THE CLINICAL UTILITY AND INFLUENCE OF HABITUAL, DEVICE-MEASURED SLEEP DURATION ON BASELINE NEUROCOGNITIVE PERFORMANCE AND TOTAL CONCUSSION SYMPTOM SEVERITY IN COLLEGE-AGED INDIVIDUALS

By

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A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

Kinesiology – Doctor of Philosophy

2021

PUBLIC ABSTRACT

THE CLINICAL UTILITY AND INFLUENCE OF HABITUAL, DEVICE-MEASURED SLEEP DURATION ON BASELINE NEUROCOGNITIVE PERFORMANCE AND TOTAL CONCUSSION SYMPTOM SEVERITY IN COLLEGE-AGED INDIVIDUALS

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Context: Sport-related concussion (SRC) consensus statements encourage the use of baseline testing to compare post-injury performance to pre-injury performance to aid in return to play decisions. Previous research suggests that short sleep duration negatively influences baseline computerized neurocognitive performance and total concussion symptoms. However, these studies have only utilized self-reported and single-night measures of sleep duration. **Purpose:** The purpose of this study was to examine the clinical utility and influence of habitual, device-measured sleep duration on baseline computerized neurocognitive performance and total concussion symptom severity in college-aged individuals. **Methods:** Participants were included in the study if they were between 18 – 25 years of age and enrolled in university classes (N = 61 mean age 20.30 years). The sleep measures included: habitual, device-measured sleep duration, device-measured single-night sleep duration, subjective, single-night sleep duration, and habitual, subjective sleep duration. Participants were instructed to wear an Actigraph GT9X monitor and to complete the National Sleep Foundation Sleep diary for 7 continuous days. After 7 days, participants were administered the Immediate Post Concussion Assessment and Cognitive Testing (ImPACT) battery. **Results:** The average habitual, device-measured total nighttime sleep time was 5.37 hours. Habitual, device-measured sleep duration did not influence baseline neurocognitive performance

and total concussion symptom severity in college-aged individuals. No agreement was found between 1) device-measured single-night sleep duration, habitual, device-measured sleep duration, and subjective, single-night sleep duration; 2) habitual, device-measured sleep duration and habitual, subjective sleep duration. Subjective, single-night sleep duration significantly overestimated device-measured single-night sleep duration and habitual, device-measured sleep duration. In addition, habitual, subjective sleep duration significantly overestimated habitual, device-measured sleep duration. **Conclusion:** Although previous research suggests that self-reported single-night sleep duration negatively influences baseline CNT performance and concussion symptoms, the current study found that habitual, device-measured sleep duration did not influence baseline CNT performance and concussion symptoms. More research is needed to determine the ImPACT battery's sensitivity to habitual sleep and sleep loss. In addition, researchers and clinicians should use caution when using device-derived and subjective measures of sleep duration interchangeably.

ABSTRACT

THE CLINICAL UTILITY AND INFLUENCE OF HABITUAL, DEVICE-MEASURED SLEEP DURATION ON BASELINE NEUROCOGNITIVE PERFORMANCE AND TOTAL CONCUSSION SYMPTOM SEVERITY IN COLLEGE-AGED INDIVIDUALS

By

Morgan Anderson

Context: Previous research suggests short sleep duration negatively influences baseline computerized neurocognitive test (CNT) performance. However, previous studies have only utilized subjective and single-night measures of sleep duration.

Purpose: The purpose of this study was to examine the clinical utility and influence of habitual, device-measured sleep duration on baseline computerized neurocognitive performance and total concussion symptom severity in college-aged individuals.

Methods: This study used a cross-sectional design and included participants aged 18 – 25 years enrolled in university classes (N = 61, mean age 20.30 years). The sleep measures included: habitual, device-measured sleep duration, device-measured single-night sleep duration, subjective, single-night sleep duration, and habitual, subjective sleep duration. Participants provided informed consent, completed a sleep diary, were issued an Actigraph GT9X monitor, and instructed to wear the monitor and complete the morning and evening sections of the sleep diary for 7 continuous days. Participants completed the Immediate Post Concussion Assessment and Cognitive Testing (ImPACT) battery. Hierarchical linear regressions were used to examine the influence of habitual, device-measured sleep duration on baseline ImPACT performance. Statistical significance was set at $p \leq .05$. Equivalence testing and Bland-Altman plots were used to determine the level of agreement between 1) device-measured single-night sleep

duration and habitual, device-measured sleep duration, and subjective, single-night sleep duration; 2) habitual, device-measured sleep duration and habitual, subjective sleep duration. **Results:** The average habitual, device-measured total nighttime sleep time was 322.13 minutes (5.37 hours). Habitual, device-measured sleep duration did not influence verbal memory ($F_{2,48}$ = 0.26, p = .77; R^2 = 0.011), visual memory ($F_{2,48}$ = 0.04, p = .96; R^2 = 0.002), visual motor processing speed ($F_{2,48}$ = 0.41, p = .67; R^2 = 0.017), or reaction time ($F_{2,48}$ = 2.29, p = .11; R^2 = 0.087). A significant model was found for total concussion symptom severity ($F_{2,48}$ = 6.63, p = .003; R^2 = 0.216); but habitual, device-measured sleep duration did not significantly contribute to the model (B = -0.002, 95% CI: -0.03, 0.02, β= -.02, p = .86). Subjective, single-night sleep duration significantly overestimated device-measured single-night sleep duration ($\bar{d}$ = 151.80; Z = -5.08, $p \leq .001$) and habitual, device-measured sleep duration ($\bar{d}$ = 158.03; t_{41} = -12.30, $p \leq .001$). Habitual, subjective sleep duration significantly overestimated habitual, device-measured sleep duration ($\bar{d}$ = -153.18; t_{48} = -15.57, $p \leq .001$). In addition, no agreement was found between 1) device-measured single-night sleep duration, habitual, device-measured sleep duration and subjective, single-night sleep duration; 2) habitual, device-measured sleep duration and habitual, subjective sleep duration. **Conclusion:** Habitual, device-measured sleep duration did not influence baseline CNT performance or total concussion symptom severity. Further research should determine ImPACT battery's sensitivity to habitual sleep and sleep loss. Furthermore, future researchers should investigate how other sleep-related variables, like sleep efficiency, may impact baseline CNT performance. Researchers and clinicians should use caution when using device-derived and subjective measures of sleep duration interchangeably.

ACKNOWLEDGEMENTS

First, I would like to thank my parents, David and Lara Anderson, and brother, Ryan Anderson. Your unconditional love and support have made this all possible. Thank you for always encouraging me throughout this entire process. Second, I would like to thank Michigan State University for providing me opportunity to further my education, research, and professional development. This project would not be possible without the funding provided by the Department of Kinesiology and the Graduate School. Next, I would like to thank my advisor and mentor, Dr. Tracey Covassin. I would have not been able to reach this accomplishment without her guidance and mentorship. I would also like to thank Dr. Chris Kuenze, Dr. James Pivarnik, and Dr. Kimberly Fenn for their guidance in completing this project. Finally, I would like to thank all of my lab mates (past and present) in the Athletic Injury and Rehabilitation Lab and friends in the Department of Kinesiology for their support and friendship.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ALGORITHMS

KEY TO SYMBOLS AND ABBREVIATIONS

ADHD	Attention Deficit Hyperactivity Disorder
ADP	Adenosine Diphosphate
AE	Athletic Exposure
AMSSM	American Medical Society for Sports Medicine
ANAM	Automated Neuropsychological Assessment Metrics
AT	Athletic Trainer
ATP	Adenosine Triphosphate
BCTT	Buffalo Concussion Treadmill Test
BESS	Balance Error Scoring System
CDC	Centers for Disease Control
CI	Confidence Interval
CISG	Concussion in Sport Group
CNT	Computerized Neurocognitive Test
DVAT	Dynamic Visual Activity Test
EEG	Electroencephalography
EMG	Electromyography
GSC	Graded Symptom Checklist
GST	Gaze Stability Test
HIS	Head Injury Scale
HIT	Head Impact Telemetry
ICC	Intraclass Correlation Coefficients

ImPACT	Immediate Post Concussion Assessment and Cognitive Testing
IQR	Interquartile Range
IRB	Institutional Review Board
KD	King-Devick
LD	Learning Disorder
MANOVA	Multivariate Analysis of Variance
MDC	Minimal Detectable Change
MEQ-SA	Morningness-Eveningness Questionnaire – Short Assessment
NATA	National Athletic Trainers Association
NCAA	National Collegiate Athletic Association
NFL	National Football League
NMDA	N-Methyl-D-Aspartate
NPC	Near Point Convergence
NREM	Non-Rapid Eye Movement
PCSI	Post-Concussion Symptom Inventory
PCSS	Post-Concussion Symptom Scale
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index (PSQI)
RCI	Reliable Change Index
REM	Rapid Eye Movement
RPE	Ratings of Perceived Exertion
RPQ	Rivermead Post Concussion Symptom Questionniare
RR	Risk Ratio

SCAT	Sport Concussion Assessment Tool
SD	Standard Deviation
SOT	Sensory Organization Test
SRC	Sport Related Concussion
TBI	Traumatic Brain Injury
VMS	Visual Motion Sensitivity
VOMS	Vestibular Ocular Motor Screening
VOR	Vestibular Ocular Reflex

CHAPTER I: INTRODUCTION

Overview of the Problem

Computerized neurocognitive test (CNT) batteries are an integral part of the multifaceted approach for sport related concussion (SRC) assessment and management.[1] Specifically, CNT batteries evaluate several facets of cognition including memory, reaction time, and attention, which are commonly impacted after SRC.[2] Traditionally neuropsychological function is evaluated using paper and pencil assessments; however, recently CNT batteries have served as an alternative to traditional paper and pencil assessment due to their ability to serve large groups of athletes concurrently, ease of administration, wide availability on electronic platforms (i.e., desktop, online, iPads), availability of normative databases, and alternate test forms to reduce practice effects.[3–5]

Current SRC consensus statements[1,4] advocate for the use of a prospective (i.e., baseline [pre-injury] and post-injury serial administration) CNT assessment approach. Furthermore, these consensus statements recommend, but do not mandate, baseline neurocognitive testing.[1,4] The utility of a prospective assessment approach is demonstrated throughout the SRC literature[6–8] and more than 90% of athletic trainers (ATs) utilize a follow-up assessment approach, with approximately 70% of ATs utilizing CNT in follow-up testing.[9] Although implementing this prospective approach takes time and money,[9] it provides several benefits to clinicians including being able to compare post-injury performance to baseline performance to aid in SRC diagnosis and return to play decisions. Second, it allows for the injured athletes to serve as their own controls when comparing to post-concussion scores. Third, baseline testing may provide an

opportunity to educate athletes about SRC.[1] Finally, utilizing individual baseline data may provide more diagnostic accuracy[10,11] and lower false positive rates[12] compared to norm-referenced values. Therefore, it is essential that baseline CNT performance is an accurate representation of the individual's cognitive status.

Previous research has identified factors that influence baseline CNT performance including sex,[13–17] concussion history,[16] motivation/effort,[18–20] attention deficit hyperactivity disorder (ADHD) and learning disability (LD),[21–23] and physical exertion.[24] Previous research[13–17] suggests that males and females differ on neurocognitive performance and symptoms at baseline, however results are mixed regarding which specific cognitive domains (e.g., verbal/visual memory, reaction, visual motor processing speed) are affected. The majority of studies suggest that female athletes demonstrate better performance than males on verbal memory.[13–15,17] Whereas other studies[15,16] suggest that female athletes also perform better on visual motor processing speed and reaction time. Others[13,17] suggest males demonstrate increased performance on visual motor processing speed and visual memory. Also, female athletes report more symptoms at baseline compared to male athletes.[14,17] Previous concussion history may interact with sex with regard to baseline neurocognitive performance. Female athletes with a history of 2 or 3 or more concussions performed better on verbal memory than male athletes with 2 or 3 or more concussions.[16] In addition, female athletes with a history of 3 or more concussions demonstrated better performance on visual memory compared to male athletes with a history of 3 or more concussions. Sub-optimal effort during baseline testing has also been documented to influence neurocognitive performance;[18–20] however, it should be noted that Immediate Post Concussion

Assessment and Cognitive Testing (ImPACT), a CNT battery used by 83.5% of athletic trainers, “flags” athletes with baseline scores below validity indicators.[25,26] In addition, individuals with ADHD and LD have been found to perform worse on CNT batteries, specifically demonstrating worse performance on verbal memory, visual memory, visual motor processing speed, reaction time, and report elevated total symptom scores.[15,21,22] Finally, physical exertion has been found to negatively influence verbal memory performance in male and female recreational athletes.[24]

Sleep is essential for maintaining health and well-being; however, 35% of adults sleep less than the recommended 7 – 9 hours.[27,28] Insufficient sleep duration is reported to adversely influence several facets of health, including metabolism and mood.[29,30] In addition, insufficient sleep may increase the risk of several chronic diseases such as cardiovascular disease and obesity.[31] Specifically, a growing body of literature suggests that short sleep duration (< 7 hours) negatively influences domains of cognition including learning and memory, reaction time, and auditory vigilance.[32,33] For college students, sleep is particularly important as it may negatively influence academic performance, health, and mood.[34] However, 25% of college students report sleeping less than 6.5 hours per night and only 29.4% report sleeping 8 or more hours per night.[35] College provides young adults with a sense of freedom that is seldom experienced in adolescence. Unlike shiftwork or clinical populations, college students may choose to practice irregular sleeping habits in order to meet academic and social responsibilities.[36] Therefore, college students provide a unique population to study sleep habits without the influence of clinical concerns or shiftwork schedules.[36] Given the public health burden of insufficient sleep duration and the adverse influence on

cognitive function, there is a need to explore the influence of sleep duration on baseline CNT performance and total concussion symptom severity.

Although several studies exist, there does not seem to be conclusive evidence to suggest short sleep duration negatively influences baseline CNT performance. Sufrinko et al.[37] observed worse verbal memory, visual memory, and slower reaction time in adolescents who reported getting less than 5 hours the night before baseline testing. McClure and colleauges[38] found that high school and collegiate athletes with less than 7 hours of sleep the night before baseline testing demonstrated deficits in verbal memory, visual memory, and reaction time. While some studies[37,38] found that short sleep duration negatively influences CNT performance, others have only found increases in symptomology as a result of short sleep duration.[39–41] Mihalik and colleagues[39] observed increased symptomology in collegiate student athletes who reported short sleep duration the night before baseline testing, but found no deficits in cognitive performance. Similarly, Moran and colleagues[40] reported that high school athletes with less that 8 hours of sleep the night before baseline testing demonstrated greater total symptom scores. These contrasting results and methodological gaps in the literature, highlight the need for further research.

Several gaps exist in the literature investigating the influence of short sleep duration on baseline CNT performance and total concussion symptom severity. First, most studies[37–41] have relied on subjective measures of sleep quantity in form of questionnaires (e.g., Pittsburgh Sleep Quality Index) or single-item questions (i.e., “How many hours of sleep did you get last night?”). Given the subjective nature of these reporting methods, it is possible that recall bias and inaccuracies in sleep duration could

be introduced. Second, previous studies[37–41] have only investigated the effects of a single night of sleep and have failed to investigate the effects of multiple nights (i.e., habitual) of sleep on baseline CNT performance and symptoms. It is possible that a single night of sleep is not representative of a typical or unusual night of sleep, whereas habitual sleep duration, or sleep accumulation across multiple nights, accounts for multiple factors such as lifestyle choices, environment, and biological drive (i.e., circadian rhythm).[42] In the only study to utilize an objective measure of sleep duration and measure sleep across multiple nights, Stocker and colleagues[43] observed that sleep loss was associated with decreased visual memory, reaction time, and visual motor processing speed.[43] Given that only one study has examined the effects of objective, habitual short sleep duration on CNT performance and symptoms, there is a need to expand on these findings.

The results of this study may contribute to the existing literature examining the influence of short sleep duration on baseline CNT performance and total concussion symptom severity. Documenting the effect of habitual, device-measured sleep duration on baseline CNT performance and total concussion symptom severity may inform best practice guidelines for baseline CNT assessment by introducing the need to identify individuals who will not perform at the level representative of normal functioning or may identify individuals at risk for producing an invalid baseline score. Furthermore, the results of the current study may have significant clinical implications for post-concussion management. Specifically, baseline CNT scores that do not accurately represent an individual's cognitive status may lead to premature return to play, which may increase the risk for further injury or prolonged recovery.[44,45]

Purpose of the Study

The purpose of this study was to examine the clinical utility and influence of habitual, device-measured sleep duration on baseline computerized neurocognitive performance and total concussion symptom severity in college-aged individuals.

Specific Aims and Hypotheses

Specific Aim 1:

To investigate the influence of habitual, device-measured sleep duration on baseline computerized neurocognitive performance and total concussion symptom severity in college-aged individuals.

Hypothesis 1:

College-aged individuals who experience short habitual, device-measured sleep duration will be associated with worse baseline computerized neurocognitive performance and total concussion symptom severity.

Specific Aim 2:

To assess the agreement between both device-measured single-night and habitual, device-measured sleep duration and subjective, single-night sleep duration in college-aged individuals.

Hypothesis 2a:

There will be a high level of agreement between device-measured single-night sleep duration and subjective, single-night sleep duration in college-aged individuals.

Hypothesis 2b:

There will be a low level of agreement between habitual, device-measured sleep duration and subjective, single-night sleep duration in college-aged individuals.

Specific Aim 3:

To examine the agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration in college-aged individuals.

Hypothesis 3:

There will be a high level of agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration in college-aged individuals.

CHAPTER II: REVIEW OF LITERATURE

Definition of Sport Related Concussion

The American Medical Society for Sports Medicine (AMSSM) defines concussion as a traumatically induced transient disturbance of brain function that involves a complex pathophysiological process.[46] Whereas, the Concussion in Sport Group (CISG) defines SRC more broadly as a traumatic brain injury induced by biomechanical forces.[1] Although commonly used synonymously with traumatic brain injury, SRC is often considered a subset of mild traumatic brain injury on the less severe end of the brain injury spectrum. As knowledge about SRC has increased, various definitions of SRC from numerous professional organizations have been published.[1,4,46–48] Although these definitions of SRC lack equivalence, there are common elements included. In a recent systematic review,[49] researchers identified 6 professional organizations with published operational definitions of concussion. In all definitions used by professional organizations, loss of consciousness was an optional presentation for making diagnosis.[1,4,46–48] In addition, all definitions suggest impairment occurs as a result of SRC, however definitions differ on the onset, duration, mechanism, and resolution of impairment or are not specific about the impairment. Finally, the CISG[1] and Team Physician Consensus[48] statement includes a statement on neuroimaging. Specifically, SRC results in functional disturbances rather than structural changes therefore, standard structural neuroimaging returns normal results.[1,48] The lack of one SRC operational definition introduces several limitations. First, different definitions complicate the comparison of injury epidemiological studies. Second, a universal definition may help researchers standardize protocols when conducting studies. Finally, one definition

may help health care providers educate and inform patients and parents when discussing SRC. In order to address these limitations, experts should validate a gold standard definition of SRC.

Epidemiology of Sport Related Concussion

Approximately 44 million children and adolescents[50] participate in organized sports and 460,000 athletes participate in varsity collegiate athletics every year.[51] Sport participation provides several benefits including physiological, psychological, and social benefits. However, despite these benefits, there is the risk of injury, specifically, SRC.

Youth Athletics (≤ 13 Years)

It is estimated that 1.1 – 1.9 million SRCs occur in children (≤ 18 years) annually in the United States.[52] Previous research suggests that concussion makes up 2.7[53] – 9.6%[54] of all injuries in middle school football players. In addition, Veliz and colleagues[55] reported that approximately 17% of eighth graders had a history of diagnosed concussion. However, there is limited research examining the incidence of SRC in middle school athletics, with the majority of the research examining incidence of concussion in middle school football athletes.[53,54,56,57] In an early study, Dompier and colleagues[53] examined time-loss and non-time loss injuries in youth (9 – 14 years) football athletes. Study results revealed a concussion injury rate of 0.5 per 1000 athletic exposures (AEs) for practice and competition combined.[53] Interestingly, Kerr and colleagues[54] reported nearly double that, 1.0 per 1000 AEs, for both practice and competition combined in American youth football players. In another study, Kontos and colleagues reported a concussion incidence rate of 1.76 per 1000 AEs in 8 – 12 year old football players.[58] Although several investigators[53,54,56,57] have examined the

incidence of concussion in middle school athletics, these studies are limited by only focusing on football and lack data on the incidence of concussion in girl athletes. Beachy and colleagues[59] examined the overall concussion incidence rates in 27 middle school sports between 1988 – 2008. The overall concussion incidence rate, 0.07 per 1000 AEs[59] was lower than those reported previously.[53,54,56,57] In one of the few studies to examine the concussion incidence rate in youth athletes, Black and colleagues[60] compared the risk of concussion between ice hockey leagues where body checking was permitted and one where body checking was not permitted. The results revealed that the ice hockey league that allowed body checking had a concussion incidence rate of 2.83 per 1000 player hours, while the league that did not permit body checking had an incidence rate of 0.91 per 1000 player hours.[60]

In addition to describing the overall incidence of concussion in middle school sports, researchers have also examined the incidence of concussion stratified by event type (competition versus practice), sex differences, and sport. First, the majority of research suggests that concussion incidence is higher in competition compared to practices.[53,54,56,58,59] The concussion incidence rate for competition ranges from 0.14 – 6.16 per 1000 AEs.[58,59] While the concussion incidence rate for practice ranges from 0.05 – 1.04 per 1000 AEs.[56,59] In middle school football players, Kontos et al. reported an incidence density ratio for concussions in competitions and practices of 25.91 per 1000 AEs.[58] Beachy and colleagues[59] reported a relative risk ratio of 0.33 suggesting that middle school athletes were more at risk for sustaining a concussion during a competition compared to practice.

Very little research exists on sex differences in incidence of concussion of middle school athletes since the majority of studies[53,54,56–58,61] have focused on football athletes, which are traditionally male only. However, Beachy and colleagues[59] reported the concussion incidence rate was almost 3 times higher for boy athletes (0.09 per 1000 AEs) compared to girl athletes (0.03 per 1000 AEs). However, when football athletes were excluded, the concussion risk was similar between boys and girls.[59]

High School Athletics

Results of early studies showed that concussions represented 5.5 – 7.5% of all high school injuries.[62,63] However, a more recent study reported that concussions represent approximately 13% of all injuries in high school athletics.[64] Previous researchers[63,65–69] have documented overall concussion incidence rates in high school athletics, with concussion incidence ranging from 0.17 – 0.51 per 1000 AEs in multiple high school sanctioned sports. Interestingly, overall concussion incidence rates have increased over time in high school athletics, as reported by previous studies.[67,68] Lincoln and colleagues[67] examined the incidence of concussion in 12 high school sports between 1997-1998 and 2007-2008 athletic seasons. The overall concussion rate increased from 0.12 per 1000 AEs to 0.49 per 1000 AEs. Similarly, Rosenthal et al.[68] reported a significant increase (0.23 to 0.51 per 1000 AEs) in overall concussion incidence in high school athletes.

There are several reasons hypothesized for an increase in concussion incidence in high school athletics. First, the increase has been be attributed to new state laws and changes in sport-governing bodies, such as the National Athletic Trainers Association (NATA), Centers of Disease Control (CDC), and CISG Consensus. Washington state

was the first to pass legislation in 2009 outlining the medical care procedures for concussion and by 2014 all states passed legislation.[70] Although each state law is slightly different, the majority of the state laws include: 1) education for coaches, parents, and/or athletes; 2) immediate removal from play for any athlete who has or is suspected of sustaining a concussion; 3) clearance from a health care professional for the athlete to return to play no sooner than 24 hours following injury.[70] In addition, the CDC launched an initiative, “Heads Up”, in 2004 to educate coaches, parents, and school professionals,[71] while the NATA[4] and CISG[1] published consensus statements on specific evidence based guidelines for identify, managing, and treating SRC. It is possible that legislation, initiatives, and consensus statements have increased SRC awareness in athletes, coaches, and parents.

Second, it is possible that having certified ATs on-site to identify and diagnose concussion may be another reason for increased concussion incidence over time. In a recent study, McGuine and colleagues[72] investigated how the presence of ATs influenced SRC reporting. Thirty-one high schools were categorized as low, mid, and high AT availability and the incidence of concussion was recorded at each high school.[72] Not surprisingly, the incidence of SRC was lower for the low-AT schools (0.24 per 1000 AEs) than the mid-AT (0.64 per 1000 AEs) and high-AT (0.87 per 1000 AEs) schools.[72] Therefore, the results of the study suggest that a higher level of AT availability is associated with increased SRC reporting.[72]

Although overall concussion incidence rates in high school athletics range from 0.17 – 0.51, all sports do not have the same risk.[63,68] Previous research suggests football has the highest concussion rates that range from 0.33 – 4.01 per 1000 AEs.[62–

[65,69] In addition, previous researchers have reported concussion rates in girls soccer (0.73 per 1000 AEs),[68] boys lacrosse (0.67 per 1000 AEs),[65] wrestling (0.57 per 1000 AEs),[68] and boys ice hockey (0.54 per 1000 AEs).[66] A majority of research suggests that in general, high school girls are at a greater risk for concussion than high school boys;[65,66,68,69,73] however, Lincoln and colleagues[67] reported a greater risk for concussion in boys compared to girls, which could be due to the inclusion of football. When evaluating sex-comparable sports, i.e., sports played by the same rules for both sexes, female athletes have a greater risk for concussion.[65,66,69,73] In a recent study, O'Connor and colleagues[65] reported a higher overall concussion rate for softball (Risk Ratio [RR] = 4.14), basketball (RR = 1.76), soccer (RR = 1.53), and track and field (RR = 3.81). In addition, previous researchers[65,66] reported greater concussion risk in boys high school lacrosse than girls high school lacrosse. However, it should be noted that boys and girls lacrosse cannot be directly compared due to significant rule differences such as, body checking and required protective equipment in boys but not girls lacrosse.[65,66]

It is hypothesized that biomechanical, social norms, and hormonal differences may explain the sex disparity in SRC incidence. First, previous research suggests that female athletes have decreased neck girth, strength, and head-neck segment.[73–76] Due to the lower neck strength and girth, greater linear and angular accelerations and displacement may occur and therefore, girl's head-neck may rotate at a greater speed resulting in more concussions.[73] Second, previous research suggests female high school athletes may be more likely to report a concussion to an authority figure than male high school athletes.[77,78] Male high school athletes may not disclose their

concussion because they did not think it was serious, did not want to let their team down, or did not want to miss a game. In addition, it is possible that the male sport culture and stigmas within male sports may contribute to this thinking.[79] Finally, there is preliminary evidence taken from animal data to suggest that estrogen may predispose girl women athletes to increased risk of concussion.[80]

Collegiate Athletics

Between the 2009-2010 and 2013-2014 athletic seasons, concussions made up 6.2% of all injuries reported in the National Collegiate Athletic Association (NCAA).[81] Previous research suggests that the risk for concussion is greater in collegiate athletics (0.43 per 1000 AEs) compared to high school athletics (0.23 per 1000 AEs; RR = 1.86).[69] Specifically, in football collegiate athletes (3.74 per 1000 AEs) the risk for concussion is higher than in football high school athletes (2.01 per 1000 AEs; RR = 1.86). Daneshvar and colleagues[82] summarized data from the NCAA Injury Surveillance System from the 1988-1989 through 2003-2004 athletic seasons. The authors reported that the concussion incidence rate increased from 0.17 to 0.34 per 1000 AEs.[82] Since then, researchers have continued to report increases in concussion incidence in collegiate athletes. Kilcoyne et al.[83] examined the concussion incidence in three collegiate Division I military academy football teams and reported an increase from 0.57 per 1000 AEs to 1.16 per 1000 AEs between the 2009-2010 and 2010-2011 athletic seasons. One season concussion incidence rate in multiple sports, not just exclusively football, have also been examined. Gessel and colleagues[69] reported a concussion incidence rate of 0.43 per 1000 AEs. Similarly, other researchers[81,84] reported an incidence rate of 0.45 per 1000 AEs in 25 collegiate sports. Although the

majority[69,81,82,84,85] of studies have examined the concussion incidence rate of collegiate athletes in multiple sports, others[57,64,83] have examined the incidence rate exclusively in football collegiate athletes. Dompier and colleagues[57] reported a concussion rate of 3.74 per 1000 AEs, while Kerr et al[64] reported a higher concussion rate of 7.29 per 1000 AEs.

Specifically, the majority[57,69,86,87] of research reports suggests that collegiate football athletes have the highest concussion incidence rate ranging from 0.38 – 3.74 per 1000 AEs.[57,86] However, one study[81] reported that collegiate wrestlers had higher concussion incidence (1.09 per 1000 AEs) than collegiate football players (0.67 per 1000 AEs). Other sports such as, women's lacrosse (1.35 per 1000 AEs)[87], women's soccer (1.07 per 1000 AEs)[87], and men's ice hockey (0.79 per 1000 AEs)[81] have higher concussion rates than football. Zuckerman and colleagues examined differences in concussion risk in sex comparable sports.[81] Similar to research in high school athletes, women collegiate basketball (RR = 1.53), soccer (RR = 1.83), lacrosse (RR = 1.64), and softball (RR = 3.65) players were at an increased risk for concussion compared to male equivalent athletes.[81] Interestingly, ice hockey was the only sex comparable sport with women and men athletes having equal risk.[81]

In response to the high concussion incidence rate in collegiate football players, Ivy League coaches recommended that the kick-off line be moved from the 35-yard to the 40-yard line and that the touchback line be moved from the 25-yard to the 20-yard line.[88] It was intended, with this rule change, to have more kickoffs land in the endzone, effectively reducing the chance of the player advancing the ball.[88] Prior to the rule change the concussion incidence rate during kickoff plays in Ivy League football players

was 10.93 per 1000 plays.[88] After implementation of the rule change, the concussion incidence rate exclusively during kickoff plays was 2.04 per 1000 plays.[88] In addition, the overall concussion incidence rate decreased from 3.04 per 1000 plays, before the rule change, to 1.22 per 1000 plays, after the rule change. Even in the National Football League (NFL), head injury incidence decreased from 3.70 per 1000 kickoffs to 1.20 per 1000 plays after implementation of rule changes.[89]

Given the risk of injury that is associated with sport participation, SRC will continue in youth, high school, and collegiate athletics. Future researchers should continue to improve concussion surveillance systems and expand strategies to reduce SRC occurrence in athletics.

Biomechanics of Sport Related Concussion

Early research[90] describes linear and rotational acceleration as the primary mechanism of concussion. Linear and rotational acceleration can occur by both direct and indirect impacts to the head. Examples of direct impacts include helmet-to-helmet, collision with a teammate or opponent, collision with equipment, whereas examples of indirect impacts include colliding with a teammate or opponent causing abrupt stopping (i.e., whiplash). Linear acceleration is hypothesized to cause transient intracranial pressure gradients, while rotational acceleration is hypothesized to cause shear strain injury in the neural tissue. However, it is possible that concussion is caused by a combination of linear and rotational accelerations. Numerous studies have recorded head impact data in various sports including: tackle[91–103] and flag[101] football, soccer[98,104–106], ice hockey[98,107–110], rugby[111,112] athletes, and major league baseball catchers and umpires[113].

The majority of studies examining the magnitude of head impacts has been conducted in football athletes of all levels, including youth, high school, and college. In youth football, median peak linear accelerations range from 10.0g to 22.0g, with only 2% of head impacts being greater than 80.0g.[99,100,102,103] Compared to youth tackle football, youth flag football players have been shown to have lower odds of sustaining head impacts less than 20g, but with higher odds of sustaining peak rotational accelerations between 2500.00 – 7499.99 rad/s^2.[101] Interestingly, concussion tolerance may be lower for youth athletes compared to older athletes. Specifically, Camplettano and colleagues[95] reported youth athletes sustained average peak linear acceleration of 62.4 ± 29.7g compared to 102.5 ± 32.7g for adults. In addition, youth athletes sustained average peak rotational acceleration of 2609.0 ± 1591.0 rad/s^2 compared to 4412.0 ± 236.0 rad/s^2.[95]

Urban and colleagues[94] reported that median peak linear accelerations for high school football players ranged from 15.2g to 27.0g. Mihalik and colleagues[91] reported that collegiate football players sustained head impacts between 21.0 and 23.0gs. Similarly, Crisco et al.[92] reported that the 50th percentile for peak linear acceleration was 20.0g and 95th percentile values 49.5g in collegiate football athletes. Interestingly, Rowson and colleagues[114] reported that only about 10% of impacts result in peak linear acceleration above 40.0g and only 8.35% of head impacts were above peak acceleration rotation of 3000.0 rad/s^2 and none of the collegiate athletes in the study sustained a concussion.

Recently researchers have utilized sideline biomechanical data collected via the Head Impact Telemetry (HIT) System, to associate head impacts with concussion

occurrence. By using this monitoring system, researchers could quantify head impacts and eventually identify single head impact events that result in concussion. Researchers have sought to identify the minimum threshold that causes concussion. In tackle football, specifically professional football, researchers have suggested that 70 – 75g were required in order for a concussion to occur and this was identified as the minimum threshold.[115] However, this study is limited by lack of real-time head acceleration data. Rather that collect real-time head acceleration data on the field of play, Pellman and colleagues[115] simulated concussions in the laboratory. Since the publication of Pellman et al.[115], researchers have sought to identify the minimum threshold associated with concussion. Funk and colleagues[116] recorded that the average linear acceleration for concussion was 145.0 ± 35.0g; however, following a risk analysis for concussion, researchers reported a minimal threshold of 100g as an identifier of concussion. In an study of high school and collegiate football athletes, Beckwith and colleagues[117] reported that single impacts associated with diagnosed concussion were peak linear accelerations of 112.1 ± 35.4g and peak rotational accelerations of 4253.0 ± 2287.0 rad/s^2. Similarly, Rowson and colleagues found that concussions in high school and collegiate football players were associated with 102.0 ± 33.0g for linear acceleration and 4412.0 ± 2326.0 rad/s^2 for rotational acceleration.[118,119] However, Stemper and colleagues[97] reported that the mean linear accelerations associated with concussion in collegiate football athletes was 71.0 ± 30.0g and mean rotational accelerations of 3379.0 ± 1775.0 rad/s^2. Interestingly, 56% of the concussions reported by Stemper and colleagues were associated with head impacts that were indicative of less than a 1% risk of injury.[97,120] Furthermore, Wilcox and colleauges[107] reported a lower threshold for

concussion in male and female ice hockey players. Specifically, the average peak linear acceleration of concussion causing impacts in collegiate ice hockey players was 43.0 ± 11.5g and the peak rotational acceleration was 4030.0 ± 1435.0 rad/s^2.[107] It is possible that the inclusion of female athletes could be a reason for the significantly lower injury threshold.[107]

Although predicting concussion occurrence using sideline head impact technology may be advantageous for injury identification and diagnosis, caution should be taken when interpreting these data. It is possible that some players sustain high acceleration impacts that do not result in a concussion. Similarly, it is possible that some athletes may sustain a concussion as a result of low acceleration impacts. Even though numerous studies have attempted to define a concussion threshold, there is still no clear impact that always results in concussion in athletes. In addition, these studies have only investigated single head impact events. Although single head impacts often result in concussion, it is argued that repetitive head impacts, at magnitudes lower than what may result in a concussion, may lower the tolerance for injury.[97,121]

Pathophysiology of Sport Related Concussion

Concussion is characterized as a functional injury, referring to microstructural injury to the neural tissue. Immediately after a concussive injury a series of cellular events, known as the neurometabolic cascade, occur.[122] The neurometabolic cascade is characterized by bioenergetic challenges, cytoskeletal and axonal alterations, impairments in neurotransmission, vulnerability to delayed cell death, and chronic dysfunction.[122] Immediately after biomechanical injury, neuronal depolarization occurs due to intracellular potassium efflux, through voltage-gated channels.[122] This event

triggers a positive feedback loop, which increases the number of open voltage-gated channels and further promoting depolarization.[122] In addition, glutamate, a potassium efflux promoting neurotransmitter, binds to N-methyl-D-aspartate (NMDA) receptors, opening potassium/calcium channels, which results in the accumulation of calcium in the cell.[123] Intracellular calcium has a demonstrative effect causing cell damage and mitochondrial impairment.[124] Consequently, a depression-like state spreads throughout the cell.[122] In an effort to restore cellular homeostasis and meet cellular demands, the mitochondria attempt to increase Adenosine Triphosphate (ATP) production by activating ATP-dependent sodium potassium pumps.[125,126] As a result, the cell begins hyperglycolysis, which causes a depletion of intracellular energy reserves and increased Adenosine Diphosphate (ADP). Simultaneously, this need for energy is occurring in an environment of normal of diminished cerebral blood flow.[122]

Changes in cerebral blood flow occur soon after concussion due to alterations in acute and chronic vasoreactivity. It is hypothesized that the primary mechanism for changes in vasoreactivity is reduced endothelial and smooth muscle responsiveness occurring due to endothelial nitric oxide production, which may contribute to vulnerability to secondary injury and chronic symptom provocation with continued exertion.[128,129] Interestingly, previous research suggests that cerebral blood flow alterations may persist beyond symptom resolution.[130,131] Specifically, researchers have observed a correlation between functional MRI cerebral blood flow and initial symptom severity; however, cerebral blood flow may take longer to return to baseline levels compared to symptoms and neurocognitive performance.[130,131] In a recent study, Meier and colleagues[130] assessed and compared the recovery of cerebral blood flow to cognitive

and behavioral symptoms in collegiate athletes. Interestingly, cognitive and behavioral symptoms resolved at 1 week and 1 month, respectively.[130] In addition, cerebral blood flow in the dorsal midinsular cortex was decreased at one month post-injury and inversely related to psychiatric symptoms in collegiate athletes.[130] Similarly, Maugans et al.[132] examined cerebral blood flow alterations in children, aged 11 – 15, after SRC. Children with SRC demonstrated symptom score resolution and recovered reaction time at 14 days and 30 days, respectively.[132] However, only 27% of children demonstrated cerebral blood flow values at control levels by 14 days post-injury.[132] By 30 days post injury, 64% of children had cerebral blood flow values at control.[132] However, another study suggests that reduced cerebral blood flow values may persist up to 40 days post-concussion.[133] Although previous research[130,132,133] suggests that cerebral blood flow is altered after SRC, more studies are needed to understand the time in which levels may normalize in concussed athletes.

In addition to metabolic and cellular changes, neuronal pathway alterations can occur following SRC. Particularly, axons are vulnerable to impact-acceleration, leading to sheer stress and tension resulting from coup-contrecoup injuries, such as SRC.[134,135] Damage occurs at the neurofilaments and microtubules, which can disrupt bidirectional axonal transport, isolate the synapse, potentially interfere with normal neurotransmission, or may lead to potential for axonal disconnection (axotomy).[122] However, it is important to note that primary axotomy, or immediate axonal disconnection, does not occur in a majority of individuals with SRC, rather it is the neurotransmitter flux that occurs immediately after injury that causes axotomy.[136,137] Consequently, axotomy is exacerbated by the accumulation of calcium ions.[138] Recent

research[122] has linked the physiological changes that occur as part of the neurometabolic cascade to clinical signs and symptoms of concussion.

Signs, Symptoms, and Impairment after Sport Related Concussion

Sport-related concussion is an unique injury in that it is a heterogeneous injury, in which individuals present with a wide variety of signs and symptoms. Given that individuals with SRC present differently, assessment and management can prove difficult for clinicians. Currently, there is no biomarker or test that clinicians can utilize to diagnose SRC on the sideline. Therefore, clinicians must utilize a series of assessments, termed a multifaceted approach, in order to appropriately identify individuals with SRC. Generally, individuals with SRC present with one or more of the following: symptoms (e.g., headache), physical signs (e.g., loss of consciousness), balance problems, behavioral changes (e.g., irritability), cognitive impairment (e.g., decreased reaction time), and/or sleep disturbance (e.g., trouble falling asleep).[1] An individual's specific post-injury signs, symptoms, and impairments have significant implications for recovery and treatment.

Signs and Symptoms

SRC diagnosis involves the assessment of signs and symptoms. However, signs and symptoms of concussion are not specific to concussion. For example, concussion symptoms can also occur in other sport related conditions including dehydration, heat-related illness, or anemia.[139] No one symptom checklist is used universally. Currently, several checklists exist that capture patient reported symptoms after concussion including the Post-Concussion Symptom Scale (PCSS), Post-Concussion Symptom Inventory (PCSI), Graded Symptom Checklist (GSC), Head Injury Scale (HIS),

Rivermead Post Concussion Symptom Questionnaire (RPQ). In addition, in 2012 the Concussion in Sport Group developed the Child Sport Concussion Assessment Tool – 3 in order to provide a developmentally appropriate symptom evaluation for children aged 5 – 12 years of age.[140]

Given the functional nature of concussion, clinicians rely on athletes to disclose their concussion symptoms in the event of injury. Therefore, it is necessary that athletes are knowledgeable about common symptoms of concussion. In an effort to improve concussion knowledge and improve concussion disclosure, the NCAA mandates that every collegiate athlete receive formal concussion education in the form of handouts, lectures, and or emails.[141] However, although considerable effort has been made to improve symptom identification and improve concussion reporting, athletes continue to not disclose their injuries. Previous research suggests that up to 50%[142,143] of high school football athletes and up to 68%[144] of collegiate football athletes reported they had sustained a concussion that they did not report. In addition, 42% of women's soccer, 36% of men's lacrosse, and 36% of wrestling collegiate athletes have not reported a concussion in the past.[144] Non-disclosure of concussion poses significant consequences, such as increased risk of catastrophic injury (e.g., second impact syndrome)[122] or prolonged recovery.[45] Given the ramifications of continuing to play with a suspected concussion, it is imperative to create targeted interventions to improve concussion reporting behaviors.

Previous research suggests that 99% of concussions result in symptoms.[145] Headache is commonly reported as the most frequently endorsed symptom, with 87.5% – 94.7% of adolescent and collegiate athletes reporting experiencing headache after

injury.[65,66,145–148] In addition, approximately 85% of children present with headache after concussion.[149] Other commonly endorsed symptoms include: dizziness (61.3% – 73.8%)[65,66,145,147–149], difficulty concentrating (61% – 54.8%)[66,145,147,148], sensitivity to light (52.6% – 46.6%)[65,147], and sensitivity to noise (39.3%)[65]. Specifically, in children, 64.2% endorsed fatigue, making it the second most reported symptom; while another study found it to be the most severe.[149,150] Early researchers believed that loss of consciousness was required for positive concussion diagnosis, however recent research suggests that loss of consciousness is an infrequent result from concussion. Specifically, McCrea and colleagues[151] reported only 6.4% of athletes experience loss of consciousness, whereas other authors have reported frequencies of less than 5%.[145,147,148]

In addition to understanding frequently reported symptoms at initial presentation, researchers have identified symptoms that take longer to resolve. Eisenberg and colleagues[149] reported that sleep disturbance, frustration, forgetfulness, and fatigue symptoms were more likely to present at follow-up in children with concussion. Similarly, Blinman et al.[150] observed that up to 38% of patients reported excess sleep as the most frequent symptom and trouble falling asleep as the most severe symptom at 2 – 3 weeks post-concussion. Interestingly, more than 25% of patients were still reporting headache after one month of injury, while another study reported 43% of patients still reported headache after 3 months.[149,152] Furthermore, 20% of children were still reporting fatigue a month after injury.[149] In addition to headache and fatigue, previous research suggests irritability, sleep disturbance, frustration, poor concentration, and fogginess take the longest to recover.[146,149] Whereas, nausea, depression, dizziness,

and double vision are the most quick to recover after concussion.[149] Results of these studies indicate that certain symptoms are slower to resolve compared to other symptoms, suggesting the need for targeted management practices.

In an effort to better inform clinical practice and provide a more targeted management approach, researchers have used factor analytical methods to create symptom factors, which are comprised of similar symptoms.[153] Identifying symptom factors at baseline (i.e., pre-injury) and post-injury could provide critical information to the clinician. Baseline symptom factors may provide the clinician with an idea of what symptoms may be prominent after injury. In addition, identifying post-concussion symptom factors may help guide concussion treatment approaches. For example, athletes with sleep symptoms may benefit more from cognitive behavioral therapy than athletes with predominately cognitive symptoms.[153]

Using the PCSS, Pardini and colleagues[154] reported 4 symptom factors: cognitive, sleep problems, emotionality, and somatic. In a follow up study, Kontos and colleagues[153] examined PCSS symptom factors in concussed athletes within 7 days of injury. The results of the study revealed a 4 factor solution: cognitive-fatigue-migraine, affective, somatic, and sleep.[153] Researchers have also examined the factor structures of other common symptom scales including the HIS[155], GSC[156], and RPQ[157,158], and Sport Concussion Assessment Tool (SCAT) symptom inventory.[159] For the HIS, researchers observed a 3 factor solution with somatic, cognitive, and neuropsychological post-concussion symptom factors.[155] Similarly, 3 post-concussion symptom factors (Mood and Cognition, General Somatic, and Visual Somatic) were revealed for the Rivermead Post-Concussion Symptoms Questionniare.[158]

Post-concussion symptom reporting, although subjective, is an important marker of recovery. Although many researchers have documented the time to symptom resolution, more research is needed to understand time to symptom resolution after concussion. Early research[151,160–165] suggested that symptoms resolved 5 – 10 days after concussion. Guskiewicz and colleagues[160] reported that, on average, symptom resolution took 3.5 days in collegiate football players. In addition, about 88% of collegiate football players were asymptomatic within one week of injury.[160] In a similar study, McCrea and colleagues[161] reported that 91% of collegiate football athlete symptoms returned to baseline by 7 days. Similarly, Erlanger and colleagues[166] reported that post-concussion symptoms alleviated on average 6.02 days after injury. In addition, Wasserman and colleagues[84] observed that 35.7% of collegiate athlete's symptoms resolved in less than or equal to 3 days after injury. By one week post injury, 60.1% of collegiate athletes reported being symptom free.[84] Only 6.2% of collegiate athletes to over 4 weeks for symptoms to subside completely.[84] Similarly, Makdissi and colleagues, found that only 10 – 15% of patients had symptom recoveries greater than 10 days.[164] However, other research suggests[149,167–170] that symptom resolution takes longer. Henry and colleagues[170] measured post-concussion symptoms weekly for 4 weeks. The results of the study suggest that symptoms improve significantly from week 1 to week 2, but improvement slows thereafter.[170] Approximately 45% of athletes reached asymptomatic status by 3 weeks and by 4 weeks, 56% of athletes were asymptomatic.[170] Interestingly, it's possible that discrepancies in symptom resolution could be due to the definition of symptom recovery used in these studies. Some studies[84,146,160,166,168,169] define symptom recovery as participants being deemed

asymptomatic meaning that the total symptom score is 0. Other studies[151,161,163,165,171,172] have defined symptom recovery as returning to baseline, comparing symptoms to a control group, normative baseline levels, or some other definition.[149,167]

Neurocognitive Impairment

The assessment and management of concussion has shifted from relying on subjective, self-reported symptoms (i.e., “Tell me how you are feeling?”) to neurocognitive assessments that provide objective, quantifiable data on the cognitive status of the injured athlete. Given that some athletes withhold and/or minimize post-concussion symptoms,[173] there is need for objective assessments to help corroborate subjective symptom reports. Neurocognitive assessment is often described as the “cornerstone” of concussion assessment and has the advantage of introducing objectivity to concussion assessment.[174] Early researchers and clinicians relied on traditional paper and pencil neuropsychological tests, such as the Trail Making Test, Stroop Color Word Test, and WAIS-III Digit Span Test.[175] Recently, traditional paper and pencil measures have been replaced by CNT due to its ability to administer baseline tests to large groups of athletes concurrently, ease of administration, wide availability on electronic platforms (i.e., desktop, online, iPads) availability of normative databases, and alternate test forms to reduce practice effects.[3–5] Computerized neurocognitive testing is one tool that has been widely implemented for concussion management and provides an objective complement to athlete symptom reports. Serial post-concussion CNT administration may help clinicians make return to play decisions as it may provide critical information, especially in the acute phase of injury.[1]

Currently, there are several different CNTs available for concussion assessment including: Automated Neuropsychological Assessment Metrics (ANAM), C3Logix, CogSport, Concussion Resolution Index, CNS Vital Signs, ImPACT, XLNTbrain, and XLNTbrain Sport. In a recent study, Lempke and colleagues examined ATs concussion assessment and management practices and they reported that 60% of ATs utilize some form of CNT. However, the ImPACT battery is the most commonly used, specifically with approximately 84% of ATs reportedly using the test for concussion assessment and management.[25]

The ImPACT battery is a neuropsychological screening tool that evaluates concentration, attention, memory, visual motor speed, and reaction time that has been used in high school collegiate, and professional athletes. The ImPACT battery calculates 5 composite scores: verbal memory, visual memory, processing speed, reaction time, and total symptoms.[176] Previous research reports that the ImPACT battery is a reliable CNT specifically designed to assess SRC. Given it is recommended that CNT be administered in a serial fashion in order to track recovery in athletes, adequate test-retest reliability must be ensured. Researchers have examined the test-retest reliability of ImPACT at 1 hour (Intraclass Correlation Coefficients [ICCs] = 0.51 – 0.85)[177], 1 week (Pearson correlation coefficient = 0.67 – 0.79)[178], 1 month (ICCs = 0.60 – 0.88)[179], 45 day (ICC = 0.70 – 0.87)[176], 50 days (ICCs = 0.74 – 0.91)[176], 1 year (0.62 – 0.82)[180], and 2 years (0.46 – 0.74) intervals[181]. Overall, previous research suggests that ImPACT is a reliable neurocognitive tool that can be used in a serial administration manner.

Previous researchers have also examined validity measures of ImPACT. Maerlender and colleagues[182,183] examined the construct, convergent, and divergent validity of the ImPACT battery in collegiate football players. Researchers compared scores on the ImPACT battery to traditional neuropsychological measures in healthy male athletes and the results revealed that all ImPACT domain scores (verbal memory, visual memory, visual processing speed, and reaction time) were significantly correlated with traditional neuropsychological measures.[183] Study results suggest that ImPACT has acceptable construct, convergent, and divergent validity.[182,183] Other studies examined the sensitivity and specificity of ImPACT. Schatz and colleagues[184] were to first to examine the diagnostic utility of ImPACT in high school athletes. The results of the study showed that 81.9% of athletes in the concussion group and 89.4% of athletes in the non-concussed group were correctly identified.[184] Similarly, Broglio et al.[185] reported a sensitivity of 91.7% and specificity of 89.4% in Division I collegiate athletes, which are considered high levels. Finally, it is important to understand the range of measurement error associated with repeat test administrations. Understanding the possible measurement error allows for the clinician to determine deterioration, improvement, and recovery following concussion.[178] Reliable change indices (RCIs) are used to assess whether the change between repeat administrations are reliable and meaningful.[186] ImPACT RCIs are: verbal memory = 6.83, visual memory = 10.59, reaction time = 0.05 seconds, processing speed = 3.89, and total symptoms = 7.17.

Previous researchers have documented neurocognitive impairment in collegiate[161,171,187–189] and high school[7,8,167,178,190–193] athletes after concussion. Iverson and colleagues[8] were one of the first groups to track neurocognitive performance across

time in athletes. Athletes were administered ImPACT 1 – 2 days, 3 – 7 days, and 1 – 3 weeks after concussion. The results of the study revealed that neurocognitive impairment were largely resolved within 5 days of concussion and fully resolved by 10 days.[8] In another study, McCrea et al.[161] examined the effects of concussion on neurocognitive performance in collegiate student-athletes. Collegiate student-athlete neurocognitive performance recovered to baseline levels within 5 to 7 days after concussion.[161] However, other studies suggest that neurocognitive impairment takes longer to resolve. McClincy et al.[6] reported that neurocognitive deficits took at least 14 days to resolve in high school and collegiate athletes. Covassin and colleagues[7] examined the cognitive performance in concussed high school athletes. Concussed high school athletes demonstrated significantly decreased reaction time up to 14 days after injury with reaction time impairment recovering at 21 days after injury.[7] In addition, verbal memory and processing speed impairment recovered by 14 days after injury.[7] Overall, this study suggests that neurocognitive impairment may take up to 14 days to resolve in high school athletes.[7] Interestingly, however, Henry et al.[170] reported that high school athletes took longer for neurocognitive impairments to recover. Specifically, in contrast to Covassin et al.[7], verbal memory did not demonstrate significant improvements until 4 weeks after concussion. In addition, visual memory, processing speed, and reaction time demonstrated a linear recovery trajectory, significantly improving over time up until 3 weeks after injury.[170] It is possible that differences in these findings, particularly in neurocognitive performance, could be due to age differences.

Covassin and colleagues[194] examined age differences in neurocognitive performance after concussion 2, 7, and 14 days after injury. Study results revealed that high school athletes demonstrated worse neurocognitive performance, particularly verbal and visual memory, compared to collegiate athletes.[194] Similarly, Field et al.[187] reported that high school athletes had significantly worse memory performance compared to collegiate athletes. Furthermore, Iverson et al.[8] reported that 37% of high school athletes were still impaired on 2 or more neurocognitive composites 10 days after concussion. Finally, Murdaugh and colleagues[193] assessed neurocognitive performance in athletes ranging from 8 – 21 years following concussion. Similar to previous research,[8,187,194] the results of the Murdaugh et al.[193] suggest that younger athletes had significantly worse neurocognitive performance compared to older athletes.[193] Interestingly, although younger athletes performed significantly worse initially compared to older athletes, younger athletes improved quickly overtime.[193] There are several hypotheses for the occurrence of age differences in neurocognitive performance after concussion. First, the structure of the youth brain differs from the adult brain related to the geometry, structure, and physiological responses to mechanical and emotional stress.[193] Second, children have less developed cervical musculature and higher head to neck ratio compared to adults.[195] Finally, children may be more vulnerable to longer term sequelae due to the brain systems in charge of skill acquisition.[196] Given the results of these studies, clinicians should consider age differences when interpreting neurocognitive performance after concussion. In addition to age differences, previous research has documented sex differences in neurocognitive performance after concussion.

Previous research[170,189,192,194,197–200] is mixed on whether sex differences exist in neurocognitive performance after concussion. Broshek and colleagues[189] were the first to evaluate the influence of sex on neurocognitive performance after concussion. Their results suggest that female high school and collegiate athletes demonstrated worse simple and complex reaction time compared to male high school and collegiate athletes.[189] Furthermore, female athletes were 1.7 times more likely than males to have neurocognitive impairment after concussion.[189] In a follow up study, Covassin et al.[199] examined sex differences in neurocognitive function after concussion. Results revealed that female collegiate athletes performed significantly worse on visual memory than male collegiate athletes after concussion.[199] This finding of decreased visual memory is replicated in several other studies.[192,194] In addition to visual memory deficits, these studies document verbal memory impairment in female athletes compared to male athletes.[192,194] However, other studies[170,197,200] suggest that sex differences do not exist in neurocognitive performance after concussion. Unlike previous researchers, Zuckerman and colleagues[200] found no differences in visual or verbal memory between concussed male and female soccer players. Furthermore, when including a wide variety of sports, Sufrinko et al.[197] reported no sex differences in neurocognitive performance after concussion. Similarly, no differences were found in neurocognitive performance between male and female athletes when followed for 4 weeks.[170] It is possible the contrasting results could be due to methodological differences such as low sample sizes, including specific sports such as soccer, and time until first test administration. Overall, research[189,192,194,198,199] suggests that there are sex differences in neurocognitive performance after concussion. Therefore, clinicians should compare

post-injury neurocognitive performance to an athlete's own baseline performance or sex comparable normative values.[201]

Vestibular and Ocular Motor Impairment

In addition to symptoms and neurocognitive impairments, vestibular and ocular motor impairments are common after concussion. Specifically, it is estimated that 60 – 81% of athletes suffer from vestibular impairments and symptoms, while 42 – 45% of athletes demonstrate ocular impairments and symptoms after concussion.[202–204] Athletes with vestibular impairments may complain of dizziness, unstable vision, difficulty focusing, motion sickness, difficulty navigating busy visual environments, and imbalance.[205] Whereas, ocular motor impairment manifests as blurred vision, diplopia, difficulty reading, eyestrain, headache, and difficulty with visual scanning.[205] Importantly, undetected vestibular and ocular motor impairments may lead to increased symptoms, academic difficulties, and anxiety/mood changes.[205] Given the frequency of vestibular and ocular motor impairments and the potential consequences of impairment, there is a need to include an assessment within the multimodal framework for concussion. Currently, the gold standard for assessing vestibular and ocular motor impairments include the Dynamic Visual Activity Test (DVAT) and Gaze Stability Test (GST). However, these assessments may not be feasible given their cost and a requirement that they are administered by trained professionals. Given that sideline evaluation is an essential component in the assessment of concussion, it is important that ATs have access to a brief tool to appropriately assess vestibular and ocular motor impairments on the sideline.

In an effort to address the need for a brief vestibular and ocular motor assessment, researchers developed the Vestibular Ocular Motor Screening (VOMS) tool,[202] which assesses symptom provocation following a series of vestibular and ocular motor tasks. The VOMS consists of 4 ocular motor components (smooth pursuits, horizontal and vertical saccades, near point convergence [NPC] distance and symptoms) and 3 vestibular components (horizontal and vertical vestibular ocular reflex [VOR] and visual motion sensitivity [VMS]). Before administering the VOMS, patients report their pretest symptoms of headache, dizziness, nausea, and fogginess on a 11-point Likert scale ranging from 0 = none to 10 = severe. Following the administration of each vestibular or ocular task, patients rate their symptoms on the scale.

Traditionally, the VOMS is scored using a total score by summing the individual symptom scores for headache, dizziness, nausea, and fogginess. Previously published[202] clinical cutoff scores for identifying concussion were determined as any total symptom score ≥ 2 on any VOMS component and average NPC distance ≥ 5cm. Recently, researchers have advocated for a change scoring method, which is calculated as the difference between pretest symptoms and the total symptom score for each VOMS component. Previous research suggests that the VOMS is a sensitive and reliable tool. The VOR, VMS, and NPC distance components of the VOMS have a combined sensitivity of 89%. In addition, the VOMS has a high internal consistency that ranges from Cronbach $\alpha = .92$ to $\alpha = .97$ in youth and collegiate athletes.[202,206–209] Furthermore, previous research reports that the VOMS has a low false positive rate that ranges from 2 – 18%.[202,206–211]

Previous researchers have examined changes in vestibular and ocular motor symptoms and impairments in adolescent and collegiate athletes.[202,212,213] Mucha and colleagues[202] administered the VOMS within 7 days of injury and showed that 61% of concussed athletes reported symptom provocation on the VOR, whereas smooth pursuit and vertical saccade only provoked symptoms in 33% of concussed athletes.[202] Compared to controls, concussed athletes demonstrated significantly worse scores on all VOMS components.[202] In addition, concussed athletes demonstrated significantly greater NPC distance than control athletes.[202] Specifically, while investigating vestibular impairment after concussion, Corwin et al.[212] reported that 81% of youth athletes demonstrated abnormal VOR. However, it should be noted that these studies are limited due to their cross-sectional designs and lack of prospective data. Utilizing a prospective method of assessment provides several advantages including allowing the patient to serve as their own control and the comparison of post-injury data to baseline data.

Elbin et al.[210] were the first to examine prospective changes in vestibular and ocular motor impairment after concussion in adolescent athletes. Athletes were administered the VOMS at baseline, 1 – 7 days, and 8 – 14 days after concussion. The results revealed that VOMS total scores were significantly worse 1 – 7 days and 8 – 14 days after concussion compared to baseline total scores. Similar to VOMS total scores, VOMS change scores were significantly worse at 1 – 7 days post injury.[210] However, when using VOMS change scores, impairments at 8 – 14 days were reported for only the vertical VOR and VMS components.[210] The results of this study suggest that vestibular and ocular motor impairments are evident after concussion, particularly in the acute period (< 7 days). Interestingly, Henry and colleagues[170] reported that vestibular

and ocular motor impairment significantly decreases from week 1 – 2 to week 3 – 4, however it is possible some athletes could still be experiencing vestibular and ocular motor impairments 4 weeks after injury.

In addition to the VOMS, the King-Devick (KD) is another sideline tool that assesses the visual system after concussion. Originally intended as a reading tool to assess ocular motor function and identify learning disabilities, the KD test is a rapid number naming test that requires attention, language, and concentration.[214] Currently, the KD test is part of the multifaceted assessment approach used on the sideline immediately after suspected concussion. Until recently, the KD test was administered using a physical, spiral bound booklet with 3 test cards. Currently there is a version designed for a tablet. Athletes are instructed to read out loud a series of numbers from left to right as quickly as possible while the clinician keep time. The total time for the athlete to read all three cards is recorded, as well as the number of errors made in reading the test cards. The fastest (best) time of the 2 trials is recorded as the KD test time. Previous research suggests that the KD test is reliable with test re-test reliability ICCs ranging from 0.60 to 0.97, indicating high test-retest reliability.[211,215–226] In addition, several studies[222,223,227] have examined the clinical utility of the KD test and have returned mixed results. Fuller and colleagues[227] revealed a sensitivity of 59.6% and a specificity of 39.2% in elite rugby athletes, whereas Hecimovich et al.[222] reported a sensitivity of 98% and a specificity of 96% in Australian football players. Finally, Naidu et al.[223] found a sensitivity of 62% and a specificity of 84% in Canadian football players. So, although the KD test is a highly reliable tool, caution should be taken due to low sensitivity and specificity.

Per the KD user manual instructions, if an athlete performs slower than their recorded baseline best total time or makes an error after a suspected concussion they should be removed from competition and evaluated further for concussion. However, previous research suggests that the KD test has a high false positive rate. In healthy, non-concussed athletes, when concussion is not suspected, athletes demonstrate slower (i.e., worse) KD performance. Elbin and colleagues[226] reported that 38% of non-concussed high school athletes demonstrated slower KD times between two assessments. Similarly, Worts et al.[211] reported a 36% false positive rate in adolescent athletes, while Breedlove et al.[228] reported a 27% false positive rate. Rather than relying on absolute change, researchers have determined minimal detectable change and RCI, which may address the high false positive rate, by calculating the smallest clinically meaningful difference between post-concussion and baseline best total KD times. In a study by Heick et al.[229] the minimal detectable change across all trials was 6.35 seconds. Similarly, Alsalaheen et al.[219] reported a minimal detectable change of 6.10 seconds. However, Elbin and colleagues[226] reported a larger minimal detectable change (MDC) of 7.55 seconds. However, these studies only included adolescent athletes. Studies that calculated MDC values in collegiate and professional samples reported lower MDC values ranging from 5.5 – 6.8 seconds.[230–232] Results of these studies may help clinicians determine if the change demonstrated by athletes is due to concussion or measurement error.

Previous researchers[218,226,233] report that KD times are significantly slower (worse) in concussed athletes compared to baseline and/or control athletes. Siedman and colleagues[233] reported that high school athletes on average took 19.1 seconds

longer to complete the KD test after concussion compared to baseline. Similarly, Galetta et al.[218] reported that MMA fighters took 18.1 seconds longer post-head trauma compared to their baseline performance. Furthermore, Elbin and colleagues[226] reported that 72% of adolescent athletes performed worse on the KD test after concussion taking, on average, 14.1 seconds longer after injury. However, other researchers[223,230,234,235] report the change in baseline to post-injury performance to be smaller. For example, King et al.[234] reported a difference in 4.7 seconds between baseline and post-concussion KD performance in amateur rugby players. Similarly, Naidu et al.[223] and Galetta et al.[230] reported decreased KD performance of 5.1 and 5.9 seconds after concussion, respectively. In addition, Dhawan and colleagues[235] reported that concussed athletes performed 7.4 seconds slower on the KD test post-concussion compared to baseline performance. However, caution should be taken when interpreting these results due to the small sample sizes. The majority of the studies[218,221,222,230,233–235] investigating changes in pre- to post-SRC KD performance had a range in sample size from 7 to 20 athletes with concussion, therefore more research is needed. In addition, several studies[223,226] have examined the percentage of concussed athletes who perform faster (better) during post-injury examination. Specifically, Naidu and colleagues[223] reported that 38% of concussed athletes actually demonstrated faster KD performance during sideline evaluation, whereas Elbin et al.[226] reported that 28% of concussed athletes improved on KD performance. It is possible that faster sideline KD times in athletes with concussion could be due to higher motivations to return to play following suspected injury.[223] Although previous research suggests that the KD test demonstrates high reliability, the KD test should not be relied

on as a stand-alone sideline concussion assessment, the KD should be used in addition to other assessments in the multifaceted approach.

Balance Impairment

Approximately 30% of athletes report balance dysfunction after SRC.[66,236] It is hypothesized that damage to the peripheral receptors or structural damage to the central processing structures resulting in inhibited sensory integration are the two mechanisms responsible for balance disturbance following SRC.[237] Balance disturbance is defined as the inability to stand in an upright position without deviating outside the base of support.[237] Given that balance deficits are common following injury, clinicians should include a balance assessment in the multifaceted approach. Several measures of balance are available: The Balance Error Scoring System (BESS), Sensory Organization Test (SOT), tandem gait, and the Romberg test. Similar to measures of vestibular and ocular motor function, balance assessments range from low-technology, subjective tests (e.g., BESS) to expensive, objective clinical assessments (e.g., SOT). The most common assessment used to measure balance deficits following SRC is the BESS test.[238]

The BESS test was originally developed to measure static posture in collegiate athletes[161,239], but has been used to measure balance impairment in high school athletes as well.[240–242] The BESS is quick, easy to administer, and inexpensive. However, it is a subjective test that should be administered by trained clinicians. During the BESS, athletes are asked to position themselves in 3 different stances (double-leg, single-leg, tandem) on a firm and foam surface, for a total of 6 stances.[243] While athletes are in these stances, trained researchers or clinicians count errors that include:

opening eyes, lifting hand(s) off the iliac crest, stepping, stumbling, or falling, moving the hip more than 30° of flexion or abduction, lifting the forefoot or heel, and remaining out of position for more than 5 seconds.[243] For each error an athlete commits, they receive a point, earning potentially the maximum of 10 points. Athletes maintain the stance for 20 seconds. Previous research has documented the intra-rater reliability and inter-rater reliability in high school and collegiate athletes of the BESS with ICCs ranging from 0.63 – 0.92 and 0. 44 to 0.96, respectively.[244–249] Although the BESS has been shown to be reliable, previous researchers suggest that the BESS may only detect impairment after 3 – 5 days after injury.[161,239] However, Oldham and colleagues[250] reported no significant differences in BESS errors between baseline and post-concussion.

In addition, to the BESS, the tandem gait has been used to evaluate postural control after SRC. Previous iterations of the SCAT (i.e., SCAT3) have included a tandem gait protocol of 4 time trials; however, more recently the SCAT5 includes it as part of the neurological assessment.[251] Recently, research suggests that the tandem gait is more sensitive and specific to postural control changes after SRC than the BESS.[250] Evaluating dynamic balance, speed, and coordination may provide a better idea of how higher control centers are functioning post-concussion. Previous researchers have shown that tandem gait is negatively impacted after concussion. Oldham et al.[250] examined tandem gait in collegiate athletes at baseline and within 48 hours after SRC. Collegiate athletes took 1.21 seconds longer to complete the tandem gait 48 hours after injury, compared to baseline.[250] Similarly, Howell and colleagues[252] reported that collegiate athletes performed 1.24 seconds worse at 72 hours post-injury compared to baseline. Furthermore, collegiate athletes took approximately 11.4

seconds to perform the tandem gait compared to uninjured controls who took approximately 9 seconds.[252] Unlike the BESS, deficits in tandem gait performance may be evident up to 2 months post-concussion.[253] Interestingly, these long-term deficits are increasingly evident with the incorporation of a dual-task, combining motor and cognitive tasks. It is postulated that utilizing a dual task paradigm mimics activities of daily living.[254]

Howell and collegaues[254] prospectively examined the effect of concussion on gait balance control in single- and dual-task conditions. The results of study suggest that concussed adolescents demonstrated decreased ability to maintain forward momentum and decreased balance control maintenance compared to healthy control adolescents.[254] In addition, concussed adolescents demonstrated higher medial/lateral center of mass velocity and displacement in the dual-task condition compared to healthy control athletes up to 2 months after injury.[254] Interestingly, Howell and colleagues reported that even after return to activity, athletes with concussion demonstrated significantly increased total center of mass medial/lateral displacement and peak velocity, suggesting that after returning to play, athletes regress in their recovery of gait balance control.[255] However, it should be noted that age could be a factor in long term gait balance control deficits. Howell et al.[253] reported that adolescent athletes with concussion demonstrated greater total center of mass medial-lateral displacement compared to adolescent healthy controls; however, concussed young adults did not differ from their controls. Utilizing tandem gait, especially with a dual-task component, within the multifaceted paradigm could provide clinicians with a more sensitive assessment for identifying postural control impairments in athletes with concussion.

Sport Related Concussion Recovery

Understanding time to recovery following SRC has been the interest of many researchers; however, it is still disputed in the current literature. Establishing SRC recovery time is difficult due to there being no gold standard management measure. Recovery time is defined as return to normal activities, including school, work, and sport.[1] Athletes are said to be recovered from concussion when post-concussion symptoms have resolved and neurocognitive and balance performance returns to baseline or pre-injury level. It was previously believed that the majority of athletes recover from concussion in approximately 7 – 10 days.[1,47,256] However, more recent research suggests that the majority of athletes recover in 10 – 14 days.[1,257] Although a majority of athletes recover within three weeks, about 20% of athletes with SRC experience protracted recovery (i.e., > 21 days).[8] The variability of recovery time between athletes may be due in part to pre-existing risk factors or post-injury clinical factors, that may predispose athletes to longer recovery times. Female sex, younger age, personal or family history of migraine, and mental health disorders have been identified as possible risk factors for prolonged recovery.[258] In addition, post-injury clinical factors such as continuing to play with SRC, initial symptom burden, and post-traumatic migraine have been identified to influence recovery time.[258] However, it is possible that longer recovery times are a reflection of the advances made in concussion management.[1] Furthermore, it is possible that studies are limited by selection bias, specifically several of these studies include athletes recruited from large concussion clinics, which may include athletes with longer recovery times.[1] Identifying factors that

negatively influence recovery time may help researchers and clinicians develop individualized treatment interventions.

Sport Related Concussion Treatment

Early consensus statements described the cornerstone of concussion management as strict physical and cognitive rest. Given the physiological events that occur during the neurometabolic cascade, concussed athletes are at an increased vulnerability for catastrophic injury or prolonged recovery. Therefore, if concussed athletes abstained from physical activity until symptom resolution, the risk for further injury might be mitigated. This management strategy, commonly known as “cocoon therapy”, restricted athletes to days in a darkened room with no sports or physical activity, no social interaction, and no screens.[259,260] However, this approach is more extreme than that prescribed to patients with traumatic brain injury (TBI).[261] Currently, no evidence exists to suggest that strict physical and cognitive rest until asymptomatic is rehabilitative.[262,263]

First, it is possible that strict, prolonged rest could lead to the development of anxiety,[264] depression-like symptoms,[265–267] or physical deconditioning.[268,269] In addition, it is possible that protracted rest may worsen outcomes and hinder recovery.[270] Thomas and colleagues[270] examined the effectiveness of recommending 5 days of strict rest compared with 24 to 48 hours of rest in athletes with concussion. Concussed athletes randomly assigned to 5 days of strict rest reported more daily post-concussive symptoms and took longer for symptoms to resolve compared to concussed athletes that were assigned 24 to 48 hours rest.[270] Another limitation of prescribing strict rest is the operational definition of rest. Operational definitions of physical and cognitive rest

are poorly defined and are defined differently across investigations.[1,270–273] Finally, the amount and duration of rest has not been empirically established.[1,274] The current consensus statement recommends that concussed athletes should practice strict rest until asymptomatic[1]; however, concussion symptoms are non-specific and it is not uncommon for healthy, non-concussed athletes to report concussion symptoms.[275–277] Therefore, it is possible that concussed athletes may never reach the asymptomatic phase. It should be noted that concussion is a treatable injury.[205] There is substantial data to suggest that exercise, specifically aerobic exercise, is an effective treatment for concussion.[278–287]

One possible active intervention that involves identifying a subsymptom threshold is the Buffalo Concussion Treadmill Test (BCTT).[288] The BCTT is an incremental treadmill test modified from the Balke protocol.[288] In addition, researchers have also developed a protocol using a bicycle ergometer for participants with orthopedic injuries and significant balance impairments.[289] The BCTT protocol starts with the participant walking on a treadmill set at 3.6 mph at a 0% incline for 1 minute.[288] After each minute, the incline is increased by 1% at the same speed.[288] During every minute of the test, researchers assess ratings of perceived exertion (RPE) and symptoms, while every 2 minutes, heart rate and blood pressure are recorded.[288] The exercise test is terminated when the participant reports an exacerbation of post-concussion symptoms, defined as three or more points compared to the participant's pre-exercise test symptom score.[288] After identifying the intensity of aerobic exercise that exacerbated post-concussion symptoms, concussed athletes are prescribed 20 minutes per day of aerobic exercise at

an intensity of 80% - 90% of the subsymptom threshold heart rate, once per day for 6 – 7 days of the week.[288,290]

In order to implement a treadmill exercise protocol as a treatment option for concussion, it is important to ensure that the protocol is safe and meets minimum standards for reliability. Leddy and colleagues[286] evaluated the safety and effectiveness of aerobic exercise training after concussion. Specifically, the participants in this study had been suffering from symptoms at rest for at least 6 weeks after injury.[286] Importantly, following the implementation of the exercise protocol, there were no instances of adverse events and no occasions where a participant could not exercise the following day due to symptom exacerbation.[286] In addition, when compared to baseline, participants' symptomology improved significantly and were able to meet consensus recommendations for return to play.[1,286] In addition, the BCTT demonstrates a high degree of interrater reliability and test-retest reliability.[284] Results of Leddy et al.[284] revealed good test re-test reliability for maximum heart rate (ICC = 0.79), but low for RPE (ICC = 0.42). In addition, the researchers achieved a sensitivity of 99% for identifying actors with symptoms and a specificity of 89% for ruling out concussion symptoms.[284] Therefore, results of the studies suggest that the modified Balke protocol is a safe and reliable, objective measure that may help clinicians make return to play decisions after concussion.[284,286]

Following preliminary studies,[284,286] researchers examined the effectiveness of aerobic exercise training compared to other forms of exercise in concussed athletes with prolonged symptomology. Kurowski and colleagues[283] randomly assigned concussed adolescent athletes with protracted symptomology to either a sub-symptom

exacerbation aerobic training protocol or full body stretching program. Concussed athletes in the aerobic training group demonstrated improved symptoms compared to the fully body stretching group.[283] However, Maerlender et al.[291] reported that an exertion protocol did not demonstrate a significant positive affect on recovery time. In fact, concussed athletes assigned to the exercise protocol demonstrated longer recovery time compared to the group prescribed standard practice (i.e., no systematic exertion beyond normal activities).[291] Interestingly, studies[283,286,291] investigating the effectiveness of aerobic exercise to treat prolonged symptoms yield mixed results. Researchers have also examined the effectiveness of early intervention as a treatment strategy for concussion.

Howell and colleagues[292] examined the association between exercise within one week post-concussion with symptom severity and time to symptom resolution. Interestingly, the group of concussed athletes who engaged in exercise post-injury did not demonstrate quicker symptom resolution compared to the group of athletes who did not engage in exercise post-injury.[292] The group that exercised reported lower symptom severity compared to the group who did not exercise.[292] In addition, Sufrinko and colleagues[287] reported that more physical activity was associated with worse outcomes. Specifically, physical activity during the first two weeks of injury was associated with worse vestibular and ocular outcomes and high and low levels of physical activity were associated with poor visual motor speed.[287] However, other researchers suggest[282,285,293,294] aerobic exercise may be beneficial for treating concussion.

Willer and colleagues[293] assigned concussed adolescents to either rest, aerobic exercise, or placebo stretching groups within 10 days of injury. The rest group

recovered fully in 16 days, while the exercise group recovered in 13 days, and the placebo stretching group recovered in 17 days. The results of this study suggest that early engagement in aerobic exercise may be an effective treatment strategy for concussion. Similarly, Lawrence and colleaues[294] wanted to examine whether earlier engagement in physical activity was associated with time to return to play and school. The results of the study revealed that earlier engagement in aerobic exercise was associated with faster return to sport and school.[294] In another study, Grool and colleagues[282] reported that children and adolescents who engage in early physical activity have a lower risk of developing persistent post-concussive symptoms. It should be noted, however several studies[282,292–294] are limited by observational and quasi-experimental designs. In one of the only randomized control trials, Leddy and colleagues[285] assessed the effectiveness of a subsymptom threshold aerobic exercise program in adolescents during the acute phase of concussion recovery. Concussion athletes were randomly assigned to either an aerobic exercise group or a stretching group. Similar to previous research, the concussed athletes prescribed aerobic exercise recovered in a median 13 days, whereas the stretching group athletes recovered in 17 days.[285] The results of this study, further suggest that aerobic exercise may be an effective strategy to treat concussion.[285] It should be noted that several studies have only utilized exercise protocols[282,283,285–287,291–294] rather than developing a multifaceted intervention that consists of aerobic exercise and other potentially advantageous interventions like education or psychosocial strategies (e.g., imagery).

Gagnon et al.[279] developed the active rehabilitation intervention (ARI) that consists of four facets: aerobic exercise, coordination/skill practice, visualization, and

education. The results of the study suggest that implementing an active rehabilitation program may promote recovery in children who were slow to recover after concussion.[279] In a subsequent case series researchers examined the effectiveness of ARI in adolescents who were slow to recover from concussion.[280] Following the implementation of the ARI program, concussed adolescents reported significantly decreased post-concussion symptoms.[280] In addition, concussed adolescents reported decreased fatigue symptoms and improved mood with the intervention.[280] Similarly, Dobney and colleagues observed significant decreases in post-concussion symptom in athletes with prolonged recovery after the implementation of the ARI intervention. Finally, Gauvin-Lepage et al.[281] compared the recovery time between youth athletes who received the ARI intervention and youth athletes who received standard rest-based/symptom limited activity recommendations. Both groups reported decreased symptoms over the span of the study, but the athletes assigned to the ARI group reported higher levels of quality of life and less anger than those in the standard care group.[281] The results of these studies[278–281] suggest that the utilization of a multifaceted treatment intervention may be a beneficial treatment option for athletes with concussion that have a protracted recovery trajectory.

Although preliminary research suggests that exercise is a safe, reliable, effective treatment option for athletes after concussion more research is warranted. Specifically, future research should continue to determine the optimal type, timing, and intensity of exercise-based treatment programs. Second, future research should investigate the mechanisms responsible for the advantageous effect of exercise-based treatments on concussion recovery. Third, researchers should investigate the effect of common

concussion risk factors (e.g., female sex, age, history of concussion) on exercise-based interventions. Finally, the majority of the previous studies[278–286] have only investigated the effect of aerobic exercise on symptomology. Given that concussion affects a multitude of domains[1] (i.e., neurocognitive, vestibular and ocular, balance), future researchers should examine how exercise-based interventions promote recovery in these domains.

Baseline Assessment

Current SRC consensus statements[1,4] advocate for the use of a prospective assessment approach, which involves baseline (i.e., pre-season) assessment and serial administration post-injury. In addition, consensus statements recommend, but do not require, pre-season baseline neurocognitive testing.[1,4] Implementing a prospective assessment approach provides the clinician with several advantages. First, with baseline and post-injury scores clinicians are able to compare post-injury neurocognitive performance to baseline neurocognitive performance to aid in SRC diagnosis and return to play decisions. Second, obtaining baseline scores allows for injured athletes to serve as their own control when comparing to post-injury scores. Finally, baseline testing may be used as an opportunity to educate athletes about the significance of SRC.[1] More than 90% of ATs utilize a follow-up assessment approach, with approximately 70% of ATs utilizing CNT in follow-up testing.[9] In the absence of a baseline assessment, normative neurocognitive data for age and sex are available for post-concussion comparison.

Previous research suggests that baseline measures may provide greater clinical utility than normative baseline values. Previous research suggest that baseline values

provide better diagnostic accuracy compared to normative values. Louey and colleagues[10] compared the sensitivity and specificity of baseline and normative methods neurocognitive scores in athletes who had sustained an SRC. Interestingly, the sensitivity and specificity of the baseline method was 96.6 and 86.9, respectively, whereas the sensitivity and specificity of the normative method was 69.0 and 91.5, respectively. Therefore, the results of this study suggest that the baseline method is more sensitive than the normative method. Similarly, Hinton-Bayre et al.[11] reported that the baseline method was more sensitive compared to the normative method. In addition, previous research suggests that baseline data provides a lower false-positive rate compared to normative data. Roebuck-Spencer and colleagues[12] examined the added value of baseline cognitive data when compared to population normative data. Interestingly, 65.7% of military personnel who would have been categorized as impaired using normative reference values showed no change in baseline cognitive performance.[12] Therefore, there is a possibility of a high false positive rate when utilizing norm referenced values. However, Haran and colleagues[295] suggest that there are no clear advantages for using baseline approach over normative approach. Given the utility of baseline neurocognitive assessment,[10–12] it is essential that neurocognitive scores obtained during pre-season testing are an accurate representation of the individual's current cognitive status.

Factors that Influence Baseline Computerized Neurocognitive Performance and Total Symptoms

While implementing baseline CNT is a valuable tool for assessing and managing SRC, it is important to identify factors that may negatively influence baseline CNT

performance and symptoms. Sex[13–17], concussion history[16,296–298], race[299,300], motivation/effort[18–20], ADHD and LD[21–23], and physical exertion[24] have all been identified as factors that negatively affect baseline CNT performance and symptoms.

Previous research[13–17] suggests that males and females differ on neurocognitive performance and symptoms at baseline, however the results are mixed for which specific cognitive domains are affected. Covassin et al.[17] was one of the first groups to examine sex differences in baseline neurocognitive performance and symptoms. The results revealed that female athletes performed significantly better on verbal memory than male athletes.[17] This finding is supported by other studies that female athletes outperform male athletes on baseline verbal memory.[13–16,301] In addition, other studies suggest female athletes also perform better than males on visual motor processing speed and reaction time.[13–16,23] However, French et al.[13] reported that male athletes perform better on visual motor processing speed. In addition, Covassin et al. reported that male athletes perform better on visual memory.[17] It is hypothesized that females perform better on verbal memory tasks due to increased estrogen levels compared to males.[17] Regarding baseline symptoms, the majority of studies[13–15,23] reported that sex does not affect total symptoms at baseline; however, Covassin et al.[17] found that female athletes report more symptoms compared to male athletes.

Furthermore, previous concussion history may interact with sex to influence baseline neurocognitive performance. Female athletes with a history of 2 or 3 more concussions concussion perform better on verbal memory than male athletes with 2 or 3 more concussions.[16] Strictly examining concussion history as a factor, the majority of previous research[296,302–307] suggests that previous history of concussion has no effect

on neurocognitive performance. It is possible that the results of these studies do not reveal neurocognitive deficits for several reasons. First, it is possible that cognitive deficits that exist following SRC recovery could be so subtle that detection is not captured using CNT batteries.[308] Second, many of these studies[302–307] have included adolescent and young adult athletes, exclusively.[308] It is possible that long-term cognitive changes may be not evident in adolescent and young adult athletes.[308] Finally, it is possible that there is a learning effect, given that athletes with a history of concussion have taken the neurocognitive tests multiple times over the course of their recoveries.[308]

Covassin and colleagues[16] examined the effect of concussion history on neurocognitive performance in male and female athletes. The results of the study revealed that athletes with no previous history of concussion performed better on baseline neurocognitive testing than athletes with a previous history of 2 or 3 or more concussions.[16] Specifically, athletes with a history of concussion demonstrated worse performance on verbal memory and visual motor processing speed.[297,298] With regards to symptom reporting, Brooks et al.[296] observed those with a previous history of 2 or more concussion reported significantly more symptoms than those without a history.

Although earlier research[309] suggested that race is not a factor that influences baseline CNT performance and symptoms, more recent research[299,300] suggests that it could be. Kontos and colleagues[309] observed no differences in baseline neurocognitive performance or total symptoms between Black and White high school and collegiate athletes. Wallace et al.[299] examined differences between Black and White students on baseline neurocognitive performance and symptoms. The results of the study suggest

that White and Black athletes differ significantly on visual motor processing speed and reaction time, with Black athletes demonstrating worse outcomes.[299] In addition, Black athletes report more symptoms compared to White athletes.[299] Similarly, Houck and colleagues utilized linear regression models to identify predictors of baseline neurocognitive functioning in collegiate athletes. Black race predicted worse neurocognitive performance in collegiate athletes. These results are supported by previous research that suggests that Black individuals score worse than White individuals on measures of language, attention, processing speed, constructional skill, and select executive skills.[310] It is possible that differences in neurocognitive performance can be attributed to educational background, reading/literacy level, and socioeconomic status.[299] These findings[299,300] support pre-season testing, as normative data may not be appropriate to compare various races and ethnicities due to cultural differences.

In addition to sex, concussion history, and race another pre-existing factor that could influence baseline neurocognitive performance and symptoms is ADHD and/or LD. Zuckerman and colleagues[22] documented that athletes with ADHD demonstrated significantly lower verbal memory, visual memory, visual motor processing speed, in addition to significantly higher reaction time and total symptoms. Similarly, Elbin et al.[21] reported that high school and collegiate athletes with ADHD performed significantly worse on verbal memory, visual memory, visual motor processing speed, and reaction time and report significantly more symptoms at baseline than athletes without ADHD. Other studies show mixed results on which specific cognitive domain is negatively influenced by ADHD. Two studies[311,312] suggest verbal memory and visual motor speed

are the only composite scores negatively influenced by ADHD, whereas Vaughn et al.[313] showed that visual memory and visual motor processing speed, reaction time, and total symptoms may be most affected. Contrary to the majority of studies, Manderino et al.[314] reported no differences in baseline neurocognitive performance and symptoms in collegiate athletes with and without ADHD. Interestingly, it is possible that the combined effects of ADHD and LD should also be considered when interpreting baseline neurocognitive scores. Elbin et al.[21] observed significant differences in verbal memory, visual memory, visual motor processing speed, reaction time, and total symptoms between athletes with combined ADHD/LD and athletes without ADHD/LD, with athletes with ADHD/LD demonstrating worse cognitive performance and higher total symptoms. However, Zuckerman and colleagues[22] reported that athletes with combined ADHD/LD had worse performance on visual motor processing speed, slower reaction time, and higher reported symptoms, but no other differences were observed. Caution should be taken when interpreting the results of these studies. Specifically, the investigators[15,21,22,300,311,312,314] relied on self-reported ADHD/LD diagnosis and did not require additional documentation to corroborate diagnosis.

In addition, to sex, concussion history, race, and ADHD/LD, motivation or effort could influence baseline neurocognitive performance and symptoms. Interestingly, athletes may intentionally try to under-perform or “sand-bag” the baseline CNT testing in the hope that post-concussion comparisons will be more favorable and will aid with quicker return to play. Bailey and colleagues[19] have observed athletes classified as “low motivation” athletes showed greater improvements than highly motivated athletes. Similarly, Hunt et al. found that highly motivated athletes performed significantly better

on baseline neurocognitive performance and symptoms compared to low motivated athletes.[18] However, more recent research suggests baseline motivation does not explain a significant amount of the variance in neurocognitive performance.[315] Traditionally, invalid baseline scores are identified by the presence of outliers, however with the introduction of CNT batteries, these tests can now "flag" baseline scores that are below pre-determined validity indicators.[26] Erdal and colleagues[316] examined how likely it was that collegiate athletes could sandbag the baseline test without being flagged by the validity indicators of ImPACT. Interestingly, 89% of collegiate athletes that attempted to sandbag their baseline performance were identified by the indices on the ImPACT battery.[316] In a separate study, ImPACT validity indicators were able to detect 75% of athletes who were instructed to perform poorly on the test.[317] Even when athletes were coached on how to perform poorly on the test in a way to not get detected, the internal indices were able to detect 65% of athletes.[317] Results of these studies[316,317] suggest that CNT batteries are able to detect poor effort by using internal indices.

Sleep

Sleep is composed of 2 states: rapid eye movement (REM) and non-REM (NREM) sleep, which can be detected using electroencephalography (EEG) and electromyography (EMG). Specifically, REM sleep is characterized by rapid eye movement, skeletal muscle atonia, and heightened physiologic activity (e.g., increased cerebral blood flow, fluctuations in heart rate and blood pressure).[318] Non-REM sleep occurs after sleep onset and can be further broken down into three stages. Stage 1 NREM sleep is characterized as relatively light sleep.[319] In addition, heart rate,

breathing and eye movements slow and muscles begin to relax.[319] Stage 2 NREM sleep is described as the period of light sleep before entering into deep sleep.[319] Heart rate and breathing continues to slow, muscles relax further, body temperature decreases, and eye movements stop.[319] Finally, Stage 3 is characterized as a period of deep sleep that is necessary to feel refreshed in the morning.[319] During this stage, heart rate and breathing slow to the lowest levels.[319] A series of NREM stages are followed by a period of REM sleep. This cycle of NREM and REM sleep takes approximately 1.5 hours, with approximately 4 – 5 cycles of REM sleep and NREM sleep occurring each night for an individual who sleeps approximately 8 hours.[319]

Sleep is regulated by two main mechanisms: the homeostatic sleep drive and the circadian rhythm.[320] The homeostatic sleep drive increases the need for sleep as the time of wakefulness increases. In other words, the sleep drive gets stronger for every hour awake and causes longer and more deep sleep after periods of sleep deprivation. Circadian rhythm, or biological clock, is responsible for the maintenance of a 24-hour sleep/wake cycle, which is regulated by exposure to ambient levels of blue light and the secretion of melatonin at night by the pineal gland.[321] Circadian rhythm causes sleepiness at night and wakefulness to occur in the daylight, typically the morning.

Currently, polysomnography (PSG) is the gold standard tool to measure sleep.[322] Typically when employing PSG, individuals spend the night in a sleep laboratory under controlled conditions.[322] Polysomnography utilizes several surface electrodes that measure physiological parameters of sleep (EEG, eye movements, muscle activity, heart physiology, and respiratory function).[322] One distinct feature of sleep is a state of immobility, relative to wakefulness. Taking advantage of this, researchers have

attempted to categorize sleep or wakefulness by measuring wrist movements.[322] Wrist actigraphy is a measure of wrist movement utilizing an accelerometer in a wrist worn device. Wrist actigraphy provides an objective, unobtrusive measure of recording sleep that is valid and has the advantage of continuously measuring sleep in the individual's home environment, improving the generalizability.[322,323] However, there are limitations to measuring sleep via actigraphy. Specifically, actigraphy may overestimate sleep time because immobility marks the beginning of sleep for actigraphy, whereas immobility and changes in brain electrical activity marks the beginning of sleep for PSG.[323] Importantly, these changes in brain electrical activity can occur after wrist immobility.

Sleep is essential for maintaining health and well-being. Insufficient sleep duration is reported to have negative consequences on several aspects of health (e.g., metabolism, mood)[29,30] and may increase risk for several chronic diseases (cardiovascular disease, obesity).[31] Despite evidence that shows that sleep is essential for humans, the exact function of sleep remains unclear. Several theories exist that attempt the explain why sleep is necessary. First, sleep is hypothesized to be physically restorative and may serve as a period for growth and repair as evidenced by the release of anabolic hormones, such as growth hormone.[324] Second, it is thought that sleep conserves energy.[325] However, the amount of energy conserved during sleep is relatively small. In addition, REM sleep is characterized by increased brain activity, metabolism, and energy use, which challenges this theory, although not much measurable energy contributes to brain activity. Third, it is hypothesized sleep is a product of evolution as a safety strategy.[326] Finally, it is hypothesized that sleep is needed for brain plasticity and essential for learning and memory.

Sleep and Neurocognition

The importance of sleep is evidenced by the consequences of sleep deprivation. Despite the evidence that sleep is essential for health and well-being, 35% of adults sleep less than the recommended 7 – 9 hours a night.[27,28] Although the effects of chronic total sleep deprivation on neurocognitive performance are well documented throughout the literature,[327] the effects of partial sleep deprivation (i.e., less than recommended hours of sleep per day) are not as well understood. Total sleep deprivation rarely occurs outside of highly controlled sleep laboratories and certain professions, such as medical personnel.[327] However, partial sleep deprivation is more pervasive in adults, adolescents and children.[35,328] Sleep deprivation negatively influences several domains of neurocognition including attention, information processing speed, reaction time, working memory, learning and immediate memory recall.[329] The majority of studies examining the effects of sleep deprivation have focused on cognitive performance, motor performance, and mood.[330]

Pilcher et al.[330] conducted a meta-analysis on the effects of sleep deprivation on functioning. Interestingly, the results of the meta-analysis suggest that the effects of sleep deprivation on neurocognitive functioning depend on the type of sleep deprivation and the type of neurocognitive measure. Previous research suggests that partial sleep deprivation (< 7 hours every 24 hours) results in worse neurocognitive outcomes compared to short-term total sleep (≤ 45 continuous hours) and long-term total sleep deprivation (> 45 continuous hours).[330] In addition, sleep deprivation has a greater effect on cognitive tasks, and especially mood, and least effect on motor tasks.[330]

Anderson et al.[331] examined the effect of sleepiness on distractibility. Young adults were assigned normal sleep or sleep restriction (less than 5 hours of sleep) and administered a monotonous task. Those assigned with sleep restriction demonstrated higher rates of distractibility.[331] In addition, Dinges and colleagues[332] investigated the effect of restricting sleep to approximately 5 hours per night for 7 consecutive nights in young adults. Results of the study suggest that young adults exhibited significant decrements in alertness, and especially in measures of sleepiness, fatigue, and psychomotor vigilance.[332] In addition to the neurocognitive implications, the results of these studies[331,333,334] have significant implications for driving safety, given sleep deprivation is a major contributor to motor vehicle accidents.[335]

In addition to alertness and distractibility, partial sleep deprivation also negatively influences accuracy. Edwards et al[336] utilized dart throwing in order to mimic everyday activities. Young adults were restricted to 3 – 4 hours of sleep, then participated in dart throwing which were assessed for accuracy by measuring the mean distance of the dart from the bullseye, number of times the target was missed, and the variability of the scores from the darts thrown.[336] The results suggest that sleep deprivation negatively influenced the accuracy and variability of the darts hitting the target.[336]

Previous research suggests that sleep plays an important role in learning and memory. Following a meta-analysis, Lowe and colleagues[327] reported that restricted sleep impaired measures of working memory and long-term memory. Working memory refers to the system(s) that provide temporary storage and manipulation of information needed for complex cognitive tasks such as comprehension, learning, and reasoning.[337] Lo and colleagues[338] investigated the effect of partial sleep restriction (5 hours of sleep

for 7 nights) on cognitive performance in adolescents. The sleep restriction group demonstrated decreased working memory and executive function compared to the control group. Casement and colleagues[339] assigned young adults to 4 hours or 8 hours of sleep for 12 days. After the 12 day sleep restriction period, the sleep restricted group was instructed to sleep for 8 hours for 9 days.[339] Even when given time to make up for lost sleep, the sleep restricted group did not improve on measures of working memory.[339] Just as obtaining recommended amounts of sleep is important, sleep prior to learning may also be necessary. Stickgold and colleagues[340] administered an episodic memory encoding task to participants and conducted a recognition test 48 hours later. After 35 hours of sleep deprivation, participants in the sleep deprived group demonstrated worse memory performance compared to the non-sleep deprived group.[340] Overall, sleep deprivation has substantial consequences on neurocognition. This is particularly important for college-aged individuals, and particularly college students, who will sometimes pull “all-nighters” to improve their chances for academic success.[341]

Sleep and College-Aged Individuals

The ages of 18 – 25 years is marked by considerable change and importance. During this time, young people are exploring world views and experiencing changes in education, work, and love.[342] For young people who attend college, the transition from high school is characterized by a shift in personal responsibilities, decreased institutional support, and changes in social enviornments.[343–345] In this time of transition to adulthood, young people experience poor sleep. Poor sleep is particularly prevalent in college students. In a large epidemiological study, 25% of collegiate students

reported getting less than 6.5 hours of sleep and only 29.4% of college students reported getting more than 8 hours of sleep per night.[35]

Sleep hygiene is characterized as behaviors that are required for normal, quality nighttime sleep and full daytime alertness. These behaviors include maintaining regular sleep-wake times, limiting alcohol consumption, caffeine, use of nicotine prior to bedtime, and establishing a sleep environment conducive to sleep.[341] Often times, college students do not practice good sleep hygiene behaviors. College students sacrifice sleep on the weekdays in order to study and socialize with friends, then sleep long hours on weekends in an attempt to make up for the missed sleep. In addition, college students may consume caffeine, alcohol, or drugs, like stimulants.[346] Furthermore, student housing may not be the best environment for restorative sleep.[341] The consequences of poor sleep in college students are documented throughout the literature. College students who report poor sleep demonstrate worse performance on academic tests.[347] Furthermore, college students that reported short sleep had lower overall grade point averages than college students who reported longer sleep.[348] Gilbert and colleagues[349] examined the relationship between sleep quality and academic performance. College students who reported poor sleep behaviors demonstrated worse concentration, missed more classes, which resulted in lower grade point averages, and had more incompletes and withdrawals from classes.[349]

For collegiate athletes, sleep is especially important given the cognitive, physiological, and physical demands needed for optimal athletic performance and recovery. In addition to academic and athletic responsibilities, collegiate athlete's schedules are variable due to travel, game and practice schedules, and team meetings

that could influence wake and bed times.[346] However, 39% of collegiate athletes report sleeping less than 7 hours on weekdays.[350] Similar to the non-collegiate athlete student population, collegiate student athletes demonstrate poor sleep hygiene habits. In order to address the pervasive sleep problems in collegiate athletes, the NCAA assembled the Interassociation Task Force on Sleep and Wellness, consisting of sleep experts, coaches, collegiate athletes, and athletic administrators. Following an extensive literature review, The Task Force recommended that sleep screening be incorporated in pre-participation exams and collegiate athletes and coaches are provided with evidence-based sleep education that includes information on 1) sleep best practices; 2) the role of sleep in optimizing athletic and academic performance and overall well-being; and 3) strategies to optimize collegiate athlete sleep.[346] Evidence suggests that poor sleep negatively influences academic performance in college students and negatively influences cognitive processes, including memory and attention.[341] Not only does poor sleep have academic implications, but also could also influence performance on pre-participation evaluation. Specifically, all NCAA collegiate athletes must complete pre-season concussion testing prior to the start of the athletic season. Several of these baseline concussion assessments measure cognition. Therefore, it is possible that poor sleep quality could negatively influence baseline concussion assessments.

Sleep and Baseline Computerized Neurocognitive Performance and Symptoms

In addition to the aforementioned factors that negatively influence baseline CNT performance and symptom reporting (e.g., sex, concussion history, etc.), it is important to consider sleep duration as an extraneous factor that may negatively influence baseline CNT performance and total symptoms. McAllister and colleagues[351] compared

baseline neurocognitive performance and symptom reporting in collegiate athletes with and without a history of diagnosed sleep disorders. The results of the study revealed that collegiate athletes with a history of diagnosed sleep disorders reported higher symptom severity than athletes without a history of diagnosed sleep disorders.[351] Previous researchers[37,39,40,43] have also examined the effect of self-reported sleep duration on neurocognitive performance and total symptoms at baseline.

Mihalik and colleagues[39] examined the effect of sleep quality and sleep quantity on commonly used concussion assessments in collegiate athletes. Utilizing the Pittsburgh Sleep Quality Index, Mihalik et al.[39] divided athletes into greatest, moderate, and least sleep quantity and categorized into high sleep quality and low sleep quality. The results showed that collegiate athletes with short sleep duration reported increased symptomology; however, no other significant effects for sleep quality were observed.[39] Similarly, Moran and colleagues[40] examined the effects of a prior night's sleep quantity on neurocognitive performance and symptoms in high school athletes.[40] High school athletes were categorized into two groups: < 8 hours of sleep and ≥ 8 hours of sleep. High school athletes that reported less than 8 hours of sleep the night prior to baseline testing reported greater symptomology.[40] Moran et al.[40] observed that high school athletes that slept less than 8 hours the night before baseline testing had greater total symptom scores. In contrast, Sufrinko and colleagues[37] observed worse neurocognitive performance, specifically worse verbal memory, visual memory, visual motor speed, and reaction time in high school athletes that reported less than five hours of sleep the night before baseline testing. In one of the only studies to utilize random assignment, Stocker and colleagues[43] randomly assigned adults to three groups: normal, sleep

restriction (50% habitual sleep), or total sleep deprivation utilizing polysomnography monitoring in a sleep laboratory. Interestingly, sleep deprivation was associated with decreased visual memory, reaction time, and visual motor speed.[43] Although the results of these studies[37,39,40] suggest that lower sleep duration influences performance on computerized concussion assessment, especially symptomology, caution should be taken when interpreting these results. Several gaps exist due to methodological issues and limited evidence.

Many studies[37,39,40,351] examining the effect of sleep on neurocognitive performance and total symptoms have relied on subjective measures of sleep quantity. For example, one study[39] utilized the Pittsburgh Sleep Quality Index and three studies[37,40,351] used a single-item question – "How many hours of sleep did you get last night?" – that is included on the ImPACT demographics section. It is possible that recall bias and inaccuracies of sleep duration could be introduced due to the subjectiveness of the assessment. Utilizing an objective measure of sleep, for example via Actigraphy, could reduce the bias and provide more accurate information about sleep duration and quality. Furthermore, previous studies[37,39,40], have only examined the effects of a single night of sleep. It is unknown whether a single night of sleep is representative of a typical or unusual night of sleep. Therefore, there is a need to investigate the effects of multiple nights (i.e., habitual) of sleep on baseline CNT performance and total symptoms. Habitual sleep duration, or sleep accumulation across multiple nights, accounts for several factors including lifestyle choices, environment, and biological drive (i.e., circadian rhythm).[42] Therefore, extending the scope to include multiple nights of sleep may provide a better representation of an individual's sleep habits. Currently, there is a

need to investigate the effects of habitual sleep duration on baseline CNT performance and total symptoms.

Conclusion

Sleep is essential for maintaining health and well-being. In addition, a growing body of literature suggests that short sleep duration negatively influences learning and memory, reaction time, and auditory vigilance. Specifically, for collegiate athletes, sleep is particularly important given the cognitive, physiological, and physical demands needed for optimal athletic performance. Current consensus statements advocate for baseline CNT assessment in order to aid in SRC diagnosis and return to play decisions. It is hypothesized that short sleep duration negatively influences CNT performance and symptoms. However, previous studies have relied on subjective measures of sleep quantity in the form of questionnaires (e.g., Pittsburgh Sleep Quality Index) or single-item questions (i.e., “How many hours of sleep did you get last night?”). Given the subjective nature of these measures, it is possible that recall bias and inaccuracies in sleep duration could be introduced by the athlete. Furthermore, previous studies have only investigated the effects of a single night of sleep and have failed to investigate the effects of habitual sleep on baseline CNT performance and total symptoms. It is unknown whether a single night of sleep is representative of a typical or unusual night of sleep, whereas habitual sleep duration considers multiple factors including lifestyle choices, environment, and biological drive. Therefore, extending the scope to include multiple nights of sleep may provide a better representation of an athlete’s sleep behaviors.

CHAPTER III: METHODOLOGY

Experimental Design

This study used a cross-sectional research design in order to investigate the clinical utility and influence of habitual, device-measured sleep duration on baseline neurocognitive performance and total concussion symptom severity in college-aged individuals. For Specific Aim 1 (To examine the influence of habitual, device-measured sleep duration on baseline computerized neurocognitive performance and total concussion symptom severity in college-aged individuals) the independent variable was habitual, device-measured sleep duration. The dependent variables were the 4 ImPACT neurocognitive composite scores (verbal memory, visual memory, visual motor speed, reaction time) and total concussion symptom severity. For Specific Aim 2 (To assess the agreement between both device-measured single-night and habitual, device-measured sleep duration and subjective, single-night sleep duration in college-aged individuals) the 3 variables examined were: device-measured single-night sleep duration, habitual, device-measured sleep duration, and subjective, single-night sleep duration. For Specific Aim 3, (To assess the agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration in college-aged individuals) the 2 variables examined were: habitual, device-measured sleep duration and habitual, subjective sleep duration.

This study was approved by the Michigan State University Institutional Review Board (IRB). In addition, it was also approved for reactivation to laboratory research by the IRB and Environmental Health and Safety.

Operational Definitions

Habitual, Device-Measured Sleep Duration

Habitual, device-measured sleep duration was calculated using the weighted average device-derived total nighttime sleep time (minutes) individuals experienced across at least 5 nights prior to ImPACT, using the Sadeh sleep scoring algorithm. The following formula (Equation 1)[352] was used to calculate the weighted average device-derived total nighttime sleep time, using the Sadeh sleep scoring algorithm: Equation 1: [5 * ((∑weekday)/nweekday)+2*((∑weekend)/nweekend)]/7.

Device-Measured Single-Night Sleep Duration

Device-measured single-night sleep duration was assessed by the total nighttime sleep time (minutes) individuals experienced the night prior to ImPACT administration as measured by the Actigraph GT9X Link Physical Activity Monitor, using the Sadeh sleep scoring algorithm.

Subjective, Single-Night Sleep Duration

Subjective, single-night sleep duration was assessed using the self-reported total nighttime sleep time (minutes) individuals experienced the night prior to ImPACT administration as measured by the National Sleep Foundation Sleep Diary.

Habitual, Subjective Sleep Duration

Habitual, subjective sleep duration was assessed by calculating the weighted average total nighttime sleep time (minutes) individuals experienced across at least 5 nights prior to ImPACT administration as measured by the National Sleep Foundation Sleep Diary.

Population and Sampling

Participants were recruited from a large Division I university in Michigan. The researcher arranged times to meet with healthy college-aged individuals currently enrolled in university classes at the time of recruitment. Researchers informed prospective participants on the purpose, procedures, risks, and benefits of participating in the study and requested any interested individuals to contact the researcher. Interested individuals were formally invited to participate in the study and inclusion and exclusion criteria were evaluated. Informed consent was obtained from eligible participants who met inclusion and exclusion criteria.

Inclusion Criteria

In order to be included in the current study, participants had to be 18 – 25 years old and enrolled in Michigan State University classes at the time of recruitment.

Exclusion Criteria

Participants were excluded if they had a diagnosed concussion within the past 6 months, had a history of moderate or severe traumatic brain injury, or if they did not speak or read English. In addition, participants were excluded from data analysis if they produced an invalid ImPACT baseline score. For Specific Aim 1, participants with less than 5 nights of device-measured sleep scored data using the Sadeh sleep scoring algorithm were excluded from data analysis. For Specific Aim 2, participants with incomplete or missing device-measured sleep duration and subjective, single-night sleep duration and participants with less than 5 nights of device-measured sleep scored data using the Sadeh sleep scoring algorithm were excluded from data analysis. Finally, for Specific Aim 3, participants with less than 5 nights of device-measured sleep scored

data using the Sadeh sleep scoring algorithm and less than 5 nights of National Sleep Foundation-derived sleep duration were excluded from data analysis.

Sample Size Estimation

An a-priori power analysis (G*Power Version 3.1, Germany) was conducted to determine the number of participants needed for regression analysis, assuming medium effect size[353] $f^2 = 0.15$, $\alpha = .05$, and statistical power = .80. Results from the power analysis revealed that a total of 55 participants were needed to reach acceptable statistical power.

Instrumentation

Demographics Form

The Demographic Form was created by the researcher that consisted of inclusion and exclusion criteria for the study. In addition, the Demographic Form asked about personal information (e.g., age, race, number of academic classes, household income) and previous medical history questions, including history of diagnosed concussion, headache/migraine disorders, learning disorder, ADD/ADHD, depression/anxiety, and sleep disturbance. See Appendix A.

Immediate Post Concussion Assessment and Cognitive Testing (ImPACT)

Neurocognitive performance and total concussion symptom severity were measured using the ImPACT battery. The ImPACT battery is a commonly used computerized neurocognitive testing assessment that includes three separate sections: demographics, the Post Concussion Symptom Scale (PCSS), and neurocognitive modules. The demographics section included personal (e.g., age, previous medical history) and sport participation (e.g., sport, position) history. Subjective, single-night

sleep duration was measured using the demographics section of ImPACT from the question “How many hours of sleep did you get last night?”. The PCSS is a 22-item self-reported symptom assessment rated on a 7-point Likert scale ranging from 0 (none) to 6 (severe). The PCSS symptoms can be categorized into four symptom factors: cognitive-sensory, sleep-arousal, vestibular-somatic, and affective.[153] The total concussion symptom severity score was calculated by summing the Likert scaled scores for the 22 symptoms, with higher scores indicating more symptoms (range 0 – 132). Four neurocognitive composite scores (verbal memory, visual memory, visual motor processing speed, and reaction time) are calculated from the neurocognitive modules. In order to control for learning effect, the ImPACT battery includes 5 different test versions. The ImPACT battery take approximately 25 minutes to complete. The ImPACT battery is able to distinguish between concussed and healthy individuals and has demonstrated acceptable test-retest reliability in collegiate athletes.[185,354]

Actigraph GT9X Link Physical Activity Monitor

Habitual, device-measured sleep duration and device-measured single-night sleep duration were measured using the Actigraph GT9X Link Physical Activity Monitor (Actigraph Corp), which is a tri-axial wrist-worn accelerometer that continuously measures sleep-related variables. Raw data were processed using the ActiLife software and were converted to sleep-wake data utilizing the Sadeh sleep scoring algorithm, which is age-appropriate for participants in the current study (18 – 25 years).[355,356] The Actigraph GT9X Link Physical Activity Monitor parameter examined in this study was total nighttime sleep time (minutes). Total nighttime sleep time was defined as the duration of nighttime sleep between the onset of sleep and the final awakening, using

the Tudor-Locke default for sleep period detection.[357] Participants were instructed to wear the Actigraph GT9X Link Physical Activity Monitor on their non-dominant wrists 24 hours a day, for 7 continuous days. Participants were instructed to take the monitor off only when doing water related activities (e.g., swimming, bathing). Non-wear time was identified via the ActiLife software and was removed for further analysis.[287,358] The Actigraph GT9X Link Physical Activity Monitor sleep parameters have been validated against the gold standard laboratory polysomnography (PSG).[355,359,360] Specifically, when compared to PSG, wrist actigraphy total sleep time demonstrated a high correlation of 0.97 in healthy adults.[361] Also, the Sadeh sleep scoring algorithm demonstrated sensitivity of 0.89 and specificity of 0.73.[359] In addition, the Actigraph GT9X Link has been used in previous research to estimate sleep timing and duration in healthy and concussed collegiate athletes.[358,362]

National Sleep Foundation Sleep Diary

The National Sleep Foundation Sleep Diary is a short sleep diary, separated into two sections: morning and evening. The National Sleep Foundation Sleep Diary asks individuals to report their wake times and bedtimes. In addition, the National Sleep Foundation Sleep Diary asks about physical activity, medication, napping, and mood in the evening section. Previous research showed moderate correlations between sleep diary and actigraphy total sleep time (r = 0.53).[363] See Appendix B.

Pittsburgh Sleep Quality Index (PSQI)

The PSQI was used in the current study to further describe the final sample. The PSQI[364] was used to assess retrospective sleep quality and quantity. The PSQI is a subjective sleep quality assessment that consists of 19 sleep-related questions relevant

to the participant's sleep habits during the previous month. The PSQI consists of 7 sleep composite scores: duration of sleep, sleep disturbance, sleep onset latency, daytime dysfunction, sleep efficiency, overall sleep quality, and use of sleeping medication. The 7 composite scores are used to calculate a total global score, which range from 0 – 21 with higher scores indicating poorer sleep quality. Total global sleep scores greater than 5 are indicative of poor sleep quality. The PSQI is a sensitive and specific measure that demonstrates acceptable internal consistency ranging from Cronbach α = .70 – 0.83.[365] The PSQI total score was poorly correlated with actigraphy (*r* = 13) total sleep time, but moderately correlated with sleep diary total sleep time (*r* = -0.31).[366] See Appendix C.

Morningness Eveningness Questionnaire – Short Assessment (MEQ-SA)

The MEQ-SA was used in the current study to further describe the final sample. The MEQ-SA[367] is comprised of sleep-related questions to determine and evaluate circadian rhythm typologies (i.e., morningness and eveningness). The questionnaire contains 19 questions that examine individual's wake and sleep habits, preferred times for physical activity and mental activity, and fatigue.[367] After completion of the questionnaire, the score can be calculated by adding the number of points of each question with scores ranging from 16 to 86. Scores on the MEQ-SA can be categorized into five typologies: definitely morning (70 – 86), moderately morning (59 – 69), intermediate (42 – 58), moderately evening (31 – 41), and definitely evening (16 – 30).[367] The reliability of the MEQ-SA is reported to be good with Cronbach's α ranging from 0.7 – 0.9.[368] See Appendix D.

Data Collection and Management

College-aged individuals were recruited from kinesiology and exercise classes. Following recruitment, interested participants met with the researcher in person to provide informed consent and were informed about the study procedures. During the Informational Session, participants were administered the demographics page, the morning section of the National Sleep Foundation Sleep Diary and were issued an Actigraph GT9X Link Physical Activity Monitor. Participants were instructed to wear the monitor on their non-dominant wrist for 24 hours a day, for 7 continuous days. Previous research suggests 7 nights is an acceptable period to obtain reliable sleep-wake patterns.[369] Participants were instructed to take the monitor off only when doing water-related activities (e.g., swimming, bathing, etc.).

Following the Informational Session, participants were sent the National Sleep Foundation Sleep Diary via text message with the Qualtrics link to complete the morning section when they wake up and evening section before bed. Participants completed the morning and evening sections of the National Sleep Foundation Sleep Diary for 7 days, for a total of 14 responses. After 7 days of Actigraph GT9X Link wear time, participants reported to the designated testing area (e.g., laboratories, classrooms) to complete the Testing Session. Participants were administered the ImPACT battery on a laboratory computer, with an external mouse,[370] and returned the Actigraph GT9X Link Physical Activity Monitor. The researcher explained the specific directions of the ImPACT demographics section per the manufacture guidelines and PCSS to the participant. Prior to the start of ImPACT, the researcher instructed the participant to perform to the best of their ability and were instructed to follow the written directions on the computer

screen before each neurocognitive section. Following the completion of the ImPACT test, participants exited out of the test and then were administered the MEQ-SA and the PSQI to complete via Qualtrics. Participants who wore the Actigraph GT9X monitor continuously throughout the study period and returned it received $25.00. In addition, participants who completed all sleep diaries surveys also received $25.00, equaling a total of $50.00 for complying with all study procedures.

Data Analysis

Descriptive statistics (means, standard deviations [SD], ranges, frequencies) were used to describe the total sample (e.g., age, sex, personal medical history, etc.), MEQ-SA and PSQI scores, National Sleep Foundation Sleep Diaries (morning and evening). All statistical analyses were conducted using the Statistical Package for the Social Sciences version 26.0 (SPSS: IBM).

Specific Aim 1: To investigate the influence of habitual, device-measured sleep duration on baseline neurocognitive performance and total concussion symptom severity in college-aged individuals.

Habitual, device-measured sleep duration was calculated using the weighted average device-derived total nighttime sleep time (minutes) individuals experienced across at least 5 nights prior to ImPACT, using the Sadeh sleep scoring algorithm. A weighted average was used to account for weekday and weekend total sleep time and because some participants wore the Actigraph GT9X Link on different days and some less than 7 days.[371,372] The following formula[352] was used to calculate the weighted average device-derived total nighttime sleep time, using the Sadeh sleep scoring algorithm (Equation 1). Total nighttime sleep time (minutes) was defined as the duration

of nighttime sleep between the onset of sleep and the final awakening. Gender,[13–17] previous concussion history,[16] history of diagnosed sleep disturbance,[351] and ADD/ADHD[21–23] were evaluated as covariates using a series of multivariate analysis of variance (MANOVA) tests due to their effect on ImPACT performance. Any demographic variables with significant differences in ImPACT composite scores or total concussion symptom severity were used as covariates to determine the influence of habitual, device-measured sleep duration on baseline computerized neurocognitive performance and total concussion symptom severity. Statistical significance was set at $p \leq .05$.

Sample mean and standard deviation for habitual, device-measured total sleep time (minutes) were calculated. Separate hierarchical linear regressions were used to investigate the influence of habitual, device-measured sleep duration on baseline neurocognitive performance and total concussion symptom severity in college-aged individuals. The independent variable was habitual, device-measured sleep duration, using the Sadeh sleep scoring algorithm, and the dependent variables were the four ImPACT composite scores (verbal memory, visual memory, visual motor processing speed, and reaction time) and total concussion symptom severity. Statistical significance was set at $p \leq .05$.

Specific Aim 2: To assess the agreement between both device-measured single-night and habitual, device-measured sleep duration and subjective, single-night sleep duration in college-aged individuals.

Device-measured single-night sleep duration was assessed by the total nighttime sleep time (minutes) individuals experienced the night prior to ImPACT administration

as measured by the Actigraph GT9X Link Physical Activity Monitor, using the Sadeh sleep scoring algorithm. Habitual, device-measured sleep duration was calculated using the Sadeh sleep scoring algorithm weighted average device-derived total nighttime sleep time (minutes) individuals experienced across at least 5 nights prior to ImPACT, as measured by the Actigraph GT9X Link Physical Activity Monitor. The following formula[352] was used to calculate the weighted average device-derived total nighttime sleep time, using the Sadeh sleep scoring algorithm (Equation 1). Subjective, single-night sleep duration was assessed by the self-reported total nighttime sleep time (minutes) individuals experienced the night prior to ImPACT administration as measured by the National Sleep Foundation Sleep Diary.

Demographic information was calculated using frequencies, means, standard deviations, and ranges. Normality of subjective, single-night, device-measured single-night, and habitual, device-measured sleep duration was assessed using the Shaprio-Wilk test and descriptive statistics (means, standard deviations, medians, and interquartile range [IQR]) were calculated. A scatter plot was created, and a Pearson correlation was performed to assess the strength of association between subjective, single-night and device-measured single-night sleep duration. A second scatter plot was created, and Pearson correlation was performed to assess the strength of association between subjective, single-night and habitual, device-measured sleep duration. However, given that correlation coefficients only determine the strength of the relationship,[373] Bland-Altman plots were used to evaluate the agreement between the sleep measurements.

Subjective, single-night sleep duration was assessed and compared to device-measured single-night sleep duration and habitual, device-measured sleep duration for the following measures of agreement: 1) the estimated fixed bias by calculating mean difference ($\bar{d}$), 2) the precision of the measurement, by calculating the 95% limits of agreement ($\bar{d}$ ± 1.96 SDs), and 3) proportional bias, by creating a regression line and 95% confidence intervals (CIs). If there was no fixed bias between the two methods of sleep duration, the $\bar{d}$ should be zero.[374] If there was no proportional bias between the two methods of sleep duration, then the regression of differences on means should produce a slope of zero.[374]

The $\bar{d}$ (fixed bias) was calculated and was displayed as a horizontal line. In addition, the standard deviation of the differences and 95% CIs were calculated. If the $\bar{d}$ (device-measured single-night sleep duration minus subjective, single-night sleep duration) was positive, subjective, single-night sleep duration underestimated device-measured single-night sleep duration. If the $\bar{d}$ was negative, subjective, single-night sleep duration overestimated device-measured sleep duration. Similarly, if the $\bar{d}$ (habitual, device-measured sleep duration minus subjective, single-night sleep duration) was positive, subjective, single-night sleep duration underestimated habitual, device-measured sleep duration. Finally, if the $\bar{d}$ was negative, subjective, single-night sleep duration overestimated habitual, device-measured sleep duration. Shaprio-Wilk tests were conducted to evaluate the normality of $\bar{d}$. A Wilcoxon signed-rank test was used to evaluate the significant difference between device-measured single-night sleep duration and subjective, single-night sleep duration, and a paired samples *t*-test was used to

evaluate the significant difference between habitual, device-measured sleep duration and subjective, single-night sleep duration. Statistical significance was set at $p \leq .05$.

Finally, Bland-Altman plots, with a proportional bias line and 95% CIs, regarding the level of agreement for 1) device-measured single-night sleep duration and subjective, single-night sleep duration and 2) habitual, device-measured sleep duration and subjective, single-night sleep duration were created to visually display the agreement between the sleep measures. The x-axis of the Bland-Altman plot represented the average of 2 sleep measures and the y-axis of the Bland-Altman plot represented the difference between 2 sleep measures.

Specific Aim 3: To examine the agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration in college-aged individuals.

Habitual, device-measured sleep duration was calculated using the weighted average device-derived total nighttime sleep time (minutes) individuals experienced across at least 5 nights prior to ImPACT, using the Sadeh sleep scoring algorithm. Habitual, subjective sleep duration was assessed by calculating the weighted average total nighttime sleep time (minutes) individuals experienced across at least 5 nights prior to ImPACT administration as measured by the National Sleep Foundation Sleep Diary. The following formula[352] was used to calculate the weighted average device-derived total nighttime sleep time, using the Sadeh sleep scoring algorithm, and subjective total nighttime sleep time (Equation 1).

Demographic information was calculated using frequencies, means, standard deviations, and ranges. Normality of habitual, device-measured sleep duration and habitual, subjective sleep duration was assessed using the Shaprio-Wilk test and

descriptive statistics (means, standard deviations) were calculated. A scatter plot was created, and a Pearson correlation was performed to assess the strength of association between habitual, device-measured sleep duration and habitual, subjective sleep duration. However, given that correlation coefficients only determine the strength of the relationship,[373] Bland-Altman plots were used to evaluate the agreement between the sleep measurements.

Habitual, subjective sleep duration was assessed and compared to habitual, device-measured sleep duration for the following measures of agreement: 1) the estimated fixed bias by calculating mean difference ($\bar{d}$), 2) the precision of the measurement, by calculating the 95% limits of agreement ($\bar{d}$ ± 1.96 SDs), and 3) proportional bias, by creating a regression line and 95% CIs. If there was no fixed bias between the two methods of sleep duration, the $\bar{d}$ should be zero.[374] If there was no proportional bias between the two methods of sleep duration, then the regression of differences on means should produce a slope of zero.[374]

The $\bar{d}$ (fixed bias) was calculated and was displayed as a horizontal line. In addition, the standard deviation of the differences and 95% CIs were calculated. If the $\bar{d}$ (habitual, device-measured sleep duration minus habitual, subjective sleep duration) was positive, habitual, subjective sleep duration underestimates habitual, device-measured sleep duration. If the $\bar{d}$ was negative, habitual, subjective sleep duration overestimates habitual, device-measured sleep duration. Shaprio-Wilk tests were conducted to evaluate the normality of the $\bar{d}$. A paired samples *t*-test was used to evaluate the significant difference between habitual, device-measured sleep duration and habitual, subjective sleep duration. Statistical significance was set at $p \leq .05$.

A Bland-Altman plot regarding the level of agreement for habitual, device-measured sleep duration and habitual, subjective sleep duration were created to visually display the agreement between the sleep measures. The x-axis of the Bland-Altman plot represented the average of 2 sleep measures and the y-axis of the Bland-Altman plot represented the difference between 2 sleep measures.

CHAPTER IV: RESULTS

Demographic Information on Total Sample

A total of 489 college-aged individuals were recruited between October 2020 and February 2021 for participation in the study and 63 were enrolled, yielding an enrollment rate of 12.9%. One participant was removed from the study after the informational session due to device malfunction and one participant was excluded after all data were collected due to a device malfunction and inability to download sleep scored data. The final analytic sample included a total of 61 college-aged individuals. The final sample (N = 61) is described below and demographics for each specific aim are presented below in each specific aim section. The final sample included 14 (14/61, 22.9%) male and 47 (47/61, 77%) female participants who were on average 20.30 (SD 1.17; range 18 – 24) years. Complete demographic information for the total sample is included in Table 1.

Table 1. Participant Demographics of the Total Sample (N = 61).

Demographic Variable	n (%)
Race	
Non-Hispanic White	53 (86.9)
Hispanic White	1 (1.6)
Asian	4 (6.6)
Asian/Latino	1 (1.6)
Asian/Non-Hispanic White	1 (1.6)
White/Asian	1 (1.6)
Academic Year	
Freshmen	6 (9.8)
Sophomore	9 (14.8)
Junior	22 (36.1)
Senior	24 (39.3)
Employed	33 (54.1)
Previous Concussion	
0	42 (68.9)
1	10 (52.6)
2	4 (21.1)
≥ 3	5 (26.3)
Headaches/Migraine Disorder	6 (9.8)
Learning Disorder/Dyslexia	1 (1.6)
ADD/ADHD	8 (13.1)
Depression/Anxiety	17 (27.9)
Sleep Disturbance	9 (14.8)

Morningness and Eveningness Questionnaire – Short Assessment (MEQ-SA)

All (61/61, 100%) participants completed the MEQ-SA. The distribution of MEQ-SA scores for the total sample were evaluated for normality and assessed using the Shaprio-Wilk test (W_{61} = 0.97, p = .12). The average MEQ-SA score for the sample was 48.75 (SD 8.90; range 33 – 66), which falls in the “intermediate” type. There were 11 (11/61, 18%) moderately morning, 34 (34/61, 55.7%) intermediate, 16 (16/61, 26.2%) moderately evening types.

Pittsburgh Sleep Quality Index (PSQI)

All (61/61, 100%) participants completed the PSQI. The distribution of PSQI scores were assessed for normality by the Shapiro Wilks test for the total sample, which

was determined to be non-normal (W_{61} = 0.95, p = .01). The median PSQI total score for the total sample was 6.0 (IQR 2.0; range 1 – 15). Approximately 64% (39/61, 63.9%) of participants had total PSQI scores greater than five indicating poor sleep quality. Descriptive statistics for PSQI components and total scores are provided in Table 2.

Table 2. Descriptive Statistics for PSQI Components and Total Score for the Total Sample (N = 61).

PSQI Component	Median [IQR] or n (%)
Duration of Sleep (min)	540.00 [375.00]
Sleep Disturbance	5.00 [5.00]
Sleep Latency (min)	1.00 [1.00]
Daytime Sleepiness	1.00 [1.00]
Sleep Efficiency	80.00 [35.18]
Overall Sleep Quality	1.00 [0.00]
Needs Medications to Sleep	0.00 [1.00]
Total Score	6.00 [2.00]
Sleep Quality Category, “Poor”	39 (63.9)

Abbreviation: IQR, Interquartile Range; Min, Minutes

National Sleep Foundation Sleep Diary

Fifty-seven (57/61, 93.4%) participants completed all sections of the National Sleep Foundation Sleep Diary, for a total of 483 nights of data. Bedtimes ranged from 9:30 PM to 7:30 AM across the 7-day study period. Waketimes ranged from 4:00 AM to 6:00 PM across the 7-day study period. Forty-nine (49/61, 86%) participants consumed at least one caffeinated drink across the 7-day period, 50 (50/61, 87.7%) participants exercised at least 20 minutes a day across the 7-day period, and 35 (35/61, 61.4%) participants reported taking at least one nap across the 7-day period.

Participants reported a 7-day average of 464.54 (SD 56.88; range 270 – 574.29) minutes of nighttime sleep. Participants reported on average of 461.40 (SD 63.10; range 246 – 588) minutes of nighttime sleep on the weekday nights (Sunday – Thursday) (mean 7.69 SD 1.05; range 4.10 – 9.80 hours) and 472.39 (SD 87.15; range

150 – 615) minutes of nighttime sleep on the weekend nights (Friday – Saturday) (mean 7.87 SD 1.45 hours; range 2.50 – 10.25 hours). There was no difference in nighttime sleep between weekday and weekend nights (t_{56} = -0.89, p = .38).

Evaluation of Specific Aims

Specific Aim 1: To investigate the influence of habitual, device-measured sleep duration on baseline neurocognitive performance and total concussion symptom severity in college-aged individuals.

Demographic Information

Ten participants (10/61, 16.4%) did not have at least 5 nights of complete device-measured sleep scored data, when utilizing the Sadeh sleep scoring algorithm. Therefore, there were 51 participants in the analytic sample. Thirty-two (32/51, 62.7%) participants had 7 nights of complete scored sleep data, 12 (12/51, 23.5%) participants had 6 nights of complete scored sleep data, and 7 (7/51, 12.7%) participants had 5 nights of complete scored sleep data. There were 38 (38/51, 74.5%) female and 13 (13/51, 25.5%) male participants who were on average 20.24 (SD 1.11; range 18 – 22) years old. There were 9 (9/51, 17.6%) collegiate student-athletes in the sample. The majority of participants identified as Non-Hispanic White (46/51, 90.2%), and were seniors (20/51, 39.2%). On average, participants were enrolled in 4.94 (SD 1.05) university classes. Twenty-six (26/51, 51%) participants were employed at the time of enrollment. The average number of days between the Informational Session and the Testing Session was 7.14 (SD 0.85; range 7 – 13) days.

Covariates

Fifteen (15/51, 29.4%) participants had a previous history of concussion, 6 (6/51, 11.8%) participants had a history of diagnosed sleep disturbance, and 6 (6/51, 11.8%) participants had diagnosed ADD/ADHD. A series of MANOVA tests were conducted in order to determine if there were any differences between demographic variables (gender, previous concussion history, history of diagnosed sleep disturbance, ADD/ADHD) and ImPACT composite scores and total concussion symptom severity. There were no significant effects of gender (Wilks $\lambda = 0.82$, $F_{5,45} = 1.93$, $p = .11$), previous concussion history (Wilks $\lambda = 0.84$, $F_{5,45} = 1.68$, $p = .16$), or ADD/ADHD (Wilks $\lambda = 0.81$, $F_{5,45} = 2.08$, $p = .09$) on the ImPACT composite scores and total symptom severity. However, there was a significant effect of history of diagnosed sleep disturbance (Wilks $\lambda = 0.76$, $F_{5,45} = 2.84$, $p = .03$) on the ImPACT composite scores and total symptom severity, specifically total concussion symptom severity ($F_{1,49} = 13.49$, $p \leq .01$).

Results

Participants wore the Actigraph GT9X Link monitor on average 9300.67 minutes (SD 25.56; range 3597 – 18322) (mean 6.46, SD 1.49; range 2.5 – 12.72 days). Using the Sadeh sleep scoring algorithm, the average total nighttime sleep was 322.13 minutes (SD 69.67; range 173.57 – 533). See Table 3 for means and standard deviations for ImPACT composite scores.

Table 3. Descriptive Statistics for ImPACT Composite Scores (n = 51).

ImPACT Composite Score	Mean (SD)
Verbal Memory	91.22 (7.83)
Visual Memory	79.00 (11.08)
Visual Motor Processing Speed	43.27 (5.57)
Reaction Time (sec)	0.57 (0.06)
Total Concussion Symptom Severity	5.86 (6.99)

Abbreviation: ImPACT, Immediate Post Concussion Assessment and Cognitive Testing Verbal Memory; SD, Standard Deviation; Sec, Seconds

For the regression models, the independent variable was habitual, device-measured sleep duration using the Sadeh sleep scoring algorithm, the dependent variables were the ImPACT composite scores, and the control variable was history of sleep disturbance.

Verbal Memory

The first model included history of sleep disturbance and was not statistically significant ($F_{1,49}$ = 0.26, p = .61, R^2 = 0.005). After adding habitual, device-measured sleep duration, the model remained non-significant ($F_{2,48}$ = 0.26, p = .77) and accounted for 01.1% of the variance (R^2 = 0.011). The R^2 change related to the addition of habitual, device-measured sleep duration (0.5%) was not statistically significant (F change$_{1,48}$ = 0.26, p = .61). In the final model, history of sleep disturbance (B = -1.41, 95% CI: -8.49, 5.68, β= -.06, p = .69) and habitual, device-measured sleep duration (B = 0.01, 95% CI: -0.03, 0.04, β= .08, p = .61) were not statistically significant.

Visual Memory

The first model included history of sleep disturbance and was not statistically significant ($F_{1,49}$ = 0.05, p = .82, R^2 = 0.001). After adding habitual, device-measured sleep duration, the model remained non-significant ($F_{2,48}$ = 0.04, p = .96) and accounted for 0.2% of the variance (R^2 = 0.002). The R^2 change related to the addition of habitual,

device-measured sleep duration (0.1%) was not statistically significant (F change$_{1,48}$ = 0.03, p = .87). In the final model, history of sleep disturbance (B = -0.98, 95% CI: -11.04, 9.09, β= -.03, p = .85) and habitual, device-measured sleep duration (B = 0.004, 95% CI: -0.04, 0.05, β= .02, p = .87) were not statistically significant.

Visual Motor Processing Speed

The first model included history of sleep disturbance and was not statistically significant ($F_{1,49}$ = 0.16, p = .69, R^2 = 0.003). After adding habitual, device-measured sleep duration, the model remained non-significant ($F_{2,48}$ = 0.41, p = .67) and accounted for 1.7% of the variance (R^2 = 0.017). The R^2 change related to the addition of habitual, device-measured sleep duration (1.3%) was not statistically significant (F change$_{1,48}$ = 0.66, p = .42). In the final model, history of sleep disturbance (B = -0.59, 95% CI: -5.61, 4.43, β= -.03, p = .82) and habitual, device-measured sleep duration (B = 0.009, 95% CI: -0.01, 0.03, β= .12, p = .42) were not statistically significant.

Reaction Time

The first model included history of sleep disturbance and was not statistically significant ($F_{1,49}$ = 2.57, p = .12, R^2 = 0.050). After adding habitual, device-measured sleep duration, the model remained non-significant ($F_{2,48}$ = 2.29, p = .11) and accounted for 8.7% of the variance (R^2 = 0.087). The R^2 change related to the addition of habitual, device-measured sleep duration (3.7%) was not statistically significant (F change$_{1,48}$ = 1.96, p = .17). In the final model, history of sleep disturbance (B = 0.04, 95% CI: -0.02, 0.09, β= .19, p = .20) and habitual, device-measured sleep duration (B = 0.000, 95% CI: 0.00, 0.00, β= -.20, p = .17) were not statistically significant.

Total Concussion Symptom Severity

The first model included history of sleep disturbance, and was statistically significant ($F_{1,49}$ = 13.49, p = .001) and accounted for 21.6% of the variance (R^2 = 0.216). After adding habitual, device-measured sleep duration, the model remained significant ($F_{2,48}$ = 6.63, p = .003) and accounted for 21.6% of the variance (R^2 = 0.216). The R^2 change related to the addition of habitual, device-measured sleep duration (0.1%) was not statistically significant (F change$_{1,48}$ = 0.03, p = .86). In the final model, history of sleep disturbance (B = 9.88, 95% CI: 4.25, 15.50, β= .46, p = .001) was statistically significant, but habitual, device-measured sleep duration (B = -0.002, 95% CI: -0.03, 0.02, β= -.02, p = .86) was not statistically significant.

Specific Aim 2: To assess the agreement between subjective, single-night sleep duration, and both device-measured single-night and habitual, device-measured sleep duration in college-aged individuals.

Demographic Information

Sixteen participants (16/61, 26.2%) did not have complete device-measured single-night sleep duration, 11 (11/61, 18%) did not have complete habitual, device-measured sleep duration, 1 (1/61, 1.6%) was excluded as an outlier. There were 42 participants in the analytic sample. Thirty-one (31/42, 73.8%) participants had 7 nights of sleep scored data, 7 (7/42, 16.7%) participants had 6 nights of sleep scored data, and 4 (4/42, 9.5%) participants had 5 nights of sleep scored data. There were 13 (13/42, 31%) males and 29 (29/42, 69%) females in the sample with an average age of 20.36 (SD 1.08; range 18 – 22) years.

Results

Upon inspection of the data, it was determined that device-measured single-night sleep duration (W_{42} = 0.98, p = .77), habitual, device-measured sleep duration (W_{42} = 0.98, p = .47), and subjective, single-night sleep duration (W_{42} = 0.95, p = .08) were normally distributed. See Table 4 for means and standard deviations for device-measured single-night, habitual, device-measured, and subjective, single-night sleep duration (minutes).

Table 4. Descriptive Statistics of Total Sleep Duration Measures in Minutes (n = 42).

Measures of Sleep Duration	Mean (SD)
Device-Measured Single-Night Sleep Duration	328.52 (96.14)
Habitual, Device-Measured Sleep Duration	322.30 (63.50)
Subjective, Single-Night Sleep Duration	480.33 (62.21)

Abbreviation: SD, Standard Deviation; IQR, Interquartile Range

Results from the Pearson's correlation revealed a non-significant relationship between device-measured single-night sleep duration and subjective, single-night sleep duration (r = 0.22, p = .16) (Figure 1) and between habitual, device-measured sleep duration and subjective, single-night sleep duration (r = 0.12, p = .44) (Figure 2).

Figure 1. Scatter Plot of Device-Measured Single-Night Sleep Duration (Minutes) and Subjective, Single-Night Sleep Duration (Minutes) (n = 42).

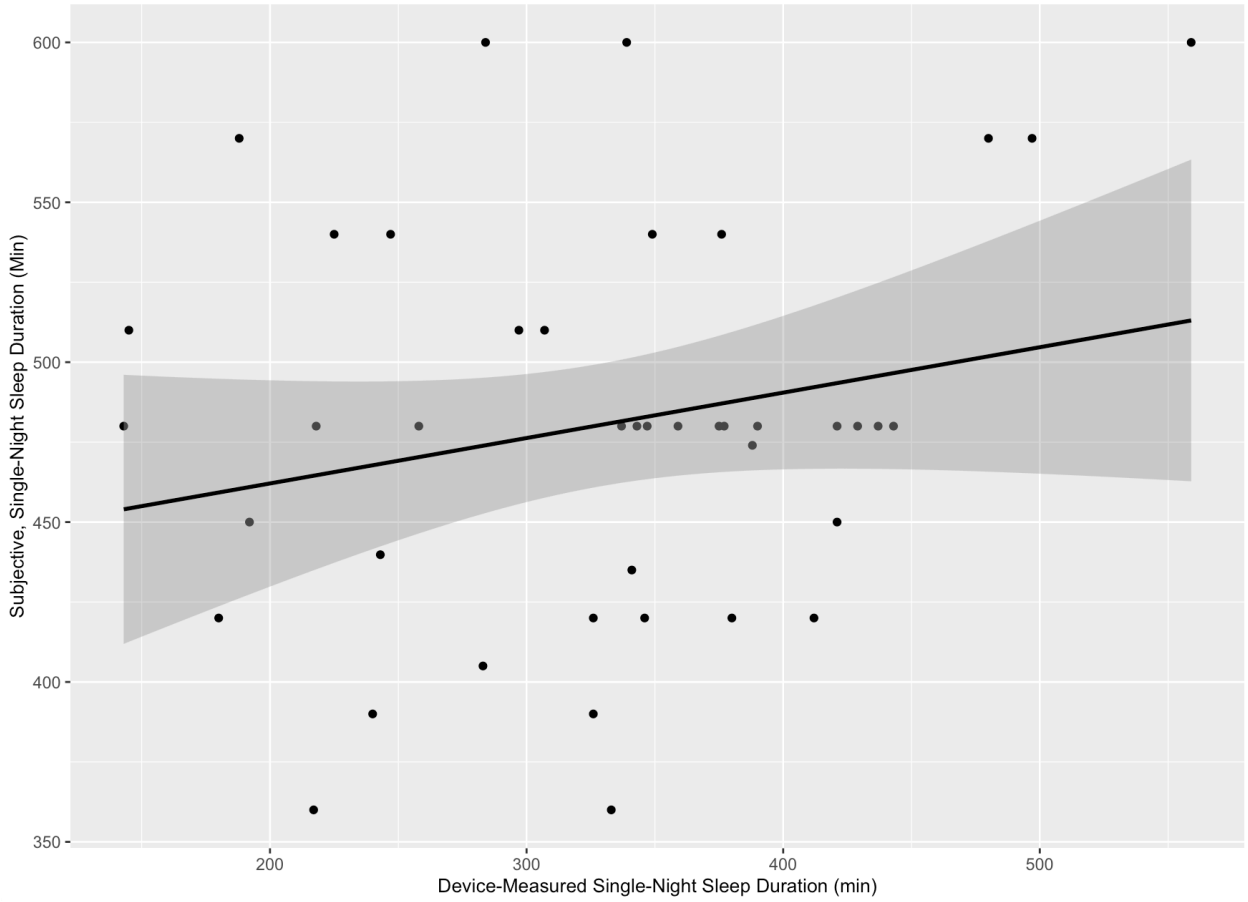
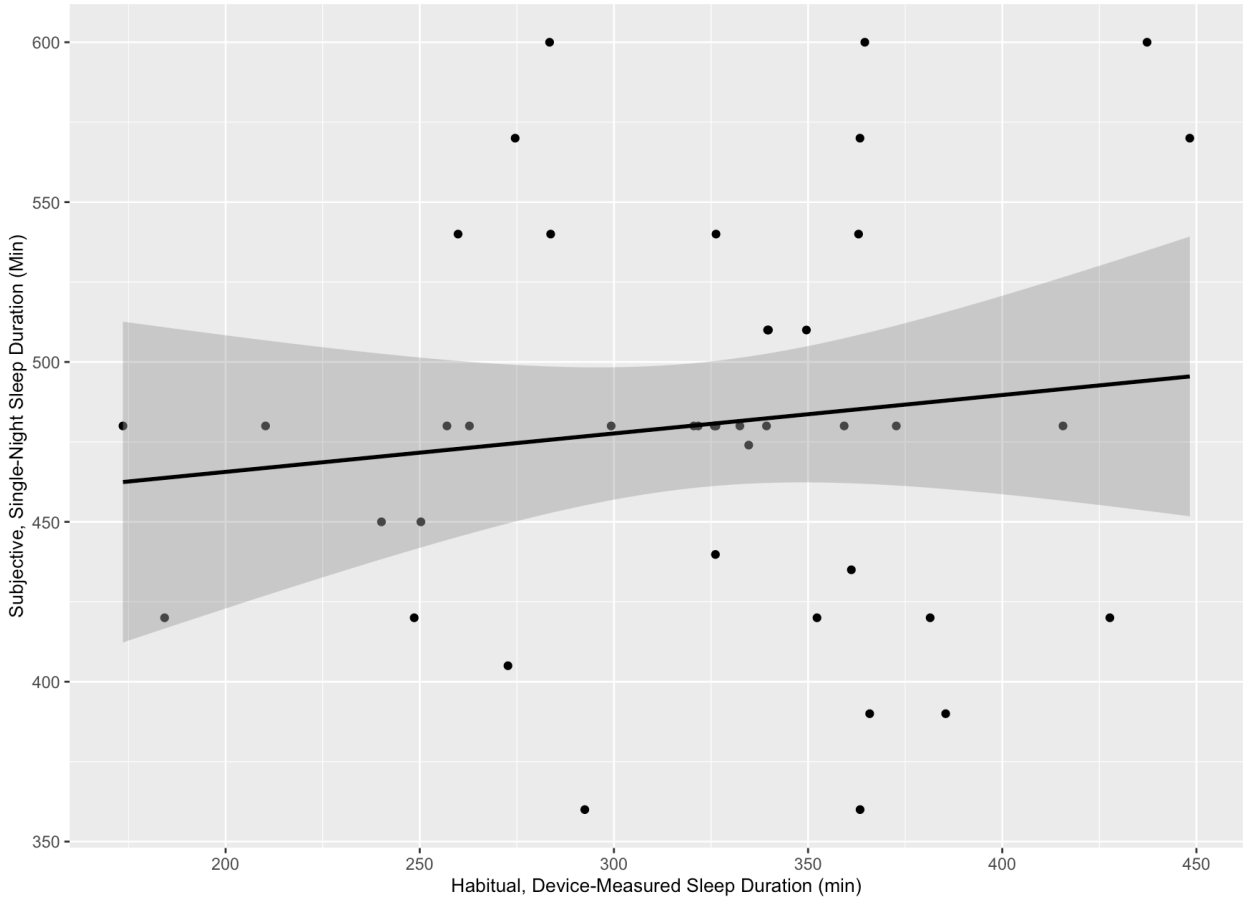


Figure 2. Scatter Plot of Habitual, Device-Measured Sleep Duration (Minutes) and Subjective, Single-Night Sleep Duration (Minutes) (n = 42).



In order to determine the fixed bias, the mean difference ($\bar{d}$) was calculated between 1) device-measured single-night sleep duration and subjective, single-night sleep duration and 2) habitual, device-measured sleep duration and subjective, single-night sleep duration (Table 5). The $\bar{d}$ was calculated in minutes. Upon inspection of the data, it was determined that the $\bar{d}$ of device-measured single-night and subjective, single-night sleep duration was not normally distributed ($W_{42} = 0.93$, $p \leq .01$), but the $\bar{d}$ between habitual, device-measured and subjective, single-night sleep duration was normally distributed ($W_{42} = 0.98$, $p = .51$).

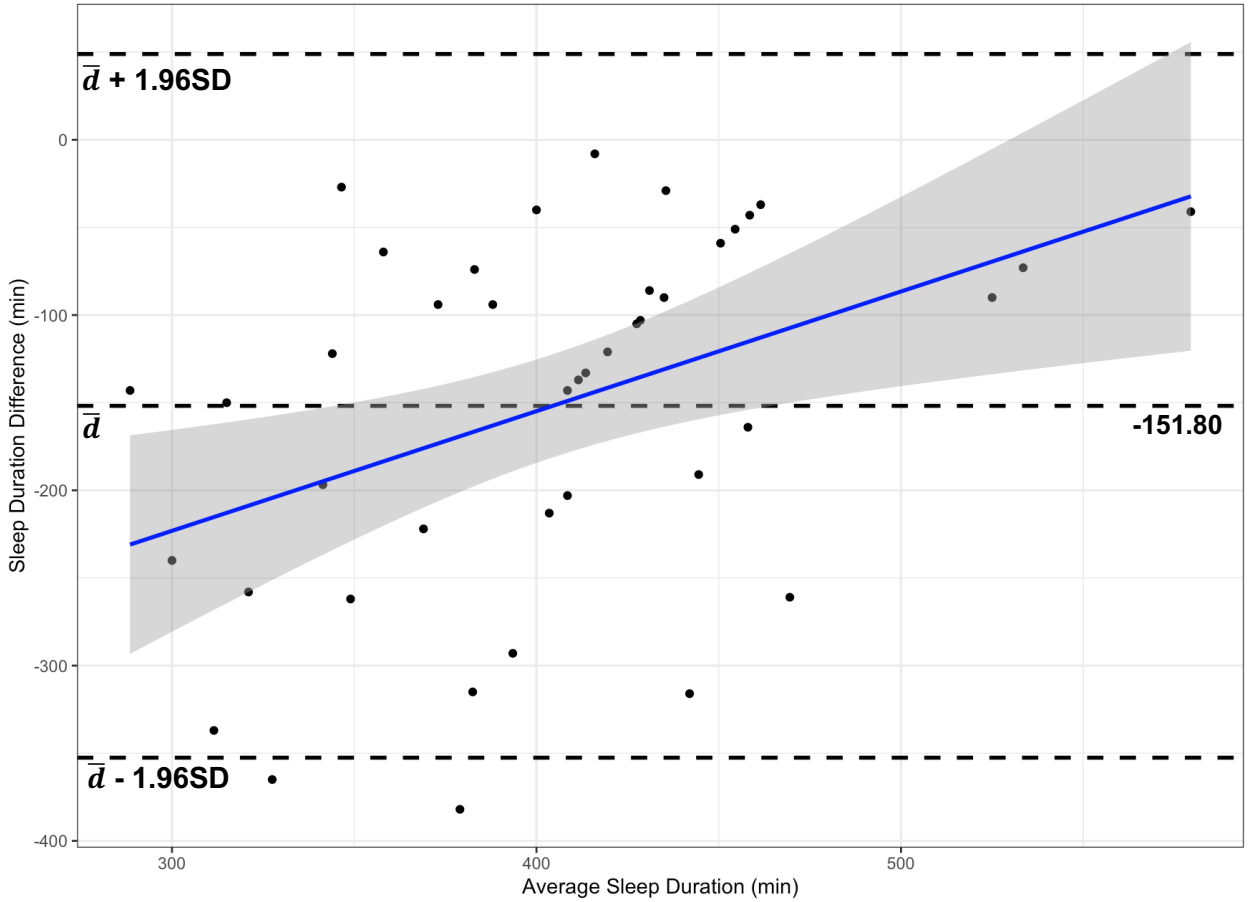
Table 5. Mean Difference, Standard Deviation, and 95% Confidence Intervals Between Device-Measured Single-Night, Habitual, Device-Measured and Subjective, Single-Night Sleep Duration (n = 42).

Measures of Sleep Duration	$\bar{d}$ (SD) (95% CI)	Lower Limit of Agreement (95% CI)	Upper Limit of Agreement (95% CI)
Device-Measured Single-Night Sleep Duration	-151.80 (102.42) (-183.72, -119.89)	-352.54 (-407.52, -297.56)	48.93 (-6.05, 103.91)
Habitual, Device-Measured Sleep Duration	-158.03 (83.27) (-183.98, -132.08)	-321.24 (-365.94, -276.53)	5.18 (-39.52, 49.88)

Abbreviation: $\bar{d}$, Mean Difference; SD, standard deviation; CI, Confidence Interval

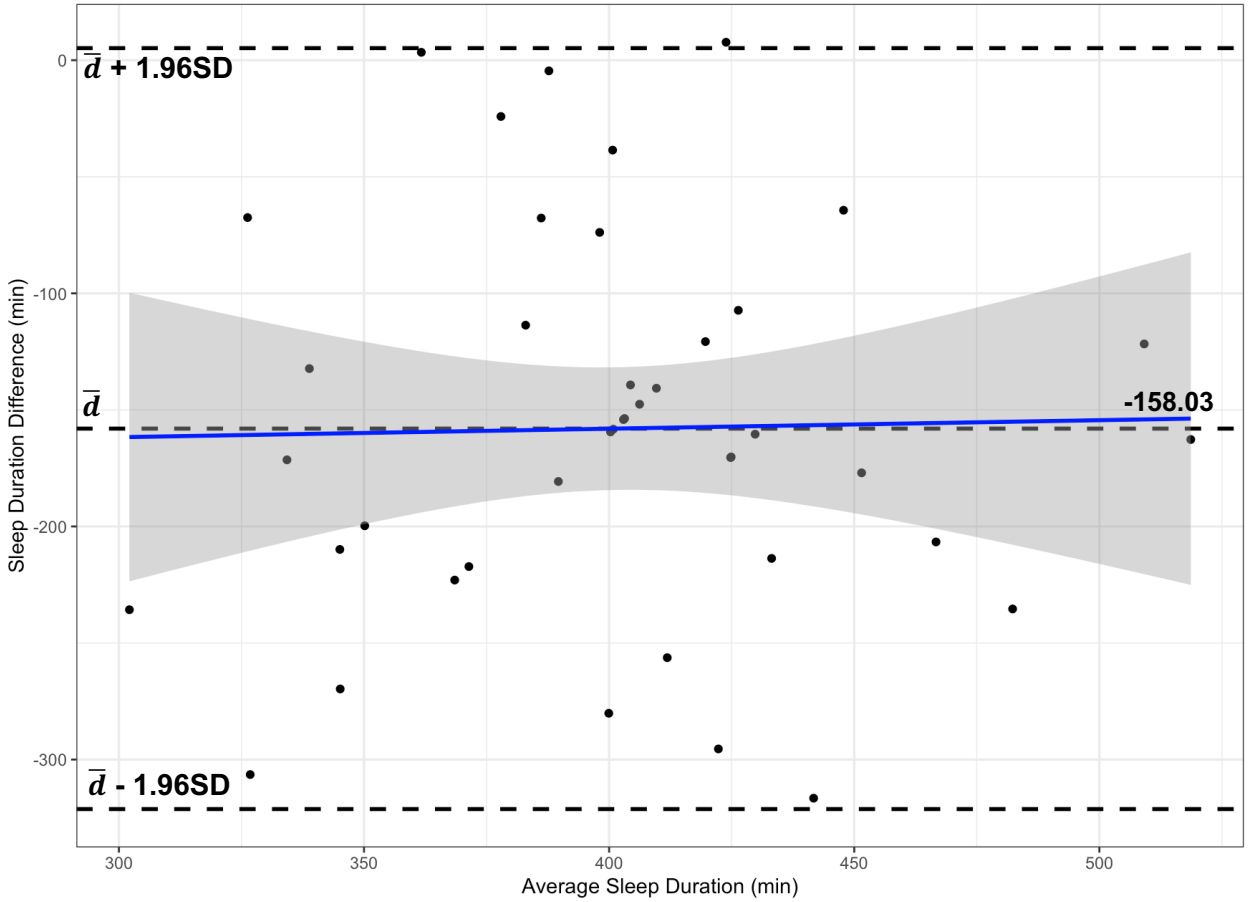
Compared to device-measured single-night sleep duration, subjective, single-night sleep duration significantly overestimated sleep duration by 151.80 minutes (Z = -5.08, $p \leq .001$). Compared to habitual, device-measured sleep duration, subjective, single-night sleep duration significantly overestimated sleep duration by 158.03 minutes (t_{41} = -12.30, $p \leq .001$). Bland-Altman plots were constructed to examine the degree of agreement between 1) device-measured single-night and subjective, single-night sleep duration (Figure 3) and 2) habitual, device-measured and subjective, single-night sleep duration (Figure 4).

Figure 3. Bland-Altman Plot of Device-Measured Single-Night Sleep Duration and Subjective, Single-Night Sleep Duration (n = 42).



The middle-dashed line is the mean difference ($\bar{d}$), the bottom-dashed line is the lower limit of agreement ($\bar{d}$ - 1.96SD), and the upper-dashed line is the upper limit of agreement ($\bar{d}$ + 1.96SD). The blue line indicates the proportional bias line (y = 0.68x – 427.93, R^2 = 0.175, $p \leq .01$) and the grey shade indicates the proportional bias line confidence intervals.

Figure 4. Bland-Altman Plot of Habitual, Device-Measured Sleep Duration and Subjective, Single-Night Sleep Duration (n = 42).



The middle-dashed line is the mean difference ($\bar{d}$), the lower-dashed line is the lower limit of agreement ($\bar{d}$ - 1.96SD), and the upper dashed line is the upper limit of agreement ($\bar{d}$ + 1.96SD). The blue line indicates the proportional bias line (y = 0.04x – 172.68, R^2 = 0.000, p = .90) and the grey shade indicates the proportional bias line confidence intervals.

Specific Aim 3: To examine the agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration in college-aged individuals.

Demographic Information

Eleven (11/61, 18%) participants did not have at least 5 nights of complete sleep scored data. Two (2/61, 3.3%) were excluded as outliers. The analytic sample included 49 (49/61, 80.3%) participants. Thirty-one (31/49, 63.3%) participants had 7 nights of sleep scored data, 11 (11/49, 22.4%) participants had 6 nights of sleep scored data,

and 7 (7/49, 14.3%) participants had 5 nights of sleep scored data. There were 13 (13/49, 26.5%) male and 36 (36/49, 73.5%) female participants with an average age of 20.27 (SD 1.11; range 18 – 22) years.

Results

Upon inspection of the data, it was determined that habitual, device-measured sleep duration (W_{49} = 0.98, p = .37) and habitual, subjective sleep duration (W_{49} = 0.97, p = .28) were normally distributed. See Table 6 for means and standard deviations for habitual, device-measured and habitual, subjective sleep duration.

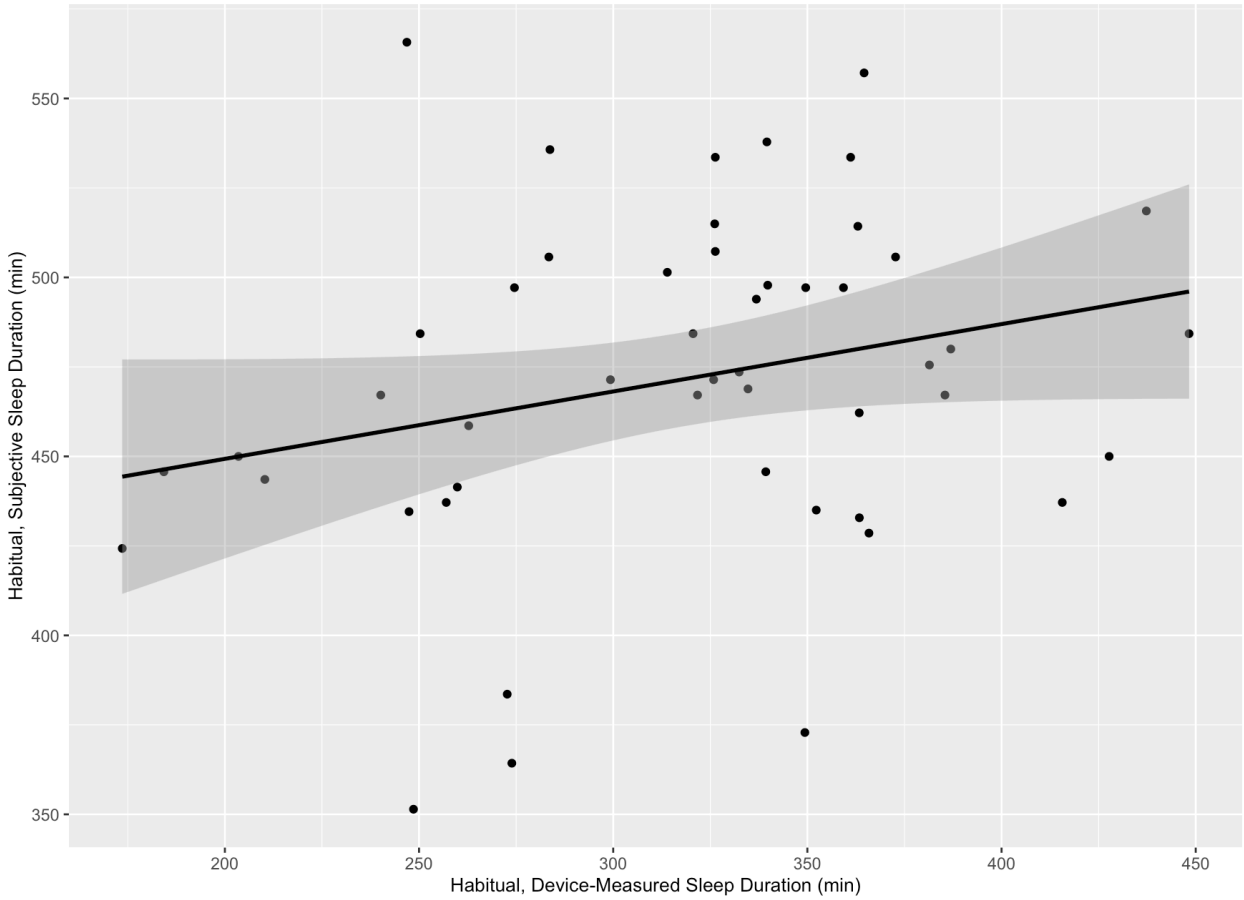
Table 6. Descriptive Statistics for Habitual, Device-Measured and Habitual, Subjective Sleep Duration in Minutes (n = 49).

Measure of Sleep Duration	Mean (SD)
Habitual, Device-Measured Sleep Duration	318.43 (64.01)
Habitual, Subjective Sleep Duration	471.61 (46.75)

Abbreviation: SD, Standard Deviation

Results from the Pearson's correlation revealed a non-significant relationship between habitual, device-measured sleep duration and habitual, subjective sleep duration (r = 0.26, p = .07) (Figure 5).

Figure 5. Scatter Plot Between Habitual, Device-Measured and Habitual, Subjective Sleep Duration (Minutes) (n = 49).

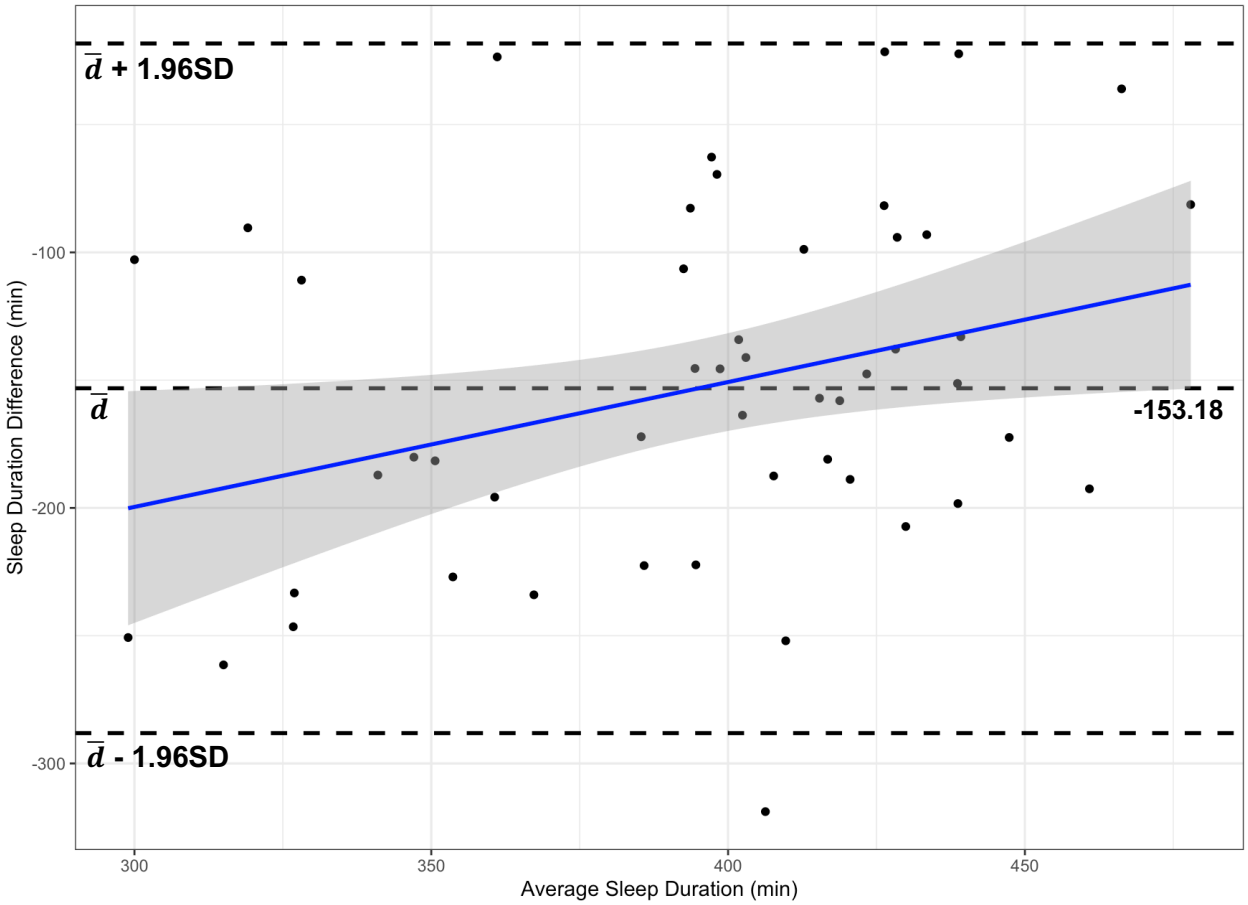


Mean difference ($\bar{d}$) was calculated between habitual, device-measured and habitual, subjective sleep duration. The $\bar{d}$ was calculated in minutes. Upon inspection of the data, it was determined that $\bar{d}$ between the two measures of sleep duration was normally distributed (W_{49} = 0.98, p = .69). The $\bar{d}$ was -153.18 (SD 68.85, 95% CI: -172.95, -133.40), the lower limit of agreement was -288.12 (95% CI: -322.15, -254.09), and the upper limit of agreement was -18.24 (95% CI: -52.26, 15.79).

Compared to habitual, device-measured sleep duration, habitual, subjective sleep duration significantly overestimated sleep duration by 153.18 minutes (t_{48} = -15.57, $p \leq .001$). Bland Altman plots were used to examine the degree of agreement

between habitual, device-measured sleep duration and habitual, subjective sleep duration (Figure 6).

Figure 6. Bland-Altman Plot of Habitual Device-Measured Sleep Duration and Habitual, Subjective Sleep Duration (n = 49).



The middle-dashed line is the mean difference ($\bar{d}$), the bottom-dashed line is the bottom limit of agreement ($\bar{d}$ – 1.96SD), and upper-dashed line is the upper limit of agreement ($\bar{d}$ + 1.96SD). The blue line indicates the proportional bias line (y = 0.49x – 346.20, R^2 = 0.10, p = .03) and the grey shade indicates the proportional bias line confidence intervals.

Supplemental Analyses

Habitual Device-Measured Sleep Variability

Rationale

It is possible that difficult sleep one night may lead to a period of sleep that is longer, deeper, and more restorative.[375] Sleep variability may be used to understand

how sleep varies across multiple nights.[376] Previous research suggests that greater sleep variability is negatively associated with reaction time[375] and mood.[377,378] Therefore, the purpose of this supplemental analysis was to examine the influence of habitual, device-measured sleep variability on baseline computerized neurocognitive performance and total concussion symptom severity in college-aged individuals. The data analysis for this supplemental analysis can be found in Appendix E.

Demographic Information

Eleven (11/61, 18%) did not have at least 5 days of sleep scored data when using the Sadeh sleep scoring algorithm. Three (3/61, 4.92%) participants were excluded as outliers. The sample consisted of 48 participants. Twenty-nine (29/48, 60.4%) participants had 7 nights of sleep scored data, 12 (12/48, 25%) participants had 6 nights of sleep scored data, and 5 (5/48, 14.6%) participants had 5 nights of sleep scored data. There were 13 (13/48, 27.1%) males and 35 (35/48, 72.9%) females in the sample. The average age was 20.25 (SD 1.14; range 18 – 22) years.

Covariates

There were 12 (12/48, 25%) participants with a history of previous concussion, 6 (6/48, 12.5%) with a history of sleep disturbance, and 5 (5/48, 10.4%) with ADD/ADHD. A series of MANOVA tests were conducted in order to determine if there were any differences between demographic variables (gender, previous concussion history, history of diagnosed sleep disturbance, ADD/ADHD) and ImPACT composite scores and total concussion symptom severity. Any demographic variables with significant differences in ImPACT composite scores or total concussion symptom severity were used as covariates for future analyses.

There was no significant effect of gender (Wilks $\lambda = 0.83$, $F_{5,42} = 1.72$, $p = .15$), previous history of concussion (Wilks $\lambda = 0.84$, $F_{5,42} = 1.65$, $p = .17$), or ADD/ADHD (Wilks $\lambda = 0.83$, $F_{5,42} = 1.78$, $p = .14$), but there was a significant effect of history of sleep disturbance (Wilks $\lambda = 0.76$, $F_{5,42} = 2.62$, $p = .04$), specifically total concussion symptom severity ($F_{1,46} = 12.51$, $p \leq .01$).

Results

Participants wore the Actigraph GT9X Link monitor on average 9385.27 (SD 2153.91; range 3597 – 18322) minutes (mean 6.52, SD 1.50; range 2.50 – 12.72 days). Using the Sadeh sleep scoring algorithm, the average total nighttime sleep variability was 95.84 (SD 29.21; range 28.34 – 161.63) minutes (mean 1.60, SD 0.49; range 0.47 – 2.69 hours). See Table 7 for means and standard deviations for ImPACT composite scores and total concussion symptom severity.

Table 7. Supplemental Descriptive Statistics for ImPACT Composite Scores (n = 48).

ImPACT Composite Score	Mean (SD)
Verbal Memory	91.54 (7.90)
Visual Memory	79.65 (10.87)
Visual Motor Processing Speed	43.22 (5.50)
Reaction Time	0.57 (0.06)
Total Concussion Symptom Severity	6.02 (7.14)

Abbreviation: ImPACT, Immediate Post Concussion Assessment and Cognitive Testing; SD, Standard Deviation

Verbal Memory

The first model included history of sleep disturbance and was not statistically significant ($F_{1,46} = 0.38$, $p = .54$, $R^2 = .008$). After adding habitual, device-measured sleep variability, the model remained non-significant ($F_{2,45} = 1.05$, $p = .36$) and accounted for 4.4% of the variance ($R^2 = .044$). The R^2 change related to the addition of habitual, device-measured sleep variability (3.6%) was not statistically significant (*F*

change$_{1,45}$ = 1.71, p = .20). In the final model, history of sleep disturbance (B = -2.34, 95% CI: -9.28, 4.60, β= -.10, p = .50) and habitual, device-measured sleep variability (B = -0.05, 95% CI: -0.13, 0.03, β= -.19, p = .20) were not statistically significant.

Visual Memory

The first model included history of sleep disturbance and was not statistically significant ($F_{1,46}$ = 0.15, p = .70, R^2 = .003). After adding habitual, device-measured sleep variability, the model remained non-significant ($F_{2,45}$ = .33, p = .72) and accounted for 1.4% of the variance (R^2 = .014). The R^2 change related to the addition of habitual, device-measured sleep variability (1.1%) was not statistically significant (F change$_{1,45}$ = 0.50, p = .48). In the final model, history of sleep disturbance (B = -1.73, 95% CI: -11.43, 7.97, β= -.05, p = .72) and habitual, device-measured sleep variability (B = 0.04, 95% CI: -0.07, 0.15, β= -.05, p = .72) were not statistically significant.

Visual Motor Processing Speed

The first model included history of sleep disturbance and was not statistically significant ($F_{1,46}$ = 0.15, p = .70, R^2 = .003). After adding, habitual, device-measured sleep variability, the model remained non-significant ($F_{2,45}$ = 0.10, p = .91) and accounted for 0.4% of the variance (R^2 = .004). The R^2 change related to the addition of habitual, device-measured sleep variability (0.1%) was not statistically significant (F change$_{1,45}$ = 0.05, p = .82). In the final model, history of sleep disturbance (B = -0.96, 95% CI: -5.89, 4.0, β= -.06, p = .70) and habitual, device-measured sleep variability (B = -0.01, 95% CI: -0.06, 0.05, β= -.04, p = .82) were not statistically significant.

Reaction Time

The first model included history of sleep disturbance and was not statistically significant ($F_{1,46}$ = 2.43, p = .13, R^2 = .030). After adding, habitual, device-measured sleep variability, the model remained non-significant ($F_{2,45}$ = 1.37, p = .27) and accounted for 5.7% of the variance (R^2 = .057). The R^2 change related to the addition of habitual, device-measured sleep variability (0.7%) was not statistically significant (F change$_{1,45}$ = 0.34, p = .56). In the final model, history of sleep disturbance (B = 0.04, 95% CI: -0.01, 0.10, β= .22, p = .14) and habitual, device-measured sleep variability (B = 0.00, 95% CI: -0.00, 0.00, β= -.08, p = .56) were not statistically significant.

Total Concussion Symptom Severity

The first model included history of sleep disturbance and was statistically significant ($F_{1,46}$ = 12.51, p = .001, R^2 = .214). After adding, habitual, device-measured sleep variability, the model remained significant ($F_{2,45}$ = 6.14, p = .004) and accounted for 21.4% of the variance (R^2 = .214). The R^2 change related to the addition of habitual, device-measured sleep variability (0.1%) was not statistically significant (F change$_{1,45}$ = 0.04, p = .85). In the final model, history of sleep disturbance (B = 9.90, 95% CI: 4.21, 15.60, β= .46, p = .001) was statistically significant, but habitual, device-measured sleep variability (B = 0.01, 95% CI: -0.06, 0.07, β= .03, p = .85) was not statistically significant.

Cole-Kripke Sleep Scoring Algorithm

Rationale

The two most common sleep scoring algorithms used to detect sleep-wake periods are the Sadeh and Cole-Kripke algorithms. The Sadeh sleep scoring algorithm was originally validated in adolescents and young adults aged 10 – 25 years old and the

Cole-Kripke was originally validated in adults aged 35 – 65 years old.[355,360] Although the Sadeh sleep scoring algorithm was appropriate in the current study based on the participant's age, previous literature[357] has reported both the Sadeh and Cole-Kripke scored data. In addition, Quante and colleagues[379] found that both algorithms were comparable to PSG in children and adults. The purpose of this supplemental analysis was to examine the relationship between Sadeh sleep scored habitual, device-measured sleep duration and Cole-Kripke sleep scored habitual, device-measured sleep duration. The data analyses for these results are found in Appendix F.

Demographic Information

Eleven (11/61, 18%) participants did not have at least 5 nights of sleep scored data using the Sadeh sleep scoring algorithm and 3 (3/61, 4.9%) participants did not have at least 5 nights of sleep scored data using the Cole-Kripke sleep scoring algorithm. There were 4 (4/61, 6.6%) participants were outliers and were excluded. There was a total of 46 participants in the sample. Twenty-nine (29/46, 63%) participants had 7 nights of sleep scored data when using the Sadeh sleep scoring algorithm, 11 (11/46, 23.9%) participants had 6 nights of sleep scored data when using the Sadeh sleep scoring algorithm, and 6 (6/46, 13%) participants had 5 nights of sleep scored data when using the Sadeh sleep scoring algorithm. Forty-one (41/46, 89.1%) participants had 7 nights of sleep scored data when using the Cole-Kripke sleep scoring algorithm, 4 (4/46, 8.7%) participants had 6 nights of sleep scored data when using the Cole-Kripke sleep scoring algorithm, and 1 (1/46, 2.2%) participant had 5 nights of sleep scored data when using Cole-Kripke sleep scoring algorithm. There were 33 (33/46,

71.7%) female participants and 13 (13/46, 28.3%) male participants in the sample with an average age of 20.26 (SD 1.16; range 18 – 22) years.

Results

The average Sadeh sleep scored total nighttime sleep time was 317.39 (SD 62.06; range 173.57 – 437.29) minutes. The average Cole-Kripke sleep scored total nighttime sleep time was 376.11 (SD 72.85; range 209 – 542.50) minutes. There was a significant relationship between Sadeh sleep scored habitual, device-measured sleep duration and Cole-Kripke sleep scored habitual, device-measured sleep duration ($F_{1,44}$ = 9.96, $p \leq .01$, $R^2 = .185$).

CHAPTER V: DISCUSSION

Specific Aim 1

The primary purpose of this study was to investigate the influence of habitual, device-measured sleep duration on baseline neurocognitive performance and total concussion symptom severity in college-aged individuals. The habitual, device-measured total sleep time for participants in the current study was 322.13 minutes (5.37 hours). Similarly, Lee and colleauges[357] reported a device-measured average total sleep time of 325.2 minutes (5.47 hours) across 4 nights in adults aged 19 – 66 years. In addition, other studies[371,379,380] have reported an average total sleep time via wearable technologies ranging from 363.6 minutes (6.06 hours) – 421.6 minutes (7.03 hours). Recently, the National Sleep Foundation updated sleep duration recommendations and concluded that healthy young adults (aged 18 – 25) should get between 7 – 9 hours of sleep, however 6 hours may be appropriate.[28]

Contrary to the hypothesis, short habitual, device-measured sleep duration was not associated with worse baseline neurocognitive performance and total concussion symptom severity. This was the first study to investigate the influence of habitual, device-measured sleep duration via actigraphy on baseline ImPACT performance. However, previous researchers have investigated the influence of subjective, single-night sleep duration on baseline ImPACT performance.[37–41] Whether or not short sleep duration affects baseline CNT performance is mixed. Sufrinko et al.[37] observed that high school athletes with less than 5 hours of sleep the night before ImPACT administration demonstrated worse verbal memory, visual motor speed, and reaction time. In addition, McClure and colleagues[38] reported that high school and collegiate athletes with less

than 7 hours of sleep the night before ImPACT baseline testing demonstrated worse performance on reaction time, verbal memory, and visual memory. Although these two studies found that short sleep duration negatively influences ImPACT composite scores, other researchers found that short sleep duration only negatively influences baseline concussion symptomology. Moran and colleagues[40] observed no differences in self-reported prior night's sleep duration on ImPACT composite scores. Similarly, Silverberg et al.[41] reported no significant relationship between subjective, single-night sleep duration and ImPACT composite scores.

There may be several reasons for why habitual, device-measured did not influence baseline neurocognitive performance in the current study. Previous studies that investigated the relationship between short, habitual sleep duration and cognition utilized acute sleep deprivation and restriction in highly controlled sleep laboratories. For example, Stocker and colleagues[43] had young adults sleep in a sleep laboratory for 3 nights and randomly assigned them to 3 groups: undisrupted normal sleep, sleep restriction (50% of habitual sleep), and total sleep deprivation. Results showed that sleep loss was associated with deficits in tasks of visual memory, reaction time, and visual processing motor speed.[43] Unlike the current study, participants were deprived of sleep from 19 hours to 40 hours.[43] In another study,[381] participants were deprived of sleep for up to 72 hours. Therefore, the sleep loss in previous studies may be greater than that in the current study. In the current study, only 2 participants reported not sleeping the night prior to ImPACT administration Studies on this topic are often designed to test participants who work in extreme environments. For example, studies have included truck drivers,[382] medical residents,[383] military personnel,[381] and pilots[384]

who are required to practice sleep deprivation in order to be successful at their job. Although college students may voluntarily limit their sleep to meet academic and social demands, the sleep restriction may not be as extreme or consistent as those that work in a sleep-deprived state.[36,39] Further research is needed to determine the ImPACT battery's sensitivity to habitual sleep and sleep loss. In addition, future researchers should investigate how other sleep-related variables, like sleep efficiency, may impact baseline CNT performance.

Although the finding that college students are sleeping less than the recommended amount is concerning, it is not surprising. College students are notorious for practicing volitional sleep restriction in order to meet academic and social demands.[36] Compounded with the COVID-19 pandemic, which may add additional stresses (e.g., social restrictions), college students may not be getting the appropriate amount of sleep. Mezick and colleagues[385] reported that stressful life events are associated with increased sleep variability; therefore, it is possible that participant stress levels could have influenced sleep duration in the current study. However, it should be noted that some individuals sleep shorter or longer than recommended without any adverse effects.[28] In addition, it is also important to consider that sleep variability may also influence baseline CNT performance. However, results from supplementary analyses revealed no relationship between habitual sleep variability and baseline ImPACT composite scores or total concussion symptom severity.

Surprisingly, and contrary to previous literature,[37–41] there was no relationship between habitual, device-measured sleep duration and total concussion symptom severity. McClure et al[38] observed significantly more concussion symptoms in

participants who got less than 7 hours of sleep compared to those who got intermediate (7 – 9 hours) and long (≥ 9 hours) sleep. In a meta-analysis, Pilcher and colleagues[330] found that sleep insufficiency affects mood more than cognitive and motor performance. However, it is possible that the combination of sleep quantity and sleep quality may affect concussion symptom severity more so than sleep quantity alone. In the current study, only sleep quantity was analyzed. Although sleep quantity and quality may overlap, sleep quantity consists of quantifiable components such as sleep duration, whereas sleep quality includes how well rested one feels upon awakening and general satisfaction with sleep.[386,387] Pilcher and colleagues[36] observed that sleep quality was better related to sleepiness and feelings of tension, depression, anger, fatigue, and confusion compared to sleep quantity. Mihalik and colleagues[39] did not observe increased severity in somatic (e.g., headache, nausea), cognitive (e.g., difficulty concentrating, feeling in a “fog”), or neurobehavioral (e.g., trouble falling asleep, drowsiness) symptoms in individuals with lower subjective, single-night sleep quantity. However, participants with lower sleep quality reported increased severity in neurobehavioral symptoms.[39] In addition, Sufrinko and colleagues[37] combined sleep quantity and sleep quality by grouping individuals with ≤ 5 hours of sleep and at least 1 or more symptoms in the sleep symptom cluster (i.e., trouble falling asleep, sleeping less than usual). The results revealed that participants in the sleep problems group (low sleep quantity and poor sleep quality) reported more concussion symptoms at baseline compared to participants in the control group.[37] Therefore, it is possible that the combination of sleep quantity and quality may influence baseline concussion symptoms.

Future research is needed to better understand the effect of sleep quality on baseline concussion symptoms.

Specific Aim 2

The secondary aim of this study was to assess the agreement between both device-measured single-night sleep duration and habitual, device-measured sleep duration and subjective, single-night sleep duration in college-aged individuals. Contrary to hypothesis 2a, the results of the current study revealed no agreement between device-measured single-night sleep duration and subjective, single-night sleep duration. However, hypothesis 2b was supported by the results of this study, which revealed no agreement between habitual, device-measured sleep duration and subjective, single-night sleep duration.

In the current study, device-measured single-night total sleep time (328.52 minutes [5.47 hours]) was significantly shorter than subjective single-night total sleep time. Compared to device-measured single-night sleep duration, subjective, single-night total sleep time significantly overestimated sleep duration by 151.80 minutes (2.53 hours). The Bland-Altman plot showed that device-measured single-night and subjective, single-night sleep duration only agreed within ± 3.35 hours (200.74 minutes). Given the $\bar{d}$ was far from zero and that the limits of agreement were large, the agreement between device-measured single-night sleep duration and subjective, single-night sleep duration was not acceptable.

In addition, the Bland-Alman plot between device-measure single-night sleep duration and subjective, single-night sleep duration provides evidence of proportional bias between the two measures (Fig. 3). Specifically, short sleepers tended to overestimate sleep duration, more so than long sleepers. Similarly, Lauderdale et al.[371] found that subjective sleep diary sleep duration was 38 minutes longer than device-measured single night sleep duration, which is considerably closer to the findings of the current study. This finding suggests that participants in the current study may not be able to accurately report how much they sleep in a single night.[371] It is possible that participants may have reported time spent awake in bed rather than actual sleep time.

Although device-measured single-night sleep duration and subjective, single-night sleep duration demonstrated no agreement, these results should be interpreted with caution. The $\bar{d}$ between device-measured single-night sleep duration and subjective, single-night sleep duration was not normally distributed. The calculation of 95% limits of agreement is based on the assumption that the difference between the two measures is normally distributed.[373] Therefore, it is possible that the limits of agreement could be wider apart than necessary for short sleep duration and narrower than necessary for longer sleep duration.[373]

The results of the study suggest no agreement between habitual, device-measured sleep duration and subjective, single-night sleep duration. Compared to habitual, device-measured sleep duration, subjective single-night sleep duration significantly overestimated sleep duration by 158.03 minutes (2.63 hours). The Bland-Altman plot showed that the two measures of sleep duration only agree within ± 2.72 hours, which may not be clinically acceptable. It is hard to say that the results of this

study are reflected in previous literature because very little research has been designed to investigate the agreement between habitual, device-measured sleep duration and subjective, single-night sleep duration. Typically, large epidemiological studies utilize questionnaires that ask the individual to report what time they typically wake up and go to bed (i.e., “During the past month, how many hours of actual sleep did you get at night?”), rather than ask single-item questions like used in the current study.[371,388,389] Given the results of this study, subjective, single-night sleep duration is not an acceptable substitute for habitual, device-measured sleep duration.

Specific Aim 3

The tertiary purpose of the current study was to examine the agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration in college-aged individuals. Current study findings do not support hypothesis 3, as the results suggest no agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration.

In the current study, habitual, subjective total sleep time (471.61 minutes [7.86 hours]) was significantly longer than habitual, device-measured total sleep time (318.43 minutes [5.31 hours]), revealing evidence of fixed bias between the two methods. Compared to habitual, device-measured sleep duration, habitual, subjective total sleep time overestimated sleep duration by almost 2.5 hours (153.18 minutes [2.55 hours]). This finding is supported by previous research.[357,371,390–393] Lee and colleagues[357] reported habitual device-measured total sleep time of 325.2 minutes via Actigraph GT9X and habitual, subjective total sleep time of 439.8 minutes via sleep diary. Similarly, Lauderdale et al.[371] reported 6.06 hours of habitual, device-measured sleep

and 6.83 hours of habitual, self-reported sleep duration. Furthermore, Arora and colleagues[390] observed a 54.2-minute difference between habitual, device-measured total sleep time (449 minutes) and habitual, subjective total sleep time (531 minutes). However, two studies[394,395] found that habitual, device measured total sleep time was longer than habitual, subjective total sleep time. Lockley et al.[394] found a longer device-measured sleep period compared with subjective sleep logs. Similarly, Landry and colleagues[395] reported 408.8 minutes for habitual, device-measured total sleep time and 398 minutes for habitual, subjective total sleep time. Although these 2 studies[394,395] present conflicting results to the current study, it is important to consider the participants. Lockley and colleagues[394] included individuals who were blind, and Landry et al.[395] included older adults, 55 years and older. Individuals who are blind are likely to have circadian rhythm disorders and sleep complaints are common among older adults.[394,396]

Results of the current study suggest that there was no agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration. This finding was confirmed upon closer inspection of the Bland-Altman plot. The lower and upper limits of agreement and their 95% CIs between the two measures of sleep duration are wide, possibly due to great variance in the differences and small sample size.[373] The Bland-Altman plot shows that habitual, subjective total sleep time significantly overestimates habitual, device-measured total sleep time and the two measures of sleep duration only agree within ± 2.25 hours, which may not be acceptable for clinical purposes.[390] Previous research[359,392] suggests that a difference in ≤ 30 minutes between 2 measures of total sleep time was satisfactory. Furthermore, the

Bland-Altman plot in the current study provides evidence of proportional bias between habitual, device-measured sleep duration and habitual, subjective sleep duration, specifically short sleepers tended to overestimate sleep duration more so than long sleepers (Fig. 6).

Similar results were found in previous studies.[357,371,392] Lauderdale and colleagues[371] observed proportional bias between habitual, device-measured sleep duration and subjective habitual sleep duration. Lee and colleagues[357] compared total sleep time as measured by wearable trackers to sleep diaries in adults. The results revealed that Actigraph GT9X compared to sleep diary produced the greatest mean difference, and therefore, the greatest fixed bias suggesting no agreement.[357] In addition, Werner et al.[392] found no agreement between total sleep time as measured by actigraphy and sleep diary.

There may be several reasons that can be attributed to the lack of agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration. Individuals may not be sure how much sleep they actually get and when asked to report the number of hours slept, they may answer what they think is normal.[371] Furthermore, individuals may confuse time in bed with actual sleep time or may disregard the amount of time spent awake in bed.[390] Alternatively, methodological issues with actigraphy may contribute to poor agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration. Results of this study and supplemental results revealed that the Sadeh and Cole-Kripke algorithm derived total sleep time had no agreement with habitual, subjective total sleep time. Actigraph devices do not measure sleep directly. Rather, they use raw counts that are translated

to sleep-wake scores via algorithms based on assumptions of physical movement, sedentary activity, and sleep behavior.[356,357]

Quante et al.[379] reported that both the Sadeh and Cole-Kripke sleep scoring algorithms demonstrated poor specificity for wake time. Therefore, participants who toss and turn frequently may skew data or others may have missing nights of data.[357] In addition, the Sadeh sleep scoring algorithm was originally validated in a healthy sample of adolescent and young adults (10 – 25 years)[355] and the Cole-Kripke sleep scoring algorithm was validated in adults aged 35 – 65.[360] However, it should be noted that the Sadeh sleep scoring validation study only included 9 young adults (20 – 25 years).[355] Furthermore, Lee and colleagues[357] observed that the Cole-Kripke sleep scoring algorithm performed better than the Sadeh sleep scoring algorithm in 19 – 66 year olds. Therefore, researchers should continue to compare the Sadeh and Cole-Kripke sleep scoring algorithms to the gold standard, PSG, to determine which algorithm is most appropriate for young adults. Therefore, more research is needed to understand which algorithm is most appropriate for young adults. Future studies should continue to develop methodologies and algorithms in order to address these limitations.[356]

Another possible reason for the lack of agreement between habitual, device-measured sleep duration and habitual, subjective sleep could be due to the specific type of actigraph or wearable device. Other wearable devices, such as the Fitbit Charge HR or the Actiwatch 2, allow for user interaction to confirm bedtimes and wake times through interactive apps or event markers, which may aid in the identification of sleep periods.[357,371] However, some of these wearable devices, such as the Fitbit Charge HR, utilize unknown proprietary algorithms to define sleep periods, which are unknown to

the researcher or clinician.[397] Wearable technologies and self-reported sleep diaries continue to be used in research studies and various populations. Therefore, researchers should continue to compare device-measured and subjective measures of total sleep time. In addition, researchers should compare other sleep-related variables such as time in bed, sleep efficiency, and wake after sleep onset between device-measured and subjective measures.

Clinical Implications

The results of this study suggest that habitual, device-measured sleep duration does not influence baseline computerized neurocognitive performance or total concussion symptom severity in college-aged individuals. In addition, when compared to subjective, single-night sleep duration, habitual device-measured sleep duration showed no agreement. Furthermore, habitual, device-measured sleep duration demonstrated no agreement to habitual, subjective sleep duration. Therefore, researchers should be cautious when interchangeably using device and subjective measures of sleep.

Actigraphy is a cost-effective method of collecting objective measures of sleep allowing researchers to continuously measure sleep in the individual's home environment. Despite these advantages, the current study highlights the need to further understand the clinical utility of actigraphy to measure sleep in college-aged individuals. This study may highlight the need to use complementary subjective methods of measuring sleep in conjunction with actigraphy. Although the Sadeh and Cole-Kripke sleep scoring algorithms provide comparable data to the gold standard PSG,[356,379] validity concerns still exist. It is possible that subjective methods of sleep, such as sleep

diaries, may provide researchers with additional information. Given that sleep scoring algorithms only look for sleep,[371] using subjective measures of sleep concurrently with actigraphy may help researchers accurately identify sleep periods or may provide points of reference for analysis. In fact, Littner and colleagues[398] recommended that sleep diaries be used in conjunction with actigraphy and that the combination may be an accepted valid, practical alternative to PSG.

Strengths and Limitations

This was the first study to investigate the influence of habitual, device-measured sleep duration on baseline computerized neurocognitive performance and total concussion symptom severity. However, this study was not without limitations. First, data collected for the current study were collected during the COVID-19 pandemic. It is possible that the global pandemic and the associated social restrictions could have influenced sleep patterns and increased stress in participants in this study. As a result of the global pandemic, reseachers[399,400] observed that college students reported going to bed later, waking up later, increasing digital media use before bed, and worsening sleep quality during the COVID-19 lockdown in Italy. It is possible that the restrictions associated with the COVID-19 pandemic could have negatively influence participant's sleep quantity in the current study. Second, research shows that Actigraphy may overestimate measures of sleep quantity compared to the gold-standard PSG. Specifically, actigraphy may overestimate sleep quantity in insomniacs as still awakenings may be counted as sleep.[401] Actigraphy is designed to start sleep data collection at the time of arm movement cessation, however it is possible that the arm may stop moving before the individual falls asleep.[356] Third, participants were asked to

complete the National Sleep Foundation Sleep Diary prior to the Testing Session and ImPACT administration. Therefore, it is possible that the sleep diary information could have informed the subjective, single-day total sleep time that is reported on ImPACT. Finally, these data were collected solely at Michigan State University, specifically recruited from kinesiology and exercise classes, which could impact the generalizability of the study results.

Conclusions

Previous research[37–41] suggests that short sleep duration negatively influences baseline CNT performance and total concussion symptom severity. However, previous studies have relied on subjective measures of sleep quantity and have only investigated the effects of a single night of sleep. Results of the current study suggest that habitual, device-measured sleep duration does not influence baseline CNT performance or total concussion symptom severity. More research is needed to understand the impact of habitual, device-measured sleep duration on baseline CNT performance and total concussion symptom severity.

The results of the current study revealed no agreement between subjective, single night sleep duration and both device-measured single night and habitual, device-measured sleep duration. Furthermore, there was no agreement between habitual, device-measured sleep duration and habitual, subjective sleep duration. Wearable devices that provide an alternative to subjective reports have significant implications for future research. As wearable technologies become more available, more research is needed to understand the utility of these devices.

APPENDICES

APPENDIX A

Demographics Form

Subject ID: _____

Screening/Eligibility – Confirm eligibility prior to enrollment

Inclusion Criteria:

18 and 25 years of age	YES	NO
Currently participate in a National Collegiate Athletic Association (NCAA) sanctioned sport	YES	NO

Exclusion Criteria:

Does not speak or read English	YES	NO
Diagnosed with a concussion within the last six months	YES	NO

Demographic Information –

Gender: M F

Date of Birth: _____/_____/_____

Age: _____

Race:

- □ Asian
- □ Native Hawaiian or Other Pacific Islander
- □ Non-Hispanic Black or African American
- □ Non-Hispanic White
- □ Other: _____
- □ Native American of Alaska Native
- □ Hispanic Black
- □ Hispanic White
- □ Unknown

Year in School: FR SOPH JR SR Graduate

How many university classes are you currently enrolled in? _____

Are you currently employed? YES NO

If currently employed, how many jobs do you have? _____

If currently employed, please describe job(s): _____

Parent's Highest Level of Education

Mother
□ Some high school
□ High school diploma or equivalent
□ Some college, no degree
□ Associate degree
□ Bachelor's degree
□ Master's degree
□ Doctoral/Professional degree

Father
□ Some high school
□ High school diploma or equivalent
□ Some college, no degree
□ Associate degree
□ Bachelor's degree
□ Master's degree
□ Doctoral/Professional degree

Parent's Occupational Category

□ Professional
□ Employer
□ Employee
□ Retired
□ Self-employed
□ Unemployed
□ Other: _____

Parent's Specific Occupation: _____

Family Household Income

□ Less than $10,000
□ $10,000 to 19,999
□ $20,000 to 29,999
□ $30,000 to $39,999
□ $40,000 to $49,999
□ $50,000 to $59,999
□ $60,000 to $69,999
□ $70,000 to $79,999
□ $80,000 to $89,999
□ $90,000 to $99,999
□$100,000 to $149,999
□$150,000 or more

Previous Medical History –

Previous Concussion	YES	NO
If yes, how many	_____	
Date of most recent	_____/_____/_____	
Headaches/Migraine Disorders	YES	NO
Learning Disorder/Dyslexia	YES	NO
ADD/ADHD	YES	NO
Depression/Anxiety	YES	NO
Sleep Disturbance	YES	NO
Prescription Medication	YES	NO

APPENDIX B.

National Sleep Foundation Sleep Diary

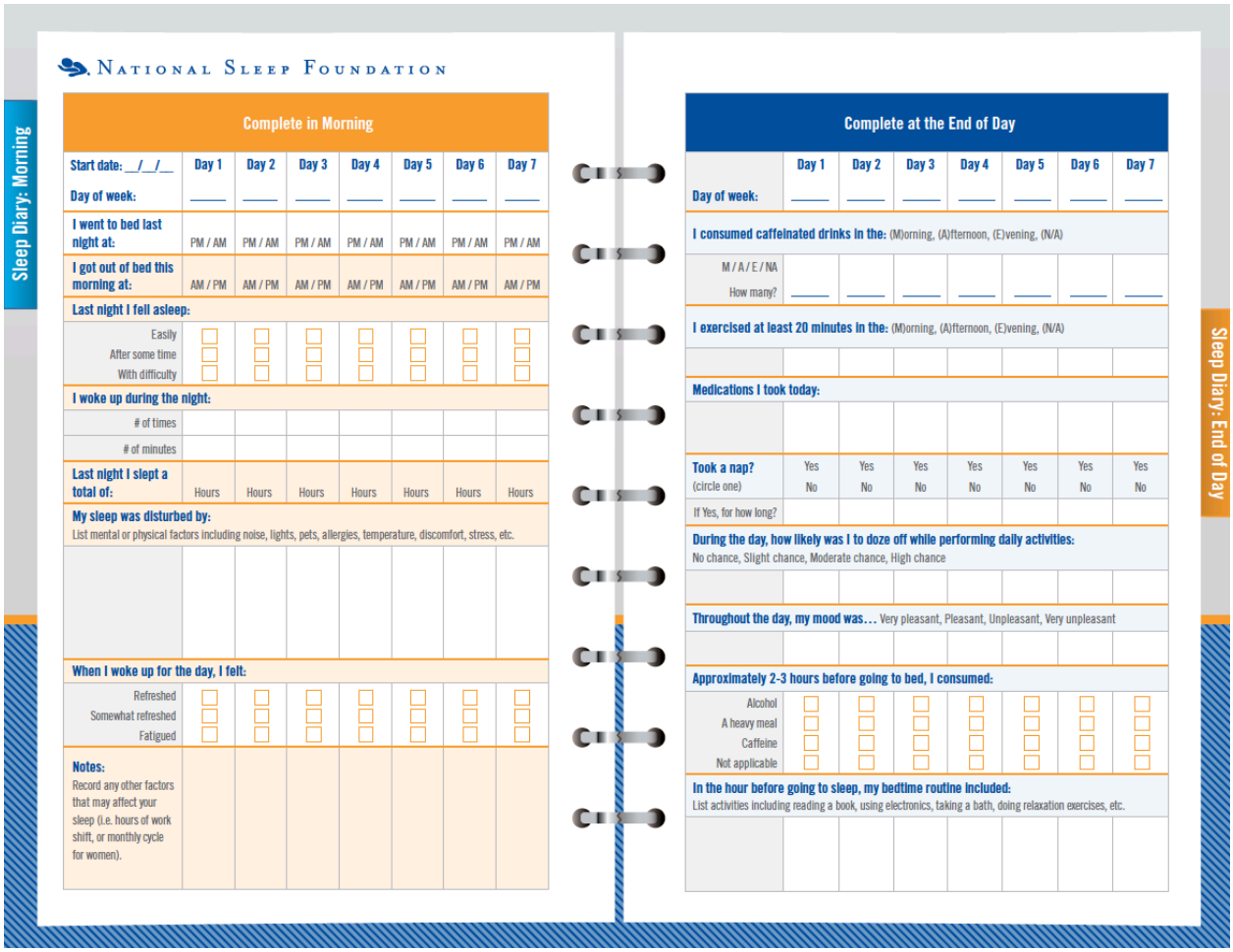


Figure 7. National Sleep Foundation Sleep Diary.

APPENDIX C.

Pittsburgh Sleep Quality Index (PSQI)

Subject's Initials_____ID#_____Date_____Time_____AM PM

PITTSBURGH SLEEP QUALITY INDEX

INSTRUCTIONS:
The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, what time have you usually gone to bed at night?

   BED TIME _____

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

   NUMBER OF MINUTES _____

3. During the past month, what time have you usually gotten up in the morning?

   GETTING UP TIME _____

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)

   HOURS OF SLEEP PER NIGHT _____

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you . . .

a) Cannot get to sleep within 30 minutes

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

b) Wake up in the middle of the night or early morning

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

c) Have to get up to use the bathroom

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

d) Cannot breathe comfortably

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

e) Cough or snore loudly

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

f) Feel too cold

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

g) Feel too hot

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

h) Had bad dreams

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

i) Have pain

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

j) Other reason(s), please describe_____

_____

How often during the past month have you had trouble sleeping because of this?

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

6. During the past month, how would you rate your sleep quality overall?

Very good _____

Fairly good _____

Fairly bad _____

Very bad _____

7. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all _____

Only a very slight problem _____

Somewhat of a problem _____

A very big problem _____

10. Do you have a bed partner or room mate?

No bed partner or room mate _____

Partner/room mate in other room _____

Partner in same room, but not same bed _____

Partner in same bed _____

If you have a room mate or bed partner, ask him/her how often in the past month you have had . . .

a) Loud snoring

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

b) Long pauses between breaths while asleep

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

c) Legs twitching or jerking while you sleep

Not during the past month_____	Less than once a week_____	Once or twice a week_____	Three or more times a week_____

d) Episodes of disorientation or confusion during sleep

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

e) Other restlessness while you sleep; please describe_____

_____

Not during the past month_____ Less than once a week_____ Once or twice a week_____ Three or more times a week_____

Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ: Psychiatry Research, 28:193-213, 1989.

APPENDIX D.

Morningness-Eveningness Questionnaire – Short Assessment (MEQ-SA)

MORNINGNESS-EVENINGNESS QUESTIONNAIRE
Self-Assessment Version (MEQ-SA)[1]

Name: _____ Date: _____

For each question, please select the answer that best describes you by circling the point value that best indicates how you have felt in recent weeks.

1. *Approximately* what time would you get up if you were entirely free to plan your day?

   [5] 5:00 AM–6:30 AM *(05:00–06:30 h)*
   [4] 6:30 AM–7:45 AM *(06:30–07:45 h)*
   [3] 7:45 AM–9:45 AM *(07:45–09:45 h)*
   [2] 9:45 AM–11:00 AM *(09:45–11:00 h)*
   [1] 11:00 AM–12 noon *(11:00–12:00 h)*

2. *Approximately* what time would you go to bed if you were entirely free to plan your evening?

   [5] 8:00 PM–9:00 PM *(20:00–21:00 h)*
   [4] 9:00 PM–10:15 PM *(21:00–22:15 h)*
   [3] 10:15 PM–12:30 AM *(22:15–00:30 h)*
   [2] 12:30 AM–1:45 AM *(00:30–01:45 h)*
   [1] 1:45 AM–3:00 AM *(01:45–03:00 h)*

3. If you usually have to get up at a specific time in the morning, how much do you depend on an alarm clock?

   [4] Not at all
   [3] Slightly
   [2] Somewhat
   [1] Very much

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[1]Some stem questions and item choices have been rephrased from the original instrument (Horne and Östberg, 1976) to conform with spoken American English. Discrete item choices have been substituted for continuous graphic scales. Prepared by Terman M, Rifkin JB, Jacobs J, White TM (2001), New York State Psychiatric Institute, 1051 Riverside Drive, Unit 50, New York, NY, 10032. January 2008 version. Supported by NIH Grant MH42931. *See also:* automated version (AutoMEQ) at www.cet.org.

Horne JA and Östberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. International Journal of Chronobiology, 1976: 4, 97-100.

4. How easy do you find it to get up in the morning (when you are not awakened unexpectedly)?

   [1] Very difficult
   [2] Somewhat difficult
   [3] Fairly easy
   [4] Very easy

5. How alert do you feel during the first half hour after you wake up in the morning?

   [1] Not at all alert
   [2] Slightly alert
   [3] Fairly alert
   [4] Very alert

6. How hungry do you feel during the first half hour after you wake up?

   [1] Not at all hungry
   [2] Slightly hungry
   [3] Fairly hungry
   [4] Very hungry

7. During the first half hour after you wake up in the morning, how do you feel?

   [1] Very tired
   [2] Fairly tired
   [3] Fairly refreshed
   [4] Very refreshed

8. If you had no commitments the next day, what time would you go to bed compared to your usual bedtime?

   [4] Seldom or never later
   [3] Less that 1 hour later
   [2] 1-2 hours later
   [1] More than 2 hours later

9. You have decided to do physical exercise. A friend suggests that you do this for one hour twice a week, and the best time for him is between 7-8 AM *(07-08 h)*. Bearing in mind nothing but your own internal "clock," how do you think you would perform?

   [4] Would be in good form
   [3] Would be in reasonable form
   [2] Would find it difficult
   [1] Would find it very difficult

10. At *approximately* what time in the evening do you feel tired, and, as a result, in need of sleep?

   [5] 8:00 PM–9:00 PM *(20:00–21:00 h)*
   [4] 9:00 PM–10:15 PM *(21:00–22:15 h)*
   [3] 10:15 PM–12:45 AM *(22:15–00:45 h)*
   [2] 12:45 AM–2:00 AM *(00:45–02:00 h)*
   [1] 2:00 AM–3:00 AM *(02:00–03:00 h)*

11. You want to be at your peak performance for a test that you know is going to be mentally exhausting and will last two hours. You are entirely free to plan your day. Considering only your "internal clock," which one of the four testing times would you choose?

   [6] 8 AM–10 AM *(08–10 h)*
   [4] 11 AM–1 PM *(11–13 h)*
   [2] 3 PM–5 PM *(15–17 h)*
   [0] 7 PM–9 PM *(19–21 h)*

12. If you got into bed at 11 PM *(23 h)*, how tired would you be?

   [0] Not at all tired
   [2] A little tired
   [3] Fairly tired
   [5] Very tired

13. For some reason you have gone to bed several hours later than usual, but there is no need to get up at any particular time the next morning. Which one of the following are you most likely to do?

[4] Will wake up at usual time, but will not fall back asleep
[3] Will wake up at usual time and will doze thereafter
[2] Will wake up at usual time, but will fall asleep again
[1] Will not wake up until later than usual

14. One night you have to remain awake between 4-6 AM *(04-06 h)* in order to carry out a night watch. You have no time commitments the next day. Which one of the alternatives would suit you best?

[1] Would not go to bed until the watch is over
[2] Would take a nap before and sleep after
[3] Would take a good sleep before and nap after
[4] Would sleep only before the watch

15. You have two hours of hard physical work. You are entirely free to plan your day. Considering only your internal "clock," which of the following times would you choose?

[4] 8 AM–10 AM *(08–10 h)*
[3] 11 AM–1 PM *(11–13 h)*
[2] 3 PM–5 PM *(15–17 h)*
[1] 7 PM–9 PM *(19–21 h)*

16. You have decided to do physical exercise. A friend suggests that you do this for one hour twice a week. The best time for her is between 10-11 PM *(22-23 h)*. Bearing in mind only your internal "clock," how well do you think you would perform?

[1] Would be in good form
[2] Would be in reasonable form
[3] Would find it difficult
[4] Would find it very difficult

17. Suppose you can choose your own work hours. Assume that you work a five-hour day (including breaks), your job is interesting, and you are paid based on your performance. At *approximately* what time would you choose to begin?

[5] 5 hours starting between 4–8 AM *(05–08 h)*
[4] 5 hours starting between 8–9 AM *(08–09 h)*
[3] 5 hours starting between 9 AM–2 PM *(09–14 h)*
[2] 5 hours starting between 2–5 PM *(14–17 h)*
[1] 5 hours starting between 5 PM–4 AM *(17–04 h)*

18. At *approximately* what time of day do you usually feel your best?

[5] 5–8 AM *(05–08 h)*
[4] 8–10 AM *(08–10 h)*
[3] 10 AM–5 PM *(10–17 h)*
[2] 5–10 PM *(17–22 h)*
[1] 10 PM–5 AM *(22–05 h)*

19. One hears about "morning types" and "evening types." Which one of these types do you consider yourself to be?

[6] Definitely a morning type
[4] Rather more a morning type than an evening type
[2] Rather more an evening type than a morning type
[1] Definitely an evening type

____ Total points for all 19 questions

APPENDIX E.

Habitual Sleep Variability Data Analysis

Habitual variability was calculated by calculating the standard deviation of total nighttime sleep time experienced across at least 5 nights prior to ImPACT, using the Sadeh sleep scoring algorithm.[385,402] The following formula[352] was used to calculated weighted average device-derived total nighttime sleep time, using the Sadeh sleep scoring algorithm (Equation 1). Total nighttime sleep time (minutes) was defined as the duration of nighttime sleep between the onset of sleep and the final awakening. Demographic information was calculated using frequencies, means, standard deviations, and ranges. Gender, previous concussion history, history of diagnosed sleep disturbance, and ADD/ADHD were evaluated as covariates using a series of multivariate analysis of variance (MANOVA) tests due to their effect on ImPACT performance. Any demographic variables with significant differences in ImPACT composite scores or total concussion symptom severity were used as covariates for future analyses. Sample mean and standard deviation for habitual, device-measured total sleep time (minutes) were calculated. Hierarchical linear regressions were used to investigate the influence of habitual sleep variability on baseline neurocognitive performance and total concussion symptom severity in college-aged individuals. The independent variable was habitual sleep variability (minutes), using the Sadeh sleep scoring algorithm, and the dependent variables were the four ImPACT composite scores (verbal memory, visual memory, visual motor processing speed, and reaction time) and total concussion symptom severity. Statistical significance was set at $p \leq .05$.

APPENDIX F.

Cole-Kripke Sleep Scoring Algorithm Data Analysis

Habitual, device-measured sleep duration was calculated using the weighted average device-derived total nighttime sleep time (minutes) individuals experienced across at least 5 nights prior to ImPACT, using the Sadeh and Cole-Kripke sleep scoring algorithms. A weighted average was used to account for weekday and weekend total sleep time and because some participants wore the Actigraph GT9X Link on different days and some less than 7 days.[371,372] The following formula[352] was used to calculate the weighted average device-derived total nighttime sleep time, using the Sadeh sleep scoring algorithm (Equational 1). Total nighttime sleep time (minutes) was defined as the duration of nighttime sleep between the onset of sleep and the final awakening.

Sample mean and standard deviation for Sadeh- and Cole-Kripke derived habitual, device-measured total sleep time (minutes) were calculated. A linear regression was used to examine the relationship between Sadeh sleep scored habitual, device-measured sleep duration and Cole-Kripke sleep scored habitual, device-measured sleep duration. The independent variable was habitual, device-measured sleep duration, using the Sadeh sleep scoring algorithm, and the dependent variable was habitual, device-measured sleep duration, using the Cole-Kripke sleep scoring algorithm. Statistical significance was set at $p \leq .05$.

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