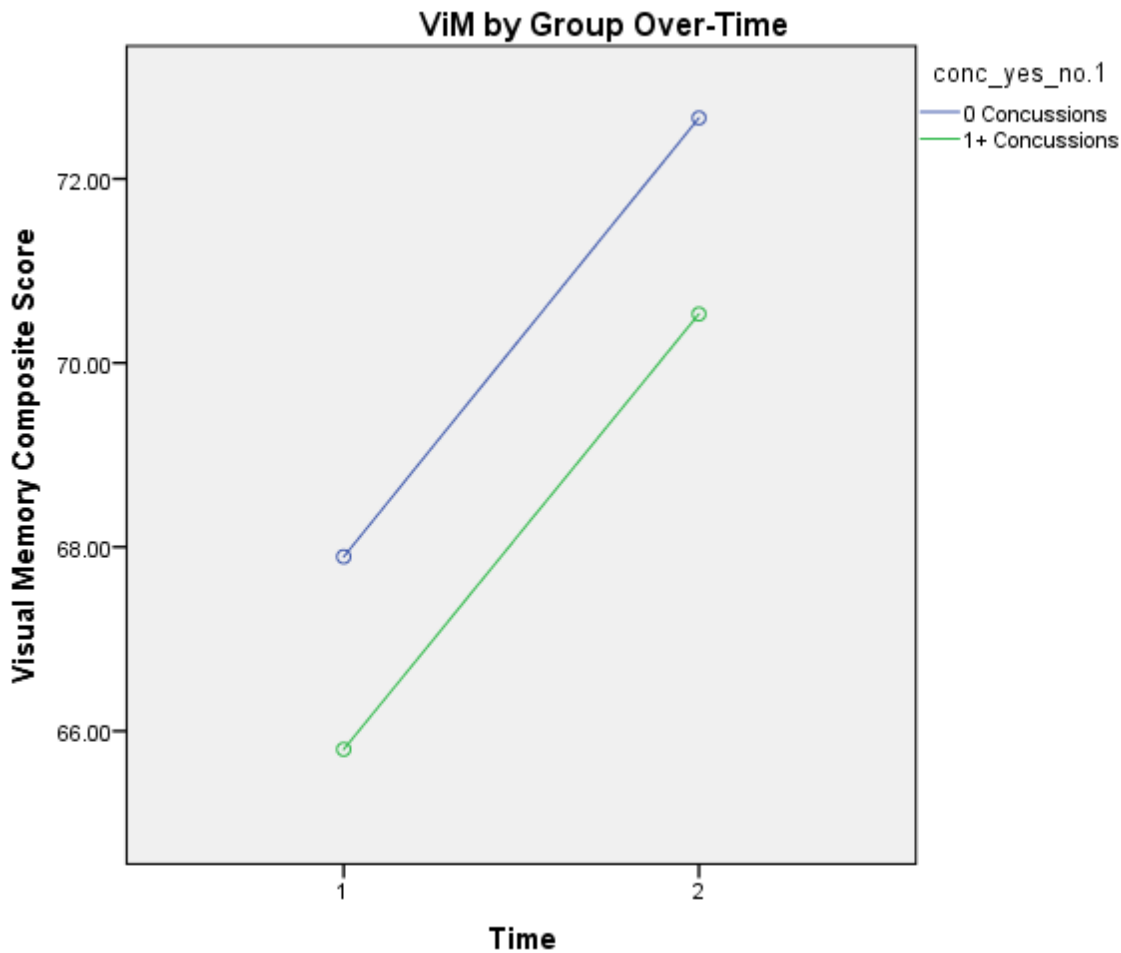


Figure 7. Visual Memory Composite Score by concussion group over-time.

**Visual Motor Speed (VMS).** Descriptive statistics for the 2 groups at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2094$ ,  $\mu = 32.98$ ,  $\sigma = 7.32$ ; Follow-Up:  $n = 2094$ ,  $\mu = 35.56$ ,  $\sigma = 7.25$ ), 1+ Concussions (Initial:  $n = 141$ ,  $\mu = 32.33$ ,  $\sigma = 7.76$ ; Follow-Up:  $n = 141$ ,  $\mu = 35.39$ ,  $\sigma = 7.22$ ). Mixed-Model ANOVA revealed no significant difference within groups as a function of rate of change over time ( $F = .909$ ,  $p = .340$ , *partial*  $\eta^2 < .001$ ). Independent Samples T-Test was employed also yielded non-significant findings ( $t(2233) = .954$ ,  $p = .340$ ,  $r^2 < .001$ ). Mixed-Model ANOVA revealed a non-significant

difference between groups ( $F = .507, p = .477, \text{partial } \eta^2 < .001$ ). This finding is illustrated in Figure 8, below.

Figure 8: Visual Motor Speed by Concussion Group Over-Time

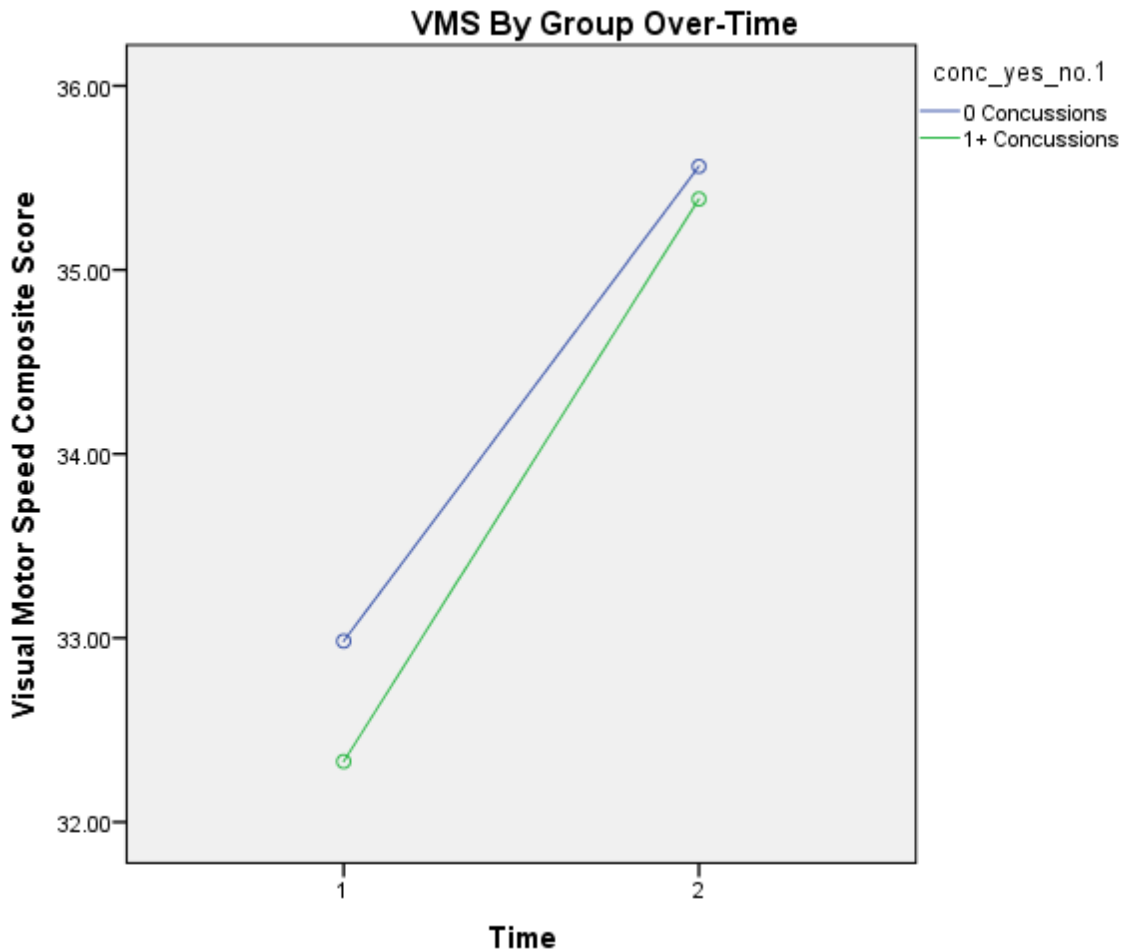


Figure 8. Visual Motor Speed Composite Score by concussion group over-time.

**Reaction Time (RT).** Descriptive statistics for the 2 groups at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2094, \mu = .651, \sigma = .129$ ; Follow-Up:  $n = 2094, \mu = .638, \sigma = .114$ ), 1+ Concussions (Initial:  $n = 141, \mu = .658, \sigma = .123$ ; Follow-Up:  $n = 141, \mu = .637, \sigma = .110$ ). Mixed-Model ANOVA revealed no significant difference within groups as a function of rate of change over time ( $F = .576, p = .448, \text{partial } \eta^2 < .001$ ). Independent Samples T-Test was employed also yielded non-significant findings ( $t(2233) = .759, p = .448, r^2$

< .001). Mixed-Model ANOVA revealed a non-significant difference between groups ( $F = .154$ ,  $p = .695$ ,  $partial \eta^2 < .001$ ). This finding is illustrated in Figure 9, below.

Figure 9: Reaction Time by Concussion Group Over-Time

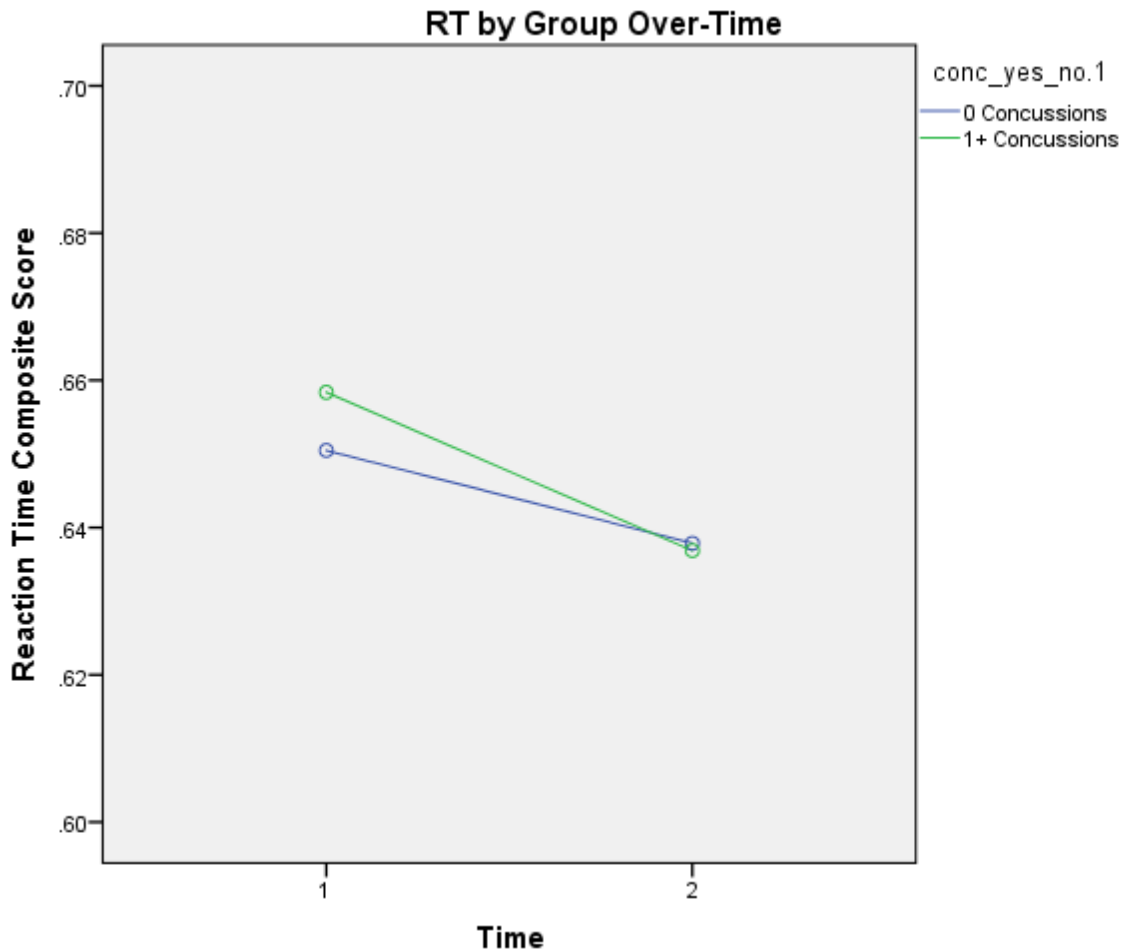


Figure 9. Reaction Time Composite Score by concussion group over-time.

**Impulse Control (IC).** Descriptive statistics for the 2 groups at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2094$ ,  $\mu = 8.11$ ,  $\sigma = 9.79$ ; Follow-Up:  $n = 2094$ ,  $\mu = 6.53$ ,  $\sigma = 6.09$ ), 1+ Concussions (Initial:  $n = 141$ ,  $\mu = 9.71$ ,  $\sigma = 10.76$ ; Follow-Up:  $n = 141$ ,  $\mu = 7.47$ ,  $\sigma = 8.83$ ). Mixed-Model ANOVA revealed no significant difference within groups as a function of rate of change over time ( $F = .600$ ,  $p = .439$ ,  $partial \eta^2 < .001$ ). Independent Samples T-Test was employed to account for unequal variances between groups and

also yielded non-significant findings ( $t(157.319) = .445, p = .657, r^2 = .001$ ). Mixed-Model ANOVA revealed a significant difference between groups ( $F = 4.764, p = .029, \text{partial } \eta^2 = .002$ ). This significant finding suggests that although groups do not differ in the rate at which they change over the two time points, the significant difference at initial baseline persists over-time. This finding is illustrated in Figure 10, below.

Figure 10: Impulse Control by Concussion Group Over-Time

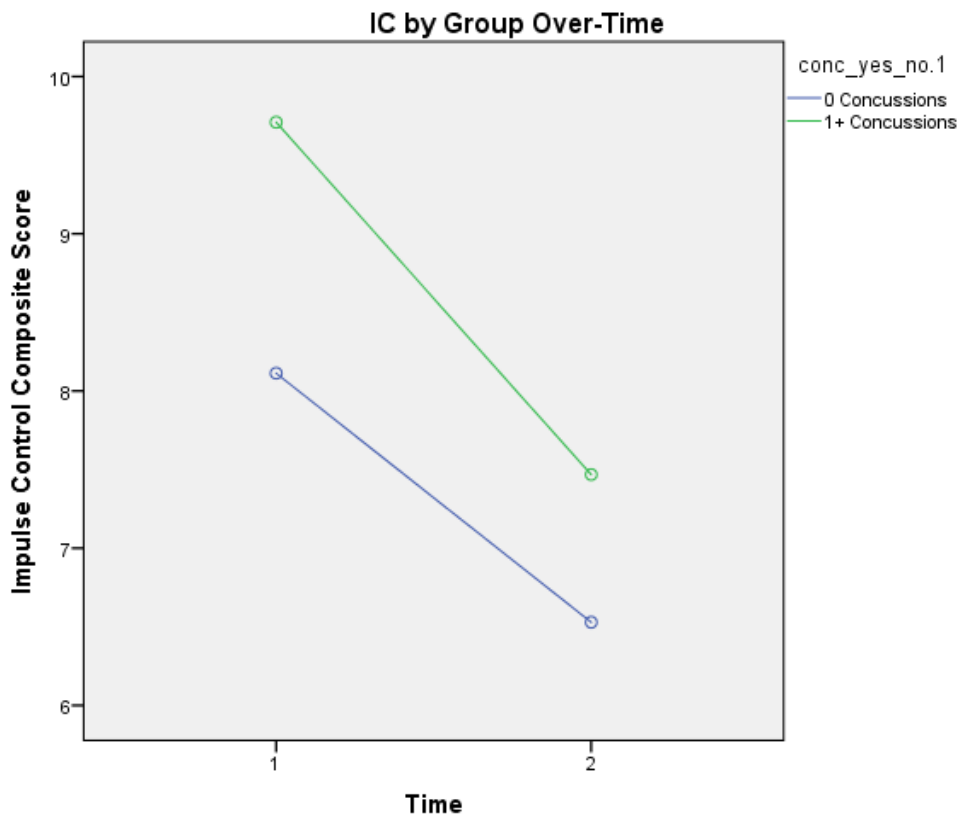


Figure 10. Impulse Control Composite Score by concussion group over-time.

**Post-Concussion Symptom Scale (PCSS).** Descriptive statistics for the 2 groups at initial baseline and follow-up baseline are as follows: 0 Concussions (Initial:  $n = 2094, \mu = 4.21, \sigma = 7.55$ ; Follow-Up:  $n = 2094, \mu = 3.56, \sigma = 7.48$ ), 1+ Concussions (Initial:  $n = 141, \mu = 5.93, \sigma = 8.10$ ; Follow-Up:  $n = 141, \mu = 4.96, \sigma = 8.40$ ). Mixed-Model ANOVA revealed no significant difference within groups as a function of rate of change over time ( $F = .218, p = .640, \text{partial } \eta^2$

< .001). Independent Samples T-Test was employed to account for unequal variances and also yielded non-significant findings ( $t(157.319) = .445, p = .657, r^2 = .001$ ). Mixed-Model ANOVA revealed a significant difference between groups ( $F = 7.707, p = .006, \text{partial } \eta^2 = .003$ ). This significant finding suggests that although groups do not differ in the rate at which they change over the two time points, the significant difference at initial baseline persists over-time. This finding is illustrated in Figure 11, below.

Figure 11: PCSS Score by Concussion Group Over-Time

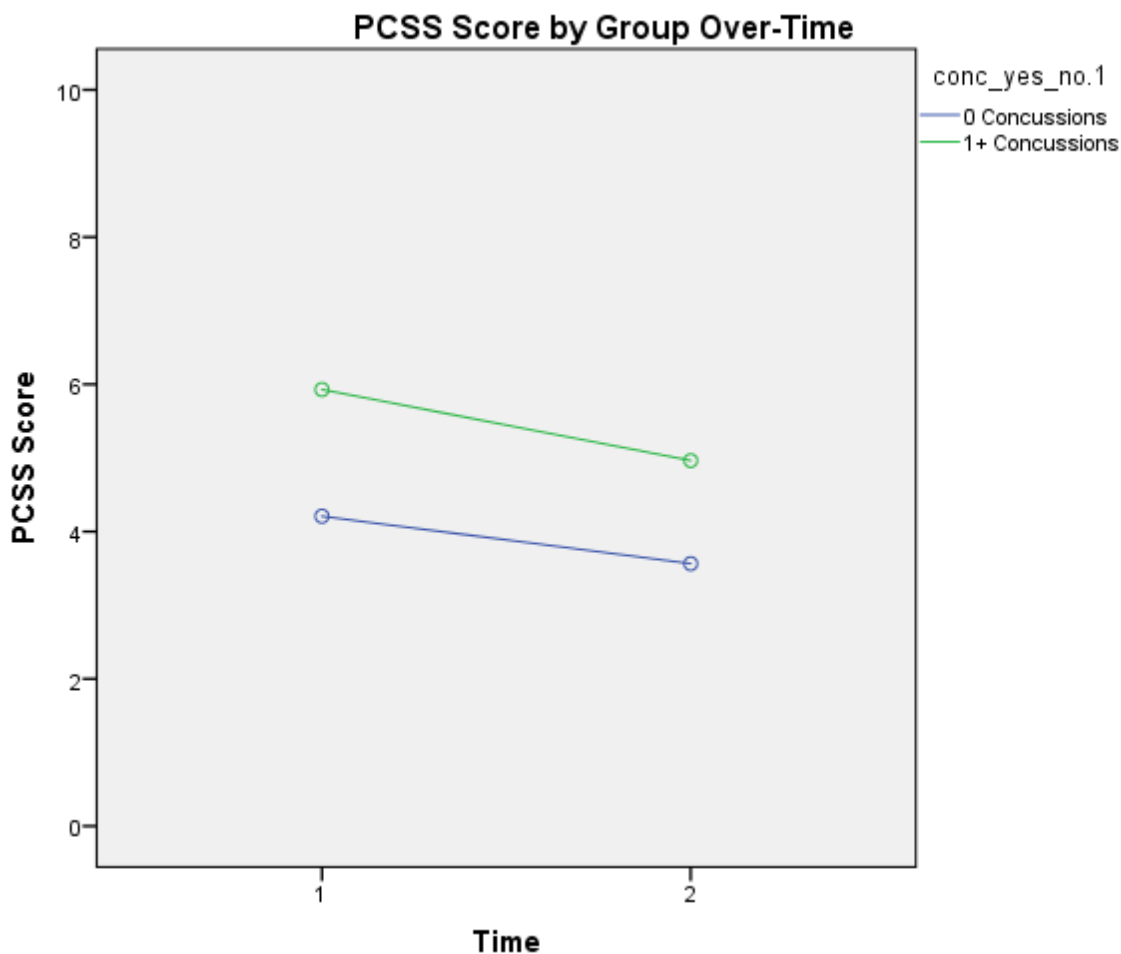


Figure 11. PCSS score by concussion group over-time.



***Hypotheses IV & V.***

The results of the analyses conducted while testing Hypothesis III limit the use of exploring moderation and the influence of indirect effects on dependent measures over time, as the effect of all differences in the rate of change over-time discovered between groups for the neurocognitive performance outcome measures were negligible. Alternative inquiries beyond the scope of this pilot study will be discussed in Chapter V.

## Chapter V

### Discussion

#### Overall Findings

The current exploratory pilot study set out to better understand the relationship between self-reported concussion history, neurocognitive functioning and associated subjective symptom experience in high school athletes. To explore this ultimate inquiry, several preliminary hypotheses were tested and analyzed using pre-season baseline neurocognitive data. Initially, neurocognitive profiles were eliminated based upon selection criteria agreed upon in the existing literature and in consultation with this dissertation committee. In total, 984 unique participants were extracted because they did not meet the required age range (ages 12 to 19), or endorsed a history of a developmental disorder typically associated with neurocognitive dysfunction (ADHD or LD). All subsequent analyses were conducted using the remaining 22,392 unique participants. The resulting sample was unique, and largely heterogeneous, with regard to age, ethnicity, and gender.

*Uniqueness of Sample.* A primary aspect of this study which makes it distinct from the existing literature is both the size and characteristics of sample. Composed of 22,392 high school athletes who completed at least one baseline neurocognitive test, the current study exceeds most, if not all articles searched with regard to volume of participants. This is especially important when considering the potential for generalizability to the population at large. Additionally, the individuals included for analyses are vastly heterogeneous when compared with samples of comparable studies. In combination, several characteristics are exclusive to this study, including gender (female participants: 38.28%), ethnicity (Black or African American: 35.23%; Hispanic or Latino: 16.97%; Asian: 1.16%), and language of origin (Spanish: 4.70%). Given the collective

makeup of these individuals, a rare opportunity is afforded to suggest epidemiological statements, should the findings support differences amongst those tested. Given the diversity of the sample, these findings are likely generalizable to multiple populations, including ethnic groups and gender.

***Hypothesis I.*** These set of analyses were employed to identify patterns of neurocognitive changes in high school athletes who did not report any history of prior concussions. Of the six Repeated Measures ANOVA's conducted (e.g., Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time, Impulse Control, and Total Symptom Score) all six yielded statistically significant changes in neurocognitive performance, or associated functioning, from initial baseline to follow-up baseline. In addition, five out of six areas tested revealed measureable, albeit small, effects of time change on average performance. That is to say, the average neurocognitive scores for high school students appear to improve over-time when tested more than once at baseline during their high school athletic career.

Several factors may be contributing to these results and warrant attention. Although possible, it is unlikely that practice effects influenced results, given the significant amount of time between testing and the established reliability of the ImPACT for use in test-retest statistical procedures (Iverson, Lovell & Collins, 2003; Nakayama, et al., 2014). Additionally, age-related normal neurological development may be a contributor to improved thinking skills, speeded processing and reduced impulsivity. Increased experience in high school academia may also assist athletes to improve these mental skills over-time. Lastly, the statistically significant, although with negligible effect (partial  $\eta^2 = .006$ ), reduction in average symptom score over-time, may relate to increased exposure to the culture of reduced symptom reporting in sport.

With these caveats in mind, the findings from Hypothesis I testing remain noteworthy and carry functional implications that can inform the literature on adolescent athlete brain development and performance, expectations for return-to-play following a suspected head injury, and allow for a measureable control group comparison for future analyses.

**Hypothesis II.** In these next sets of analyses, participants were divided into 3 groups based upon self-reported concussion history. Six one-factor between-group ANOVA's were conducted with post-hoc comparisons employed for 5 of the 6 outcome variables evaluated at initial baseline. Both outcome variables with memory components (verbal and visual) yielded a similar pattern, as the group with no reported concussions, on average, performed significantly better than their peers with reported histories of 1 and 2+ concussions. This trend, although small in effect, is measureable and carries important implications with regard to memory development in adolescent athletes.

With regard to the speeded components of testing, variable results were revealed between average group performances on outcome measures of Visual Motor Speed and Reaction Time. No differences were found between groups when compared on average VMS composite score. However, Reaction Time was significantly slower in the groups with higher reported incident of concussion for all 3 comparisons (0 Concussions vs. 1 Concussion ( $t(1115.458) = 2.130, p = .033, r^2 = .004$ ), 0 Concussions vs. 2+ Concussions ( $t(268.124) = -2.040, p = .042, r^2 = .015$ ), and the 1 Concussion vs. 2+ Concussions comparison ( $t(305.004) = -2.540, p = .012, r^2 = .021$ ). In addition, those with no reported concussion history were significantly better at inhibiting on average than their peers with 1 reported prior concussion. Taken together disinhibition and slowed reactivity are a dangerous combination that can result in risky behaviors. Certainly, the extent to which these findings are clinically significant are minimal within this study. However,

these findings add further evidence to the notion of poorer neurocognitive performance associated with higher reported concussion histories.

Lastly, the subjective nature of symptomatology associated with brain injury was evaluated between groups at initial baseline. Those with higher reported concussion history on average endorsed significantly higher symptoms associated with concussion as compared to their peers. This trend remained consistent for all three post-hoc comparisons. Similarly, the clinical significance of these differences revealed during this study is minimal. However, it does add to the current literature and provides context for the analyses conducted in Hypothesis III.

*Hypothesis III, IV & V.* Analyses conducted to test Hypothesis III aimed to evaluate changes in neurocognitive functioning over-time as a function of reported concussion history. Given the findings from Hypothesis I, similar findings in the no concussion history group were expected when groups changed to no reported concussion history vs. 1 or more reported concussions. These groups were evaluated at initial baseline and at any follow-up and change scores were analyzed. Overall, groups were not statistically different on any of the composite outcome variables, thus not necessitating the exploration of indirect effects and moderation. These findings suggest that although groups were significantly different at initial baseline on most neurocognitive measures, the interaction of group over time did not differ significantly. However, the differences that favored no concussion history on neurocognitive performance to previously concussed peers remained constant and persisted over-time.

### **Implications**

*Chronic Disease Model Interpretation.* This pilot study revealed several statically significant, unique findings which may inform the current literature regarding concussion in high school athletes. Although the rate of change between groups of previously concussed and non-

concussed adolescent athletes does not appear to differ, it is concerning that the relative weaknesses demonstrated by the previously concussed groups persisted over-time. The primary clinical contributions of the current findings may be best understood through a chronic disease model of interpretation. Small effect sizes were found at early ages for research in this area. While the degree to which groups differed on many of the statistically significant measures would not be considered to be clinically significant based on the parameters outlined in ImPACT® interpretation guide (Iverson, Lovell & Collins, 2003), several factors have the potential to manifest clinically significant deficits over the lifespan, given the young age of the study population. A combination of substantial underreporting of head injury demonstrated in the literature, coupled with the propensity for young athletes to remain engaged in their sports (and thus risk further exposure to potentially harmful mechanical forces inflicted on the brain), may contribute to more profound clinical findings in neurocognitive performance over the course of several years. Multiple hits to the head has been associated with degenerative brain dysfunction and death in the case of Chronic Traumatic Encephalopathy. Prolonged exposure to repeated blows to the head could have further effects when initiated at or before adolescence. Therefore, the observed effect sizes may increase and demonstrate both statistical and clinically meaningful significance with a decades-long progression.

***Clinical Implications.*** Noted within the results are several statistically significant findings, suggesting that many of the domains tested differ amongst concussion groups. Statistical significance, while useful, may be better understood within the context of clinical relevance and implications for those who diagnose, treat or experience head injury. This distinction between clinical and statistical significance assists in framing the findings as it pertains to their practical meaning. Of particular importance for this study is understanding how

sample size could influence the results. Given the large sample size, we would expect a higher probability of finding statistically significant results, as power (e.g., the probability of detecting a difference between groups on neurocognitive performance) holds a direct relationship with sample size. This increased likelihood of obtaining significance is meaningful when describing practical implications.

The reporting of effect size indices becomes increasingly important when extrapolating clinical significance of statistically significant results. As previously discussed, several significant findings are demonstrated with limited effect size indices. The array of statistically significant findings, while interesting and meaningful, may be tempered due to the small effect size findings. However, many results remain clinically relevant and assist in expanding the literature on brain injury, neurocognitive performance, and implications for concussion management during adolescence.

Several of the findings involved the functional impairment in high school athletes associated with a positive concussion history. In particular, adolescents with multiple head injuries are reporting consistently worse symptoms than non-injured counterparts both at baseline and with persistence over-time (PCSS Score by Group Over-Time:  $F = 7.707$ ,  $p = .006$ ,  $partial \eta^2 = .003$ ). Additionally, high school athletes with multiple head injuries are consistently worse at inhibiting impulses than their non-injured counterparts both at baseline and over-time (IC by Group Over-Time:  $F = 4.764$ ,  $p = .029$ ,  $partial \eta^2 = .002$ ). The implication of these findings are germane to the national health care conversation, as deficits in these two areas of functioning are common reasons for adolescents to seek appointments with primary care physicians.

At later ages, a leading reason for many adults and older adults to see their primary care providers are subjective memory concerns. The current findings suggesting differences in memory function that persist over-time in adolescents endorsing any concussion history compared with non-injured peers in verbal memory (VeM by Group Over-Time:  $F = 14.721$ ,  $p < .001$ , *partial*  $\eta^2 = .007$ ). Consistent with the chronic disease model, differences at this early age could lead to clinically concerning memory problems over several years with repeated exposure to additional head injury and aging in the context of multiple blows to the head.

For these reasons, dissemination of information is paramount in the prevention of a disease progression of the brain, beginning during adolescence. All involved in the organization of competitive high school sports should take responsibility for the protection and safety of the adolescent participants. From the administrators down to the players, each person can assist in the prevention of neurocognitive and symptomatic changes due to youth sport. Armed with more knowledge regarding head injury, players can make informed decisions regarding the reporting of head injury incurred during sport. The parents may also recognize signs that their adolescents are experiencing concussion-like symptoms, requiring appropriate medical attention.

Administration, coaches and athletic staff have a duty to cultivate a culture of safety for the player, and encourage reporting of suspected head injuries without fear of penalty. Collectively, these strategies may assist in the prevention of change in neurocognitive functioning at the high school level and beyond.

### **Limitations**

The current pilot study dissertation yielded several statistically significant and meaningful results, however, the study could have been improved in several ways. It is acknowledged that the massive size of the sample contributed, at least in part, to the small to



negligible effects discovered. Additionally, several of the significant findings, it can be argued, are driven by the large sample size in this study. However, large sample size is also one of this study's greatest strengths, as it lends itself to generalizability of findings. Validity may have been increased had the participants been closely monitored and matched to other like-participants on demographic and concussion related variables (e.g., time since injury, ImPACT® testing each year of high school). A significant limitation of this study was the participant self-report of concussion history which informed the group selection on these identifications. An explanation of method variance is warranted, given this potentially confounding variable.

***Method Biases.*** Potential problems in research may occur when common method variance (e.g., variance that is attributable to the measurement method rather than to the constructs the measures represent) is not minimized. Method biases are one of the main sources of measurement error and can threaten the validity of the conclusions about the relationships between measures. Podsakoff, MacKenzie, Lee, & Podsakoff (2003), review potential sources of method biases along with explanations regarding their impact on findings. For the purposes of this study, a source of potential confounding variance is attributable to the self-report gathering of information (e.g., concussion history, symptoms). A specific aspect of the common source (e.g., each student athlete rating their own experience) method effect may be social desirability. As a contributor to the well-documented underreporting of concussions in sports, the student athlete participants may have chosen to respond to questions in a manner reflective of the cultural or socially appropriate norm (e.g., sport culture, adolescent culture). Minimized self-reporting may have deflated the findings in this study. In an attempt to reduce the risk of method bias, this study utilized test results from healthy participants pre-season. Data collection occurring prior to the beginning of the athletes' respective sport may have reduced the likelihood

of secondary gain from underreporting of symptoms during the season (e.g., losing playing time). Given the limited reliability of self-reporting of concussion, the use of objective data (e.g., medical records) may have added reliability to the data and thus the findings. Despite this limitation, the current study revealed several meaningful and statistically significant results. Expanding on the current findings, future directions for research will be evaluated below.

### **Future Directions**

The current dissertation has evaluated a preliminary exploration into the effects of self-reported concussion history in high school athletes on neurocognitive performance and associated sequelae as a function of their progression over-time. Results suggest that further exploration is warranted and future studies may be conceptualized as follow-up and expansion on these findings.

Many of the findings in this study suggest that an evaluation of detailed longitudinal data extending from adolescence to adulthood would be warranted. Ideally, a sample consisting of athletes and non-athletes who are matched in age cohort and followed yearly until completion of the study would allow for a robust and informative study. Additionally, multiple data points would assist in reducing confounding variables and increasing power. For example, longitudinal neuroimaging, although not specifically for tracking concussion, could assist in clarifying the differences between non-concussed athletes with variable concussion histories. In addition, the use of medical charts for objective information will make the study more reliable. Lastly, the psychological correlates associated with concussion should not be understated. Changes may occur directly related to the effects on brain injury or secondarily in recovery from “an invisible injury.” These suggestions may assist in providing new information to the field of concussion management and rehabilitation from brain injury.

## References

- Aubry, M., Cantu, R., Dvorak, J., Graf-Baumann, T., Johnston, K., Kelly, J., & Schamasch, P. (2002). Summary and agreement statement of the First International Conference on Concussion in Sport, Vienna 2001. Recommendations for the improvement of safety and health of athletes who may suffer concussive injuries. *British Journal of Sports Medicine*, 36(1), 6-10.
- Barkhoudarian, G., Hovda, D. A., and Giza, C. C. (2011). The molecular pathophysiology of concussive brain injury. *Clinical Sports Medicine* 30(1), 33-38. Cantu, R. (2003). Brain injury-related fatalities in American football. *Neurosurgery*, 52, 846-853.
- Brooks, B. L., McKay, C. D., Mrazik, M., Barlow, K. M., Meeuwisse, W. H., & Emery, C. A. (2013). Subjective, but not objective, lingering effects of multiple past concussions in adolescents. *Journal of Neurotrauma*, 30(17), 1469-1475.  
doi:<http://dx.doi.org/10.1089/neu.2012.2720>
- Cantu, R. C., & Gean, A. D. (2010). Second-impact syndrome and a small subdural hematoma: An uncommon catastrophic result of repetitive head injury with a characteristic imaging appearance. *Journal of Neurotrauma*, 27(9), 1557-1564.  
doi:<http://dx.doi.org/10.1089/neu.2010.1334>.
- Center for Disease Control and Prevention (CDC). (2009) Heads up: concussion in sports.  
Retrieved from: <http://www.cdc.gov/concussion/HeadsUp/youth.html>.

- Cobb, S., and Battin, B. (2004). Second impact syndrome. *Journal of School Nursing, 20*(5), 262-267.
- Collins, M. W., & Hawn, K. L. (2002). The clinical management of sports concussion. *Current Sports Medicine Reports, 1*, 12-21.
- Collins, M., Stump, J., and Lovell, M., (2004). New developments in the management of sports concussion. *Current Opinion in Orthopedics, 15*(2), 100-107.
- Conners, C. K., Epstein, J. N., Angold, A., & Klaric, J. (2003). Continuous performance test performance in a normative epidemiological sample. *Journal of Abnormal Child Psychology, 31*(5), 555-62. Retrieved from <http://search.proquest.com.ezproxylocal.library.nova.edu/docview/204985614?accountid=6579>
- Covassin, T., Elbin, R. J., Crutcher, B., Burkhart, S., & Kontos, A. (2013). The relationship between coping, neurocognitive performance, and concussion symptoms in high school and collegiate athletes. *The Sport Psychologist, 27*(4), 372-379.
- Daneshvar, D. H., Nowinski, C. J., McKee, A.C., and Cantu, R.C. (2011). The epidemiology of sport-related concussion. *Clin Sports Med, 30*(1), 1–17, vii.  
doi:10.1016/j.csm.2010.08.006
- Daneshvar, D. H., Riley, D. O., Nowinski, C. J., McKee, A. C., Stern, R.A., and Cantu, R. C. (2011). Long-term consequences: effects on normal development profile after concussion. *Phys Med Rehabil Clin N Am, 22*(4), 683–700, ix.  
doi:10.1016/j.pmr.2011.08.009

- Fairchild, A. J., & Mackinnon, D. P. (2009). A general model for testing mediation and moderation effects. *Prevention Science, 10*(2), 87-99.  
doi:<http://dx.doi.org/10.1007/s11121-008-0109-6>
- Ferguson, C. J. (2016). An effect size primer: A guide for clinicians and researchers. In A. E. Kazdin (Ed.), *Methodological Issues and Strategies in Clinical Research, Fourth Edition* (pp. 301-310). doi:<http://dx.doi.org.ezproxylocal.library.nova.edu/10.1037/14805-020>
- Eime, R. M., Young, J. A., Harvey, J. T., Charity, M. J., & Payne, W. R. (2013). A systematic review of the psychological and social benefits of participation in sport for adults: Informing development of a conceptual model of health through sport. *The International Journal of Behavioral Nutrition and Physical Activity, 10*.
- Giza, C., & Hovda, D., (2001). The neurometabolic cascade of concussion. *Journal of Athletic Training, 36*(3), 228-235.
- Gómez, J. E., & Hergenroeder, A. C. (2013). New guidelines for management of concussion in sport: Special concern for youth. *Journal of Adolescent Health, 53*(3), 311-313.  
doi:<http://dx.doi.org/10.1016/j.jadohealth.2013.06.018>
- Gysland, S. M., Mihalik, J. P., Register-Mihalik, J. K., Trulock, S. C., Shields, E. W., Guskiewicz, K. M. (2012). The relationship between subconcussive impacts and concussion history on clinical measures of neurologic function in collegiate football players. *Ann Biomed Eng, 40*(1),14–22. doi:10.1007/s10439-011-0421-3.
- Harmon, K. G., Drezner, J. A., Gammons, M., Guskiewicz, K. M., Halstead, M., Herring, S. A., Kutcher, J. S., Pana, A., Putukian, M., and Roberts, W. O. (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.

"ImPACT®." Retrieved 6/24/14, 2012, from <http://www.impacttest.com>.

Iverson, G. L., Lovell, M. R., and Collins, M. W. (2003). Interpreting change on ImPACT following sports concussion. *The Clinical Neuropsychologist*, *17*(4), 460-467.

Iverson, G. L., Lovell, M. R., and Collins, M. W. (2006). Validity of ImPACT for measuring attention & processing speed following sports-related concussion. *Journal of Clinical and Experimental Neuropsychology*.

Johnstone, S. J., Pleffer, C. B., Barry, R. J., Clarke, A. R., & Smith, J. L. (2005). Development of inhibitory processing during the Go/NoGo task: A behavioral and event-related potential study of children and adults. *Journal of Psychophysiology*, *19*(1), 11-23.  
doi:<http://dx.doi.org/10.1027/0269-8803.19.1.11>

Kontos, A. P., Dolese, A., Elbin, R. J., III, Covassin, T., & Warren, B. L. (2011). Relationship of soccer heading to computerized neurocognitive performance and symptoms among female and male youth soccer players. *Brain Injury*, *25*(12), 1234-1241.  
doi:<http://dx.doi.org/10.3109/02699052.2011.608209>

Kushner, D. (1998). Mild traumatic brain injury: Toward understanding manifestations and treatment. *Archives of Internal Medicine*, *158*(15), 1617-1624.

Lam, J. M., Hsiang, J. N., Poon, W. S. (1997). Monitoring of autoregulation using laser Doppler flowmetry in patients with head injury. *Journal of Neurosurgery*, *86*(3), 438-445.  
doi:10.3171/jns.1997.86.3.0438.

Lovell, M. R., & Solomon, G. S. (2013). Neurocognitive test performance and symptom reporting in cheerleaders with concussions. *The Journal of Pediatrics*, *163*(4), 1192-1195. doi:<http://dx.doi.org/10.1016/j.jpeds.2013.05.061>

- McCrea, M., Iverson, G. L., McAllister, T. W., Hammeke, T. A., Powell, M. R., Barr, W. B., & Kelly, J. P. (2009). An integrated review of recovery after mild traumatic brain injury (MTBI): Implications for clinical management. *The Clinical Neuropsychologist*, *23*(8), 1368-1390. doi:<http://dx.doi.org/10.1080/13854040903074652>
- McCrory, P. (2001). Does second impact syndrome exist? *Clinical Journal of Sports Medicine*, *11*(3), 144–149.
- McCrory, P., Meeuwisse, W., Johnston, K., Dvorak, J., Aubry, M., Malloy, M., & Cantu, R. (2009). Consensus statement on concussion in sport: The 3<sup>rd</sup> International Conference on Concussion in Sport held in Zurich, November 2008. *Journal of Athletic Training* *44*, 434-448. PubMed doi : 10.4085/1062-6050-44.4.434
- McCrory P., Meeuwisse W.H., Aubry M., Cantu, B., Dvorak, H., Echemendia, R.J., . . . Turner, M., et al. (2013). Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012. *British Journal of Sports Medicine*, *47*, 250–258. doi:10.1136/bjsports-2013-092313
- McKee, A.C., Cantu, R.C., Nowinski, C.J., Hedley-Whyte, E.T., Gavett, B.E., Budson, A.E., . . . Stern, R.A. (2009). Chronic traumatic encephalopathy in athletes: Progressive tauopathy following repetitive head injury. *Journal of Neuropathology and Experimental Neurology*, *68*(7), 709-735.
- McKee, A. C., Stein, T. D., Nowinski, C. J., Stern, R. A., Daneshvar, D. H., Alvarez, V. E., . . . Cantu, R. C. (2013). The spectrum of disease in chronic traumatic encephalopathy. *Brain: A Journal of Neurology*, *136*(1), 43-64. doi:<http://dx.doi.org/10.1093/brain/aws307>.

- Mori, T., Katayama, Y., Kawamata, T. (2006). Acute hemispheric swelling associated with thin subdural hematomas: pathophysiology of repetitive head injury in sports. *Acta Neurochirurgica Supplement*, 96, 40–43.
- Nakayama, Y., Covassin, T., Schatz, P., Nogle, S., & Kovan, J. (2014). Examination of the test-retest reliability of a computerized neurocognitive test battery. *The American Journal of Sports Medicine*, 42(8), 2000-2005. doi: 10.1177/0363546514535901
- Omalu, B.I., DeKosky, S.T., Minster, R.L., Kamboh, M.I., Hamilton, R.L., & Wecht, C.H. (2005). Chronic traumatic encephalopathy in a National Football League player. *Neurosurgery*, 57, 128-134. doi:10.1227/01.NEU.0000163407.92769.ED
- Podsakoff, P. M., MacKenzie, S. B., Lee, J., & Podsakoff, N. P. (2003). Common method biases in behavioral research: A critical review of the literature and recommended remedies. *Journal of Applied Psychology*, 88(5), 879-903.
- Pokhrel, P., Herzog, T. A., Black, D. S., Zaman, A., Riggs, N. R., & Sussman, S. (2013). Adolescent neurocognitive development, self-regulation, and school-based drug use prevention. *Prevention Science*, 14(3), 218-228. doi:http://dx.doi.org/10.1007/s11121-012-0345-7
- Povlishock, J. T. (1992). Traumatically induced axonal injury: pathogenesis and pathobiological implications. *Brain Pathology*, 2(1), 1–12.
- Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, 40(3), 879-891.
- Proctor, M. R., & Cantu, R. C. (2000). Head and neck injuries in young athletes. *Clinics in Sports Medicine*, 19(4), 693-715. doi: 10.1016/j.csm.2010.09.001.



- Rieger, B. P., Lewandowski, L. J., Callahan, J. M., Spenceley, L., Truckenmiller, A., Gathje, R., & Miller, L. A. (2013). A prospective study of symptoms and neurocognitive outcomes in youth with concussion vs orthopaedic injuries. *Brain Injury, 27*(2), 169-178.  
doi:<http://dx.doi.org/10.3109/02699052.2012.729290>.
- Saffary, R., & Chin, L. S. (2012). From Concussion to Chronic Traumatic Encephalopathy : A Review, 351–362.
- Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience and Biobehavioral Reviews, 24*(4), 417-463.
- Tomei, K. L., Doe, C., Prestigiaco, C. J., & Ghandhi, C. D. (2012). Comparative analysis of state-level concussion legislation and review of current practices in concussion. *Neurosurgery Focus, 33*(6), 1-9. doi: 10.3171/2012.9.FOCUS12280