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The Effect of Sleep Restriction on Declarative Memory in Individuals With Mild Traumatic Brain Injury

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THE EFFECT OF SLEEP RESTRICTION ON DECLARATIVE MEMORY IN INDIVIDUALS WITH MILD TRAUMATIC BRAIN INJURY

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Aim: This study investigated whether or not partial chronic sleep restriction would unmask residual memory deficits in individuals with a history of a mild TBI. It was hypothesized that 1) there would be a significant difference in performance on attention and memory tasks between the control group and the TBI group, and 2) there would be a significant difference in performance on attention and memory tasks between individuals with one TBI and individuals with multiple TBI.

Method: A total of 42 undergraduate students from the Indiana University of Pennsylvania were enrolled in the study. Data from only 30 subjects were analyzed after 12 subjects were excluded due to non-compliance with sleep restriction requirements. Subjects in the control (no mild TBI) and experimental (at least one mild TBI) groups were administered the Degraded Stimulus – Continuous Performance Test (DS-CPT) and the Rey Auditory Verbal Learning Test (RAVLT) at baseline and after restricting their sleep to six hours per night over four nights. All subjects were instructed to utilize the SleepTime smartphone application to collect objective sleep data each night leading up to their second testing session.

Results: Results of a mixed multivariate analysis (MANOVA) revealed non-significant findings at $p < .05$ (Wilks $\lambda=.892$, $F [7, 22]=.381$, $p=.903$, $\eta^2=.108$). A meaningful statistical analysis could not be run to test the second hypothesis due to an inadequate sample size, variability of sample size per cell, and insufficient power.

Conclusion: The first hypothesis was not supported as there was no statistically significant difference in attention and memory outcomes at baseline and post-test between the control group and mild TBI group. As noted above, the second hypothesis could not be tested. Findings from this study are consistent with previous literature on TBI recovery that suggests individuals with one or more mild TBI typically recover cognitively within three to four months of the injury. Implications, limitations, and future directions are discussed.

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CHAPTER ONE

INTRODUCTION

Study Aims

This study focuses on recovery from mild traumatic brain injury (TBI) and potential latent deficits that may not manifest unless they are unmasked following exposure to a physiological stressor. Recovery from a mild TBI is generally expected to occur within one month (McCrea, 2003). Among high school and college students, a meta-analysis review found that only seven to ten days may be necessary for recovery based on reported symptoms and performance on cognitive testing (Williams et al., 2015). Despite seeming resolution of sequelae related to the injury, as many as 30% to 70% of individuals who experience a mild TBI report sleep disturbances beyond the expected recovery period (Ouillet, Savard, & Morin, 2004). Studies also found that sleep disturbances were associated with deficient hormone regulation in individuals with mild TBI (Baumann et al., 2005; Baumann et al., 2007). Given that deficient hormone regulation may interfere with TBI recovery and sleep plays a significant role in hormone regulation, the literature suggests that sleep deprivation is a unique form of stress that has the potential to unmask residual cognitive deficits following a mild TBI.

Mild Traumatic Brain Injury, Sequelae, and Recovery

A TBI generally occurs as a result of three different kinds of trauma events. An individual should be screened for traumatic brain injury after blunt trauma to the head, an acceleration or deceleration force, or exposure to a blast (VA/DoD, 2009). Despite variability in assessment of traumatic brain injuries by different facilities, Table 1 illustrates the common factors that are evaluated for determining presence and severity. A common factor in the assessment of traumatic brain injury is that symptoms of altered consciousness or mental state

must occur immediately after the head trauma and typically include confusion, disorientation, difficulty thinking clearly or responding appropriately to mental status questions, and the inability to describe events that occurred immediately prior to or after the trauma event.

The literature has used the terms "concussion" and "mild traumatic brain injury" interchangeably (Wood, 2004). For consistency purposes, the term "mild traumatic brain injury" will be used for this research study. At this time, the most commonly used diagnostic screening criteria used at this time is the one provided by the American College of Rehabilitation Medicine (1993), which states that a mild TBI is "a physiological disruption of brain function as a result of a traumatic event as manifested by at least one of the following: alteration of mental state, loss of consciousness (LOC), loss of memory or focal neurological deficit, that may or may not be transient; but where the severity of the injury does not exceed the following: post-traumatic amnesia (PTA) for greater than 24 hours, after the first 30 minutes Glasgow Coma Score (GCS) 13 - 15, and loss of consciousness is less than 30 minutes."

Table 1

Classification of TBI Severity

Criteria	Mild	Moderate	Severe
Structural imaging	Normal	Normal or abnormal	Normal or abnormal
Loss of consciousness	0-30 min	>30 min and <24 hrs	>24 hrs
Alteration of consciousness/mental state	0-24 hrs	>24 hrs	>24 hrs
Posttraumatic amnesia	0-1 day	>1 and <7 days	>7 days
Glascow Coma Scale	13-15	9-12	<9

VA/DoD Clinical Practice Guideline for Management of Concussion/Mild Traumatic Brain Injury (mTBI). Retrieved from http://www.healthquality.va.gov/mtbi/concussion_mtbi_full_1_0.pdf

Although somatic, cognitive, and behavioral symptoms generally dissipate after two to four weeks, people who experience a mild TBI may also experience "post-concussion syndrome" (PCS) (McCrea, 2003). PCS refers to the presence of symptoms that occur at least one to three months after the mild TBI and consists of at least two persistent nonfocal, neurologic symptoms, such as dizziness, headache, cognitive deficits, behavioral changes, mood changes, and/or sleep disturbance. There have been two theories proposed about post-concussive symptoms (Alexander, 1997). Psychogenesis is when PCS is a reflection of psychological factors that are premorbid or post-injury. On the other hand, physiogenesis asserts that mild TBI can lead to significant neuropathology that then causes PCS through alteration in the brain. Most PCS that occur after mild TBI have been found to resolve overall approximately three months post injury (Fay et al., 2010). Complicated versus uncomplicated mild traumatic head injuries and premorbid cognitive abilities can predict residual PCS after three months (Fay et al., 2010). Complicated mild TBI refer to TBIs with abnormal findings on neuroimaging, such as the presence of a skull fracture and/or intracranial abnormalities such as hemorrhage, contusion, or edema (Iverson et al., 2012).

Despite the name and its association with concussions, it is difficult to accurately identify the etiology of PCS due to inconsistencies between the most commonly used definitions provided by the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV-TR, 2000) and the International Statistical Classification of Diseases and Related Health Problems – 10th Revision (ICD-10). In addition to a poor correlation between the two definitions, one study found that the definitions provided by the ICD-10 was more suitable one month after the mild TBI but inaccurate after 3 months (Boake et al., 2004; Kashluba et al., 2006). Therefore, rather than assume that PCS is exclusive to mild TBIs, it is important for clinicians to consider

alternative etiologies behind PCS for appropriate intervention. For instance, studies suggest that a strong correlation between perception of sleep quality and psychiatric symptoms may better explain symptoms of PCS than mild TBIs (Polusny et al., 2011; Rona et al., 2012). Examples of other etiologies that may be associated with PCS include medical conditions, such as chronic pain, fibromyalgia, and pharmacological side-effects (Iverson, 2003; Iverson, 2007).

Another difficulty in assessing symptoms and recovery of mild TBIs involves structural deficits that cannot always be detected by neuroimaging techniques. This can be particularly problematic when, despite no clear brain lesions that are visible through neuroimaging, some people with mild TBI report clearing of symptoms within three months while other individuals report physical, cognitive, and emotional symptoms more than one year post-injury (Witt, Lovejoy, Pearlson, & Stevens, 2010). Examples of cognitive and emotional symptoms after a single mild TBI include a decline in memory, attention, concentration, executive function, depression, and anxiety (Bergman & Bay, 2010). In a study looking at changes in brain default network during resting state, data suggest that individuals with mild TBI have a reduced number and strength of connections in the posterior cingulate and lateral parietal cortices, an increased number and strength of connections in the medial prefrontal cortex, and an overall loss of neural connectivity with increasing number of mild TBIs (Johnson et al., 2012). According to fMRI studies, the posterior cingulate and lateral parietal cortex have been found to play an important role in episodic memory retrieval (Cabeza, Ciaramelli, Olson, & Moscovitch, 2008; Uncapher & Wagner, 2009; Wagner, Shannon, Kahn, & Buckner, 2005).

One study investigated the relationship between severe TBI and declarative memory impairment using the Rey Auditory Verbal Learning Test (RAVLT) as a measure of declarative memory (Palacios et al., 2013). The RAVLT consists of five trials of immediate recall in

addition to a delayed recall after being exposed to an interference word list. It evaluates episodic declarative memory, immediate memory, verbal learning, susceptibility to proactive and retroactive interferences, consolidation, retention, free recall, and recognition (Lezak, Howieson, & Loring, 2004). When performance of control subjects were compared with TBI patients, TBI patients performed significantly worse on the memory task and presented with greater brain atrophy. This is consistent with other memory studies that indicate that declarative memory is not hippocampus-dependent. In addition, the authors found a positive correlation between reduction in cortical thickness, atrophy of white matter, and deficits in declarative memory. Findings suggest that brain plasticity may partially compensate for memory deficits that result from damage to the brain. Recent literature also indicates that a reduction in medial temporal functionality may be correlated with impairments in declarative memory following a mild TBI (Bay, Kalpakjian, & Giordani, 2012; Stulemeijer et al., 2010).

CHAPTER TWO

LITERATURE REVIEW

Sleep Deprivation as a Public Health Concern

Sleep deprivation is becoming a significant problem within society. According to research conducted by the National Heart, Lung, and Blood Institute (NHLBI), as many as 50 to 70 million U.S. adults have sleep or wakefulness disorders with approximately 12 to 15 million adults diagnosed with sleep apnea. In addition, approximately one out of three Americans report getting fewer than seven hours of sleep per night, approximately one out of three Americans report sleepiness on a daily basis during daylight hours, and 70% of U.S. high school students are not getting the recommended amount of sleep on school nights (NHLBI, 2011). Beginning as early as adolescence, people begin to forsake sleep for other activities during the day or to accommodate early school start times and report a significant decrease in total sleep time (Leger et al., 2012). Sleep is a biological necessity, yet people are getting fewer hours of sleep than ever before (McKnight-Eily et al., 2009).

Given the various necessary functions of sleep, sleep deprivation has been found to carry a multitude of consequences. For instance, epidemiological studies indicate that sleep deprivation is associated with chronic health problems such as obesity, diabetes, hypertension, and even mortality (Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008). The seemingly practical solution of reducing sleep in order to increase productivity during the day makes it difficult to keep in mind the long-term consequences of poor sleep quality and sleep deprivation on performance during the day. Perhaps due to discrepancies between objective and subjective measures of sleep per night, cognitive impairments associated with sleep deprivation have led to an increase in injuries and deaths via vehicular accidents (Horne & Reyner, 1995). Studies by the

NHLBI indicate that as many as 5,000 to 6,000 fatal vehicular accidents each year may be caused by drowsy drivers.

In addition to this growing problem of sleep deprivation, the separate issue of concussions has been gaining attention. According to a report released by the Center for Disease Control and Prevention (2011), there has been a 60% increase over a span of 8 years in emergency room visits by children and adolescents for brain injuries. Among individuals 19 years and older, the report also found that there was an increase in emergency room visits from 153,375 in 2001 to 248,418 in 2009. These numbers exclude brain injuries in the adult population from contact sports, vehicle accidents, and falling. Approximately 300,000 sports-related concussions alone occur annually within the U.S., and there is great risk for serious sequelae of concussions (Guskiewicz et al., 2003). In 2008, approximately 320,000 soldiers in Iraq and Afghanistan reported at least one traumatic brain injury, most of which were mild (Rona, 2012). Unfortunately, many concussions go unreported and untreated due to a lack of consensus in diagnostic criteria. The seriousness of concussions is minimized in comparison to moderate or severe traumatic brain injuries where physical and cognitive impairments are generally more apparent. The following sections provide an overview of the literature on sleep studies, long-term memory, mild traumatic brain injuries, as well as the relations between sleep and memory in individuals with a history of head injuries.

Sleep and Neurobehavioral Functioning

The concept of sleep has changed over the years. Sleep was assumed to be an unconscious state that people enter for a period of time before they regain consciousness. However, research indicates that there is actually a great degree of activity that occurs during sleep. For example, there are various biological functions that change and occur during sleep.

The endocrine system is controlled by the circadian rhythm, which influences secretion of hormones such as growth, follicle stimulating, and luteinizing hormones (National Institutes of Health, 2007). Different brain structures influence the function of sleep as well. For instance, researchers have found that the pons plays a heavy role in initiating and modulating rapid eye movement (REM) sleep (Brankačk et al., 2012; Ramaligam, Chen, Saper, & Lu, 2013). In addition, it also sends signals to the spinal cord in order to temporarily inhibit movement during sleep. The basal forebrain, on the other hand, is responsible for non-rapid eye movement (NREM) stages of sleep (Lelkes, Porkka-Heiskanen, & Stenberg, 2013; Mallick & Kumar, 2012).

Furthermore, sleep is believed to be a homeostatically regulated behavior (Benington, 2000). In other words, a negative feedback loop occurs that involves cellular and molecular changes within the brain and body. Evidence for homeostatic regulation includes extended sleep loss that is followed by cognitive and behavioral impairments in addition to longer durations of sleep (Porkka-Heiskanen, 2013). In addition to the homeostatic process, there are two other processes that underlie the regulation of sleep: the circadian process and the ultradian process (Borbely & Achermann, 1999). This two-process model of sleep was first proposed by Borbely using data gathered from sleep regulation in rats, which was later applied to sleep regulation in humans. He proposed that homeostatic regulation increases during the wakefulness state and declines during sleep, which activates the circadian regulation. Contrary to previous hypotheses, these two seemingly antagonistic functions are interactive rather than mutually exclusive; this may be observed in neurobehavioral functioning when sleep debt becomes a factor in the interaction between homeostatic regulation and circadian regulation (Daan et al., 1984; Pomplun et al., 2012). For example, one study found that memory performance was unexpectedly

jeopardized in the early morning when wakefulness was less than 16 hours, whereas it was spared in the evenings despite more than 24 hours of wakefulness combined with partial sleep deprivation (Lo et al., 2012; Zhou et al., 2011).

Stages of Sleep

In addition to playing a role in sleep regulation, the ultradian process occurs within each sleep episode. It regulates the switch between the REM and NREM sleep stages, which are detected by electrophysiological signs that are measured by a polysomnography (Pace-Schott & Hobson, 2002). The NREM stages comprise approximately 75% of sleep and are further divided into stages one, two, three, and four. The NREM stages exhibit increasing low-frequency electroencephalographic (EEG) activity across the stages with what are called *sleep spindles* and K-complex waveforms characteristic of stage two. The deepest sleep occurs in stages three and four, which are also referred to as "slow wave sleep" (SWS). An average of four to five cycles of REM and NREM sleep occur per night with each cycle lasting approximately 90 to 110 minutes. Thus, the sleep stages in conjunction with the interaction of the circadian and ultradian processes provide a parsimonious overview of the physiological processes involved in sleep. This leads to the next question of how much of it is necessary for optimal functioning.

Total Versus Partial Sleep Deprivation

According to Van Dongen, Rogers, and Dinges (2003), sleep is separated into two types of sleep: core (or obligatory) sleep and optional sleep. Core sleep refers to SWS required for physical recovery in addition to sustenance of adequate wakefulness during the day and to minimize sleep debt (Horne, 1988). Sleep debt is a result of prior sleep-wake history that involves sleep loss over the span of more than one day (Banks et al., 2010). On the other hand, optional sleep may or may not contribute to daytime sleepiness, and it does not lead to sleep

debt. In a study by Horne (1995), cognitive deficits were detected after obtaining only four to five hours of sleep. Findings also suggest that core sleep generally consists of six hours of sleep that is of good quality. This study did not address the concept of basal sleep need, which is operationalized as "habitual sleep duration in the absence of pre-existing sleep debt" with a threshold of eight hours per night (Van Dongen, Rogers, & Dinges, 2003). The need for an average of eight hours of habitual sleep suggests that people may be even more vulnerable to moderate levels of sleep deprivation than previously presumed.

Sleep deprivation may be categorized as either total sleep deprivation or partial sleep deprivation. Total sleep deprivation is defined as the length of time since the end of the last sleep period (Bonnet & Arand, 2003). This is not to be confused with partial sleep deprivation, which is defined as the length of sleep period and chronicity of the shortened sleep schedule. Partial sleep deprivation has not been studied as much as total sleep deprivation. There are three ways partial sleep deprivation may occur (Banks & Dinges, 2007). The first is through sleep fragmentation, which disrupts the sleep stages and leads to the loss of total physiological sleep relative to the amount of time spent in bed. An example of this is obstructive sleep apnea. Selective sleep stage deprivation is the second type of sleep fragmentation, which occurs during an isolated sleep stage, such as when a medication affects a specific sleep stage. Sleep restriction, also sometimes referred to as sleep debt, is the third type of partial sleep deprivation. This occurs when sleep duration is reduced overall.

Theories and Empirical Findings on Sleep Deprivation and Cognitive Abilities

The literature on sleep continues to explore how sleep deprivation affects cognitive functioning. Theories on the relationship between sleep and cognitive abilities date as far back as 1924 when Jenkins and Dallenbach proposed that the role of sleep was to protect memory from

daytime interferences. In 1997, Plihal and Born introduced the theory that SWS promoted consolidation of episodic memory while REM sleep regulated consolidation of procedural consolidation, which contributed to a surge in neuropsychological research studying the different stages of sleep and the sleep-stage research paradigm. Another theory on sleep deprivation and cognitive abilities is the controlled attention hypothesis, which states that sleep deprivation affects the top-down cognitive processes (Lo et al., 2012). Therefore, it is when people engage in more complex tasks that they are less influenced by sleep deprivation. When people experience sleep deprivation and the tasks are not complex enough, cognitive impairments are seen as the working memory and executive functioning processes fail to get activated. This suggests that cognitive performance is affected by the type of task, circadian phase, genetic differences, and prior sleep debt.

In addition to these factors, deficits in one area of cognitive functioning after sleep deprivation may influence other cognitive abilities. For instance, the literature suggests the effect sleep deprivation has on cognitive abilities may all be strongly associated with vigilant attention (Goel, Rao, Durmer, & Dinges, 2009; Lim et al., 2010). Therefore, it is important to consider the effect sleep deprivation may have on vigilance because impaired attention may be a confounding variable when assessing other cognitive functions. The psychomotor vigilance test (PVT) in particular has been found to be very sensitive to sleep deprivation and is used within one of the most common paradigms for measuring vigilant attention (Basner & Dinges, 2011; Lim & Dinges, 2008). The PVT is a 10-minute long task that measures vigilant attention by recording the amount of time it takes to respond to visual or auditory stimuli that occur at random intervals. In sleep research, it tracks changes in behavioral alertness based on the reaction time of pressing a button at varying inter-stimulus intervals (Basner & Dinges, 2011). Use of this paradigm in

numerous studies have led to findings that suggest overall delay of responses occur after sleep deprivation, there is a significant increase in the likelihood of omission and commission errors, and there is increased variability in performance. This may be partly explained by the state instability hypothesis, which states that sleep initiating mechanisms influence one's ability to sustain attention, which leads to an "unstable state" that neither falls under the awake or asleep category and continues to fluctuate within as little as a few seconds (Doran, Van Dongen, & Dinges, 2001). In addition to vigilance, sleep deprivation has been associated with deficits in numerous other areas of cognitive functioning. For the purpose of this study, the following review will focus primarily on memory.

The Dual System Framework of Memory

Human memory is currently believed to consist of two long-term storage and retrieval systems: declarative and non-declarative (Tulving, 1985). Although there is a set of subsystems that work together for working memory, the focus of this research study will be on long-term memory. Declarative memory, also referred to as explicit memory, consists of facts and events that are consciously accessible. According to Mayes (2000), declarative memory is further divided into episodic memory (autobiographical events) and semantic memory (general knowledge). The formation of declarative memory involves explicit learning and is believed to be associated with the medial temporal lobe, particularly the hippocampus (Walker & Stickgold, 2004). In contrast, non-declarative memory includes procedural memory (habits and skills) and involves implicit learning. It requires more repetition and longer exposure to information compared to when information gets processed through declarative memory. Examples of procedural memory include motor skill learning, cognitive skill learning, and "how to" learning. Nondeclarative memory has been found to be intact in most patients with amnesia (O'Connor &

Verfaillie, 2002). Declarative memory, on the other hand, is a more complex system that is vulnerable to amnesic conditions.

There are three processing stages of declarative memory (Lezak, Howieson, & Loring, 2004). The first stage is registration memory, which refers to a level that holds sensory information and either gets transferred into working memory or it decays. If the information does not decay, it generally lasts approximately 30 seconds up to several minutes. Of note, the literature on memory is mixed when it comes to whether short-term memory and working memory are overlapping constructs or two distinct processes. Generally, short-term memory refers to brief storage of information prior to consolidation while working memory expands on the former definition to include both brief storage and mental manipulation of information (Cowan, 2008). Given the argument within the literature that the term "working memory" has replaced "short-term memory" and working memory is broadly used within experimental studies to refer to the information maintenance process rather than information manipulation, the following review of the literature on memory will use "working memory" to refer to brief storage of information prior to consolidation (Aben, Stapert, & Blokland, 2012; D'Esposito et al., 1999; Gray, 2007).

The second stage of declarative memory is immediate memory. The concept of reverberating neural circuits states that information is retained in short-term memory storage through neural networks, which maintain nerve impulses by routing them through the same network in a cycle. However, the trace of immediate memory decays and information cannot be retained long-term if the nerve impulses are not converted into a more stable biochemical organization. The duration of a memory trace may be lengthened through rehearsal of that information, which refers to a repetitive mental process (Brown & Craik, 2000). This leads to the

third stage of declarative memory, which is learning information and consolidating it into long-term storage.

Sleep and Memory Consolidation

The definition of memory consolidation has changed over time. Buzsáki (1989) originally proposed the 2-stage model of long-term memory consolidation, which states that new information is temporarily stored in the hippocampus and then transferred to the neocortex during post-learning sleep where long term consolidation of information occurs. Memory consolidation used to be defined as a process in which information becomes increasingly resistant to interference (McGaugh, 2000). This definition has now been expanded to recognize that memory consolidation occurs through stabilization and enhancement of memories. More recently, it has been hypothesized that memory as a whole is a function of memory consolidation and memory reconsolidation (Stickgold & Walker, 2005). After initial encoding of information, protein synthesis occurs for memory consolidation. The novel concept of memory reconsolidation is based on the idea that recalling a memory at a later point in time may destabilize it. Therefore, it is necessary for the memory to go through a restabilization phase or else it may weaken or become irretrievable.

As previously discussed, the hippocampus is a significant component of the memory system and particularly important for the formation of new memories as they undergo the consolidation process. One theory is that the hippocampus creates an index to assign each experience and its context to parts of the neocortex where it may be reproduced as a long-term memory when activated (Lezak, Howieson, & Loring, 2004). Therefore, damage to the hippocampus may lead to impairments in storage processes as seen in anterograde amnesia, which is the inability to learn or retain new memories.

In addition to the impact structural damage to the brain may have on memory, it has been proposed that synapses may play a role in memory function. The synaptic homeostasis hypothesis proposes that, as a consequence of learning during wakefulness, a net increase in synaptic strength occurs in many brain circuits (Tononi & Cirelli, 2012). The theory argues that sleep counteracts the increasing energetic needs of the brain related to long-term potentiation by downscaling synaptic strength during slow wave sleep to achieve brain plasticity (Born & Feld, 2012). Even a few hours of sleep restriction has been found to change the molecular composition and efficacy of synapses that prevent long-term potentiation and may help to explain changes in neurobehavioral functioning after partial and total sleep deprivation (Cirelli, 2012). However, the hypothesis is not without criticism. Limitations of this hypothesis include insufficient studies on the mechanism behind how sleep alters synaptic strength, and the role of other biological processes that may be confounding variables have not been carefully controlled (Frank, 2013).

Research findings on brain plasticity generally appear to complement the literature on changes in memory after manipulations are made to sleep. For instance, subjects were administered a visuomotor task and retested three days later (Maquet et al., 2003). They also underwent a functional MRI. Results indicated that subjects who were deprived of sleep on the first night did not exhibit any enhancement in learning nor exhibit changes in brain activity whereas subjects who slept regularly over all three nights demonstrated memory enhancement as well as an increase in brain activity in the superior temporal sulcus region.

Common Paradigms for Sleep and Memory Studies

Most research on sleep and memory fall under one of three paradigms (Waters & Bucks, 2011). The first paradigm focuses on finding associations between sleep duration and memory. Often times, these studies are epidemiological studies and may include looking at whether or not

there is a correlation between sleep duration and various components of memory. Despite the ability to use a large sample size, a limitation of this paradigm is that measures of sleep and memory are generally subjective.

Unlike the first paradigm, the second paradigm involves total sleep deprivation and uses objective measures of sleep and memory. Researchers may choose to have only a total sleep deprivation condition or they may also include a sleep recovery condition in order to observe whether or not memory functioning returns to normal after sleep duration also returns to normal. For instance, researchers may design a study to assess the effects of two nights of total sleep deprivation on an immediate recall task of words that are semantically unrelated (Zanini et al., 2012). This was followed by one night of recovery sleep after which subjects in the total sleep deprivation condition were asked to recall the words again. Results suggested that learning was evident in the total sleep deprivation group only after they had had one night of recovery sleep whereas the control group exhibited earlier learning of words.

In contrast, the third paradigm uses partial sleep deprivation in order to accumulate sleep debt. Partial sleep deprivation generally consists of three to six hours of sleep per night, and may take place from two consecutive nights up to more than a week (Waters & Bucks, 2011). However, researchers may also choose to combine or refine existing paradigms, such as having a total sleep deprivation group and a partial sleep deprivation group. In one study, healthy subjects slept in increments of 4, 6, or 8 hours for 14 consecutive nights. Results displayed impairments in behavioral alertness were the equivalent in four and six hour sleep restriction groups as the one, two, and three nights of total sleep deprivation groups (Van Dongen et al., 2003). Psychomotor vigilance and working memory also decreased. In another study, subjects were randomly placed into one of two conditions: one night of total sleep deprivation or four nights of

partial sleep deprivation that would consist of restricting sleep to four hours per night (Drummond et al., 2012). Researchers were interested to see the effect of these different sleep deprivation conditions and capacity and filtering efficiency of visual working memory. Results indicated that filtering efficiency was impaired in the total sleep deprivation group whereas no significant impairment in filtering efficiency was found in the partial sleep deprivation group, and neither group demonstrated impaired visual working memory capacity. Therefore, a conclusion that may be drawn from a study using this paradigm would be that different types of sleep deprivation impact components of visual working memory to varying degrees.

Influential Factors on Memory Consolidation

One of the reasons why common paradigms look at specific stages of sleep is because the literature thus far suggests that different types of memory may be vulnerable or active in different sleep stages. For instance, REM sleep has not been found to be actively involved in consolidation of declarative memory, but it is correlated with consolidation of procedural memory. In one study, human subjects demonstrated improvement in performance on procedural tasks such as finger-tapping, auditory perception, and visual discrimination; performance on these tasks were impaired with REM sleep deprivation (Smith, 2001). Researchers are currently debating whether sleep is directly correlated with memory consolidation or if it is an overall homeostatic recovery process. The consolidation hypothesis of sleep suggests that neuronal activity during REM sleep may become reactivated and thus be a prerequisite for memory consolidation (Brankačk, Platt, & Riedel, 2009). Evidence to support this hypothesis includes observations of an increase in sleep spindles and rapid eye movements after subjects have been exposed to procedural learning tasks, such as the Tower of Hanoi task, which suggests that

cohesive binding of neuronal activity may explain improvements in motor performance after sleep (Porte, 2005; Smith, Nixon, & Nader, 2004).

As previously mentioned, sleep spindles occur in Stage two sleep, and they appear to play a significant role in memory consolidation. One study required female subjects to learn simple motor procedural tasks (pursuit rotor, simple tracing, ball-and-cup, "Operation") and found that both Stage two sleep and sleep spindle density increased. These increases were correlated with better performance on the tasks after one week (Fogel & Smith, 2006; Fogel & Smith, 2012). In another study, male subjects were administered a face-name association test in the evening followed by a free recall and recognition test of the faces and names the next morning (Clemens, Fabo, & Halasz, 2005). It was found that retention of verbal information overnight was strongly correlated with the number of sleep spindles, specifically in the left frontocentral areas of the brain. On the other hand, retention of visual information was strongly correlated with NREM sleep and not associated with sleep spindles.

Another factor that may influence memory consolidation overnight is the individual's intent to learn the material. Sleep might erase weak or irrelevant pieces of information from our memory and even protect information from interference when engaging in multiple tasks in order to allow for adaptive memory functioning (Crick & Mitchinson, 1983; Ertelt et al., 2012; Payne, Chambers, & Kensinger, 2012). In one study, subjects learn a series of words and were informed of certain words that should be remembered versus forgotten (Rauchs et al., 2011). Afterward, the subjects in the control condition were allowed to sleep regularly for three nights while subjects in the sleep deprivation condition were totally deprived of sleep the first night and then allowed to sleep regularly the following two nights. Subjects were administered a recognition task three days after they had learned the series of words. Results revealed that larger

hippocampal activity was positively correlated with items to be remembered compared to items that should have been forgotten. Subjects who were deprived of sleep on the first night of post-learning recognized more words they had been told to forget compared to subjects who slept regularly across three nights, which suggests that sleep deprivation may interfere with the ability to reorganize relevant from irrelevant information in long-term memory.

Despite the accumulation of empirical evidence in support of sleep-dependent learning and memory consolidation, some critics have argued that changes in REM sleep, learning, and memory consolidation are confounded by stress (Walker & Stickgold, 2004). More specifically, they state that increases in activity during REM sleep is due to stress from training tasks, and decreases in performance after REM deprivation is due to stress from being deprived of sleep (Siegel, 2001). Although major antidepressants are known for suppressing REM sleep, they do not necessarily lead to memory deficits possibly due to changes in learning strategies (Watts et al., 2012). Hence, the function of REM sleep may be to promote recovery through sustained activity of the central nervous system rather than to process or consolidate memory. In addition to stress from training and extended periods of wakefulness, it is also possible that sufficient memory consolidation occurs but performance is impaired due to a decrease in alertness and sustained attention from sleep deprivation. Therefore, whether attention is intact or impaired may play a role in performance on tasks after sleep deprivation.

In addition to attentional deficits, another factor that may interfere with sleep and thus memory consolidation is traumatic brain injury. Since the neuronal synaptic circuitry and other mechanisms that regulate sleep are distributed throughout the brain, a traumatic brain injury may result in disruptions to the sleep cycle. A meta-analysis study found that 50% of people who experienced a traumatic brain injury suffered from a form of sleep disturbance in addition to

being 2 to 4 times more vulnerable to problems with sleep efficiency, excessive sleepiness, early awakenings, and nightmares (Mathias & Alvaro, 2012). However, many of these studies did not specify the severity of the traumatic brain injuries, so it remains unclear whether or not people with mild traumatic brain injuries continue to experience sleep disturbances after the acute recovery period of three months. Therefore, the next section explores current literature that focuses on mild traumatic brain injuries and symptoms associated with this particular severity level.

Study Rationale

Overall, there is growing research that suggests sleep deprivation negatively impacts memory. Additionally, the literature on mild TBI and memory is vast and clearly documents the deleterious effect of TBI on memory. However, despite real world implications, there is surprisingly limited research looking at the interaction of sleep deprivation, mild TBI, and memory. Thus far, there have been two studies that looked at a variation of all three domains. The first study implemented a sleep intervention on subjects with sleep/wake disorders after they experienced TBIs of varying severity (Wiseman-Hakes et al., 2013). Objective and subjective measures were administered to measure cognitive and mood impairments. Findings revealed that sleep/wake disorders induced by a TBI appeared to exacerbate cognitive and mood impairments, but they improved after subjects participated in the sleep intervention. A limitation of this study is that the sample size was very small with only two of the subjects having experienced mild TBI. In addition, it looked at the role of sleep disorders on cognitive and mood impairments, but failed to address the effects of partial sleep deprivation in the absence of a sleep disorder.

The other study examined the effects of sleep on cognitive functioning of veterans with mild TBI who were seen at the Veterans Affairs for a neuropsychological assessment (Waldron-

Perrine et al., 2012). Similar to the previous study, researchers found that depression and anxiety were significantly correlated with subjective reports of sleep satisfaction and cognitive functioning. However, when the two variables were controlled, the objective number of hours slept was independently correlated with memory. One of the limitations of this study is that sleep satisfaction and number of hours slept were gathered through subjective measures. Therefore, given discrepancies that exist between subjective and objective measures of sleep, it remains unclear whether or not sleep deprivation is correlated with memory among individuals with mild TBI. Additionally, the study failed to screen subjects for PTSD and other psychopathology. Although previous studies suggest that there is a stronger correlation between psychological conditions (e.g., depression, anxiety) and PCS compared to the correlation between mild TBIs and PCS, researchers have acknowledged that subtle cognitive deficits may remain long after a TBI and independent of psychopathology (Clarke, Genat, & Anderson, 2012). Therefore, this study aims to refine the methodology of previous studies by excluding subjects who have psychopathology to control for the effect of comorbidity on memory.

In addition, there are inconsistent findings on the relationship between the number of mild TBIs and recovery duration. The National Collegiate Athletic Association conducted a study using a cohort of 2,905 football players from 25 U.S. colleges to measure the type and duration of symptoms as well as any sequelae of concussions and the course of recovery (Guskiewicz et al., 2003). They found that the occurrence of three or more mild TBIs was associated with slower recovery and a significantly higher likelihood of future concussions. On the other hand, a well-controlled study by the National Football League contradicted the previous results and found that there was no such relationship between number of brain injuries and duration of recovery (Pellman et al., 2004). Mixed findings may be due to confounding variables

as well as the subjectivity of self-reports. Therefore, considering the increasing occurrence of mild TBIs within society and the need for appropriate intervention, it is important to identify if and how the accumulation of mild TBIs correlates with recovery rates. This study will address the limitation from previous studies by including objective measures of sleep and cognitive functioning.

The purpose of this study is to study whether or not residual memory impairments may be detected after individuals with a history of one or more mild TBIs are partially deprived of sleep. In other words, are there potential deficits that remain after a TBI that are in a sense unmasked by partial sleep deprivation? There is some evidence to support such a concern. Asymptomatic patients with a history of mild TBI were found to demonstrate impairments in short-term visual memory when the body was placed under stress, such as normobaric hypoxic stress to mimic oxygen levels at certain altitudes (Temme et al., 2013). Short-term visual memory returned to normal in the mild TBI group once the group was returned to normal conditions, which suggests that undetected residual effects of mild TBI may be fairly common. This study will use the sleep paradigm of partial sleep deprivation to accumulate sleep debt prior to measuring sustained attention and declarative memory (Waters & Bucks, 2011). Mild TBIs appear to have a stronger correlation with attentional difficulties than with learning or other memory difficulties (Jamora, Young, & Ruff, 2012; Sinclair et al., 2013). Therefore, sustained attention will also be measured in order to assess whether memory deficits may be correlated with attentional deficits.

It is hypothesized that there will be a significant difference in attention and memory between people who have a history of one or more mild TBIs compared to people who have no history of mild TBI after they are both exposed to partial sleep deprivation. When sleep is

restricted to 6 hours of sleep, it is likely that cognitive decline goes undetected due to compensatory actions taken by the brain (Van Dongen et al. 2003). Therefore, it is expected that there will be no changes in attention or memory under both sleep conditions for people with no history of mild TBI. On the other hand, we would predict that partial sleep deprivation will have an impact on attention, memory, or both cognitive abilities for people with a history of one or more mild TBIs based on research that suggests TBI patients need longer sleep durations than non-TBI patients (Sommerauer et al., 2013), and asymptomatic individuals demonstrate memory deficits when taken outside of normal conditions (Temme et al., 2013).

Although the literature consists of mixed findings, some studies support the argument that a sequelae of mild TBI results in more residual cognitive effects and slower recovery (Guskiewicz et al., 2003; Kontos et al., 2013). Therefore, it is also hypothesized that a higher number of reported mild TBIs will be associated with worse performance on attentional and memory tasks following partial sleep deprivation compared to people who report a history of only one mild TBI.

CHAPTER 2

METHOD

Subjects

After running a power analysis, it was determined that a small (0.3) to moderate (0.5) effect size could be achieved using a sample size of 34 to 50 subjects. Subjects of consenting age were recruited from the Introduction to Psychology courses through the Indiana University of Pennsylvania (IUP) subject pool office over the spring 2013 and fall 2014 academic semesters. The majority of subjects who completed the study by the end of the spring semester in 2013 fell in the control group. Therefore, IRB approval was obtained for a modification request to complete the same procedure during the fall semester in 2014 for only subjects who reported at least one mild TBI.

By the end of data collection, a total of 42 subjects enrolled in the study. Out of the 42 eligible subjects who enrolled in the study, data analysis was conducted using a total of 30 subjects. The 12 subjects were excluded from data analysis due to failure to meet sleep restriction requirements. Of the 12 subjects who were excluded for having difficulty following the sleep restriction requirements using their smartphones, six of the subjects did not have a history of mild TBI and six of the subjects reported a history of one or more mild TBI. According to a meta-analysis review, high school and college students demonstrated recovery from a mild TBI in as little as seven to ten days (Williams et al., 2015). All of the subjects in the TBI condition reported having experienced their head injuries at least one month ago. Therefore, based on the literature and given the college population, it is assumed that no acute sequelae would confound the data. A chi-squared test was run for the categorical variables (i.e., sex and race) between the two groups, and an independent t-test was run for the continuous variables

(i.e., age, education, and WRAT scores). None of the demographic variables revealed a significant difference between the control group and the TBI group at $p < 0.05$. Subjects' demographic information and comparisons of demographic variables across conditions are provided in *Table 2* and *Table 3* below.

Table 2

Demographic Summary

Demographics	N (%)	Mean (SD)
Sex		
Male	20 (67%)	
Female	10 (33%)	
Age		18.87 (0.16)
Education		12.3 (0.12)
Race		
White	26 (87%)	
Black	1 (3%)	
Hispanic	1 (3%)	
Asian	0 (0%)	
Multiracial	2 (7%)	
TBI		
None	12	
One	11	
Multiple	7	
WRAT		44.97 (5.30)

Table 3

Demographic Variables Across Two Groups

Demographics	*t* value	χ^2 value	*p* value
Age	-.573		.571
Education	-1.782		.088
Sex		2.5	0.114
Race		2.276	0.517
WRAT	1.778		.086

Inclusion and Exclusion Criteria

Inclusion criteria were 1) individuals who owned an iPhone or Android smartphone with operating systems that allowed for utilization of the Sleep Time application, and 2) either no history of a TBI or at least one mild TBI. Given the absence of objective data related to the head injury to help categorize the severity of the TBI (e.g., neuroimaging, medical records, or GCS score), criteria for a mild TBI would be considered met if it was reported that, following an acute event, there was altered mental status (e.g., dazed or saw stars) for no more than 24 hours, LOC under 30 minutes, and/or posttraumatic amnesia for no more than one day. Exclusion criteria were 1) non-fluent in English or English is a second language, 2) a known serious medical condition, and 3) current use of prescription psychotropic medications. Major antidepressants, such as monoamine oxidase inhibitors, tricyclic antidepressants, and selective serotonin reuptake inhibitors, have been found to reduce REM sleep, which may potentially confound performance on cognitive tasks (Smith, 2001).

Materials and Measures

Objective Sleep Measure

The literature indicates discrepancies in objective measures of sleep and subjective reports of sleep and sleepiness. For instance, a large scale study called the Coronary Artery Risk Development in Young Adults Study collected sleep measurements from eligible subjects using a wrist actigraphy, a sleep log, and questions about usual sleep duration (Lauderdale et al., 2008). Despite the average objective measure of sleep being six hours, the subjective reports were approximately 0.80 hours longer than the objective measures. There was a correlation of 0.45 between subjective and objective sleep duration. There was also a discrepancy between objective sleep and subjective reports of sleepiness, which suggests that subjective sleepiness does not necessarily mean it is due to sleep deprivation. Therefore, this study obtained an objective measure of sleep.

Objective measure of sleep was obtained using a smartphone application called Sleep Time. Smartphone applications are becoming a daily part of life and are frequently utilized for everyday functioning, such as physical activity for fitness, caloric intake for weight management, and GPS navigation. More recently, engineers have begun to develop smartphone apps for the purpose of being able to measure and improve sleep. People download the app onto their smartphone and place the device near the top corner of their mattress or underneath the sheets where the device is unlikely to be moved. The accelerometer feature on the device tracks body movement to measure sleep duration and quality (Kogure et al., 2011). These mobile applications purport to measure sleep length, sleep cycles, and sleep quality comparable to that of other studied sleep measures, such as polysomnography. The technology used by mobile apps is similar to another commonly used objective sleep measure called an actigraphy, which records

body movement to identify states of sleep and wakefulness (Pollak et al., 2001). Actigraphy has been well studied and is often used as an alternative to polysomnography (Ancoli-Israel et al., 2003).

Neuropsychological Measures

According to Fay et al. (2011), premorbid intellectual functioning levels should be measured due to the possibility of cognitive reserve acting as a moderator between TBI and cognitive function post-injury. Cognitive reserve refers to the degree of resilience to cognitive impairments when exposed to neuropathology or other pathology, and it is based on factors such as intellectual activities and the environment (Barulli & Stern, 2013). A test such as the Wide Range Achievement Test-3 (WRAT-3) reading subtest is able to serve as a proxy of pre-injury intelligence. Stevens & Price (1999) found that reading tests were primarily used by neuropsychologists to obtain an estimate of premorbid intelligence in situations where it was necessary to determine both the presence and extent of changes in cognitive functioning following TBI. Test-retest reliability of the WRAT-3 reading subtest was found to be very high in a study that used a TBI population over one year ($r = 0.88$), and there was a moderate correlation with Full Scale IQ scores from the WISC-III ($r = .57$-$.66$) (Orme et al., 2004).

The Rey Auditory Verbal Learning Test (RAVLT) is one of the oldest and most studied measures of memory available for neuropsychological testing (Lezak et al., 2004). It is used to evaluate episodic declarative memory, immediate memory span, verbal learning, susceptibility to proactive and retroactive interferences, retention, free recall, and recognition of information. Administration of the RAVLT consists of a total of 5 trials and a delayed recall trial. The first 4 trials require the person to learn a word list. This is followed by one trial of learning a new word list and one trial of immediately recalling words from the original list. There is a delayed recall

of the original word list followed by a recognition task. de Sousa Magalhães, Malloy-Diniz, and Hamdan (2012) conducted a study to assess convergent and divergent validity as well as internal consistency and test-retest reliability of the RAVLT. Subjects of both genders were administered the RAVLT, and performance was compared to their performances on the Benton Visual Retention Test (BVRT) and the Trail Making Test (TMT). All test-retest correlation coefficients ranged between 0.36 and 0.68, and Cronbach's Alpha coefficient was 0.80. Although performance on the RAVLT did not significantly correlate with performance on the TMT, there were modest correlations with measures from the BVRT that ranged from 0.37 to 0.44. Overall, results suggest that the RAVLT has adequate divergent and convergent validity as well as good internal consistency.

The degraded stimulus continuous performance test (DS-CPT) was developed by Keith Nuechterlein to address the insensitivity of the basic Continuous Performance Test on people who were at risk for schizophrenia (Heinsichs, 2001). The DS-CPT involves presentation of blurred digits in the center of a computer screen where the remaining part of the screen is black. Each stimulus is presented for 42 ms with an interstimulus interval of 1 second. The task is to press the space bar of the keyboard or click the mouse as fast as possible whenever the person is presented with the digit "0," which makes up approximately 25% of all stimuli and are randomly distributed (Mass et al., 2000). Sustained attention refers to the ability to maintain focus on a stimulus over an extended period of time. It is strongly correlated with other cognitive abilities, such as information processing (Mass et al., 2000). Therefore, the DS-CPT was administered pre and post sleep deprivation in order to identify whether impairment in sustained attention plays a role in memory deficits.

Procedure

During the recruitment phase, subjects were informed that they would fulfill their entire Introduction to Psychology course research requirements (i.e., six research credits) through their participation in this study in order to provide incentive. They were pre-screened for inclusion and exclusion criteria by having them complete a demographics questionnaire (see Appendix A). Subsequently, eligible subjects were contacted by the research investigator by email with instructions on how to schedule their two study sessions. The paired time slots were posted online on the IUP Psychology Research Participant System (SONA system).

Subjects were sent an appointment reminder email two days prior to their first scheduled session stating the appointment date, time, and location in addition to a reminder to sleep eight hours each night leading up to their first session. Both of the testing sessions took place on the ground level of the Psychology Department building. When subjects arrived for their first scheduled sessions, they completed the informed consent process by reviewing the informed consent form with either the research investigator or a research assistant (see Appendix D). They were informed that they would need to restrict their sleep to six hours each night for four consecutive nights between their first session and second session. The number of nights to implement sleep restriction was based on a study by Van Dongen et al. (2003), who found that sleep restriction to six hours per night over four consecutive nights yielded changes in neurocognitive functioning. Subjects were reminded that partial chronic sleep restriction may lead to mild cognitive impairment so that they could make an informed decision about participating in the study based on individual factors (e.g., upcoming academic exams).

After completing the informed consent process, subjects were administered a demographics questionnaire, the WRAT reading subtest, and the RAVLT. In between the

RAVLT immediate and delayed recall tasks, subjects completed the DS-CPT task on the computer and were subsequently instructed by the research assistant on how to download the free version of the Sleep Time mobile application onto their phones. They were provided a handout with instructions to follow in order to correctly obtain objective measures of sleep during their participation in the study (see Appendix C). After completing the DS-CPT task and having reviewed the sleep restriction requirements over the next four nights, subjects completed the RAVLT delayed recall and recognition tasks. They were reminded of their second scheduled session at the end of the first session and also by email the day prior to the second session. The research investigator allotted one research credit for their Introduction to Psychology course on SONA.

When subjects returned for the second session, the research assistant collected the accrued objective sleep data from each subject's phone to confirm fulfillment of sleep restriction criteria by asking the subject to open the sleep application to the screen that exhibited the sleep log. Afterward, subjects were administered an alternative form of the RAVLT and re-administered the same version of the DS-CPT in between the RAVLT immediate and delayed recall tasks. After completing the DS-CPT, subjects were asked to wait outside the testing room in order to fulfill the required delay period before they were asked to return to the testing room to complete the RAVLT delayed recall and recognition tasks. At the end of their second session, subjects were given a debriefing form and the opportunity to ask questions regarding the study. The remaining five research credits for their Introduction to Psychology course were submitted by the research investigator on SONA.

As previously noted, the majority of subjects who completed the study by the end of the spring semester in 2013 fell in the control group. Therefore, IRB approval was obtained for a

modification request to complete the same procedure during the fall semester in 2014 for only subjects who reported at least one mild TBI.

Operational Definitions and Descriptive Statistics

The subjects' cognitive abilities were measured using the DS-CPT and RAVLT tests. Raw scores were used due to the homogeneity of the subjects for age and education. The operational definitions for each outcome measure and the following descriptive statistics for each group following sleep restriction are provided in *Table 4* and *Table 5* below.

Table 4

Operational Definitions for Outcome Measures

Outcome Measure	Operational Definition
Sustained attention	Sensitivity d' index on the DS-CPT
Encoding	Number of correct items from List A on Trial 1 of the RAVLT
Learning	Number of correct items from List A on Trials 1-5 of the RAVLT
Immediate Recall	Number of correct items from List A on Trial 6 of the RAVLT
Delayed Recall	Number of correct items from List A on Trial 7 of the RAVLT
Percent Retention	Number of correct items from Trial 7 divided by items from Trial 6
Recognition (True Positives)	Number of correct items from List A

Table 5

Descriptive Statistics for Control and TBI Groups at Baseline and Post-Test

	Group	Mean	SD
Pre-Sensitivity (d')	Control	1.425758	.4791001
	TBI	1.566339	.6299905
	Total	1.510107	.5697639
Post-Sensitivity (d')	Control	1.992158	.7688634
	TBI	1.830900	.5550422

	Total	1.895403	.6413110
Pre-Trial 1	Control	6.167	1.5859
	TBI	6.500	1.8550
	Total	6.367	1.7317
Post-Trial 1	Control	6.667	1.3707
	TBI	6.500	2.1213
	Total	6.567	1.8323
Pre-Learning Total	Control	49.250	7.8291
	TBI	50.444	9.2496
	Total	49.967	8.5882
Post-Learning Total	Control	50.000	7.6634
	TBI	48.056	10.9570
	Total	48.833	9.6743
Pre-Immediate Recall	Control	11.000	2.5937
	TBI	11.111	2.6321
	Total	11.067	2.5722
Post-Immediate Recall	Control	10.083	2.7122
	TBI	9.944	3.2625
	Total	10.000	3.0057
Pre-Delayed Recall	Control	11.083	2.8110
	TBI	11.333	2.7653
	Total	11.233	2.7378
Post-Delayed Recall	Control	8.833	3.2983
	TBI	9.222	3.3878
	Total	9.067	3.3003
Pre-Percent Retention	Control	100.6721	8.76331
	TBI	102.0264	11.77383
	Total	101.4847	10.52837
Post-Percent Retention	Control	87.5858	21.50338
	TBI	91.4730	21.35722
	Total	89.9181	21.13125
Pre-Recognition	Control	14.500	1.1677
	TBI	14.722	.6691
	Total	14.633	.8899
Post-Recognition	Control	13.417	1.7816
	TBI	13.556	2.0065
	Total	13.500	1.8892

CHAPTER 3

RESULTS

As previously noted, a total of 42 subjects were enrolled in the study. However, data from only 30 subjects were included in the following analyses after 12 subjects were excluded due to non-compliance with sleep restriction requirements. Specifically, during the first session, each subject was instructed to use the mobile sleep application to objectively log their total sleep time of approximately six hours each night over four nights leading up to the second session. At the beginning of the second session, these objective data were directly collected from each subject by the research assistant by logging in to the sleep application and documenting total sleep time hours and minutes per night. Subjects who were excluded from data analysis 1) did not have any objective sleep data available for the research assistant to collect from their smartphones (n=7), 2) they were missing data for one or more nights (n=3), or 3) they slept seven or more hours on at least one out of the four nights (n=2). Although they were still granted research credits for their participation in the study, it was necessary to exclude data from these subjects in order to control for compensatory mechanisms for potential cognitive reductions given that the sleep restriction paradigm from previous research suggested there was no significant impact of sleep restriction on cognitive functioning when people slept more than six hours over four nights.

Hypothesis 1

Levene's test was used to verify that the assumption of univariate normality was met for each dependent variable. Results yielded non-significance at $p < 0.5$, indicating equality of variances for sustained attention, encoding, learning, immediate recall, delayed recall, retention, and recognition (see Table 5 below).

Table 6

Levene's Test of Equality of Error Variances

	F	df1	df2	Sig.
Pre-Sensitivity (d')	0.904	1	28	.350
Post-Sensitivity (d')	3.791	1	28	.062
Pre-Trial 1	0.374	1	28	.546
Post-Trial 1	2.947	1	28	.097
Pre-Learning Total	0.898	1	28	.351
Post-Learning Total	2.417	1	28	.131
Pre-Immediate Recall	0.450	1	28	.508
Post-Immediate Recall	0.976	1	28	.332
Pre-Delayed Recall	0.007	1	28	.932
Post-Delayed Recall	0.026	1	28	.873
Pre-Percent Retention	1.020	1	28	.321
Post-Percent Retention	0.030	1	28	.863
Pre-Recognition	1.384	1	28	.249
Post-Recognition	0.011	1	28	.918

It was originally hypothesized that there would be a significant difference in sustained attention and memory outcomes between the control group and the mild TBI group following partial sleep restriction over four nights. A mixed multivariate analysis of variance (MANOVA) was run to investigate these outcomes due to the presence of a between-subjects variable (absence or presence of a mild TBI) and a within-subjects variable (baseline and post-sleep restriction) and multiple dependent variables. Contrary to the first hypothesis, there was no overall significant difference in attention and memory outcomes at baseline and post-test between the control group and mild TBI group (Wilks λ=.892, F [7, 22]=.381, p=.903, η^2=.108).

Univariate ANOVAs on Outcome Measures

Since a MANOVA rejects or accepts the null hypothesis as a whole instead of discriminating between each dependent variable, univariate ANOVAs were run to explore potential differences within and between groups. A Bonferroni correction was used to adjust the alpha level (α=0.05/7) and to address the issue of inflation of Type I error. Overall, consistent with the MANOVA, the statistical analyses of each univariate analysis of variance (ANOVA) yielded non-significant findings at α=0.007 when looking at within-group and between-group differences for each outcome measure (sensitivity d' index: $F [1,28]=1.833$, $p=.187$, $\eta^2=.061$; trial 1: $F [1,28]=.446$, $p=.510$, $\eta^2=.016$; trials 1-5: $F [1,28]=.595$, $p=.447$, $\eta^2=.021$; immediate recall: $F [1,28]=.078$, $p=.782$, $\eta^2=.003$; delayed recall: $F [1,28]=.016$, $p=.901$, $\eta^2=.001$; percent retention: $F [1,28]=.063$, $p=.804$, $\eta^2=.002$); and recognition of true positives: $F [1,28]=.013$, $p=.909$, $\eta^2=.000$).

Although there were no statistically significant findings, the mean performance within and between groups yielded subtle differences that may have been significant with a larger sample size to increase power. Graphs of the estimated marginal mean scores for each outcome measure at baseline sleep and post-sleep restriction for each group (control and TBI) are depicted in Figures 1 through 7. There appeared to be an effect of sleep restriction on both groups as indicated by a mild decline in performance post-sleep restriction on the immediate recall, delayed recall, and recognition variables. An unexpected outcome was that the TBI group demonstrated slightly better performance than the control group on the delayed recall, retention, and recognition tasks following sleep restriction. In addition, both groups performed better following sleep restriction on the sustained attention task than they did at baseline.

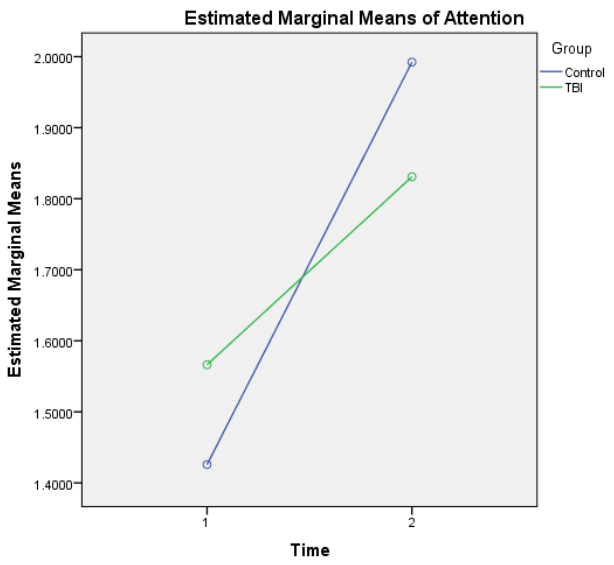

Figure 1. Estimated marginal means for attention. Pre-Test (Control: M=1.426, SE=.166; TBI: M=1.566, SE=.136) and Post-Test (Control: M=1.992, SE=.187; TBI: M=1.831, SE= .153).

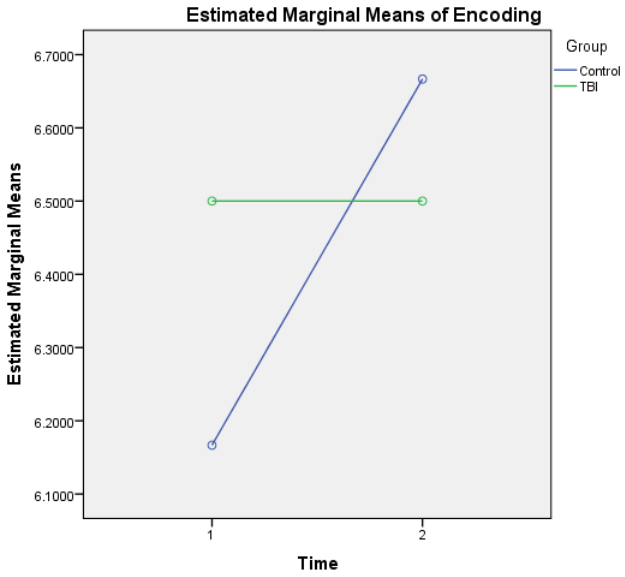

Figure 2. Estimated marginal means for encoding. Pre-Test (Control: M=6.167, SE=.506; TBI: M=6.500, SE=.413) and Post-Test (Control: M=6.667, SE=.538; TBI: M=6.500, SE=.439).

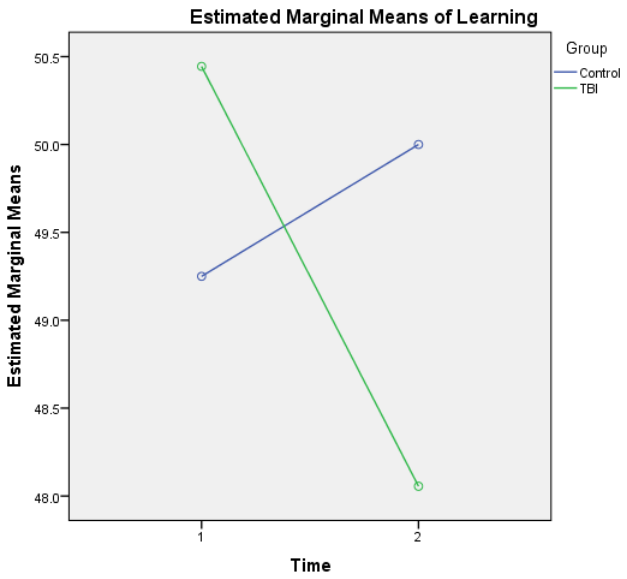

Figure 3: Estimated marginal means for learning. Pre-Test (Control: M=49.250, SE=2.517; TBI: M=50.444, SE=2.055) and Post-Test (Control: M=50.000, SE=72.828; TBI: M=48.056, SE=2.309).

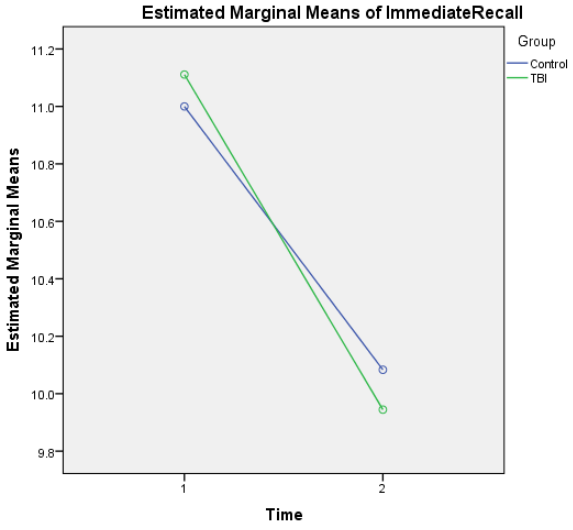

Figure 4: Estimated marginal means for immediate recall. Pre-Test (Control: M=11.00, SE=.755; TBI: M=11.111, SE=.617) and Post-Test (Control: M=10.083, SE=.883; TBI: M=9.944, SE=.721).

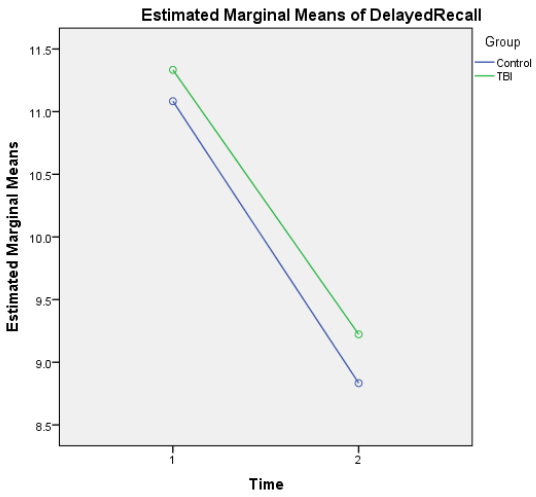

Figure 5: Estimated marginal means for delayed recall. Pre-Test (Control: M=11.083, SE=.803; TBI: M=11.333, SE=.656) and Post-Test (Control: M=8.833, SE=.968; TBI: M=9.222, SE= .790).

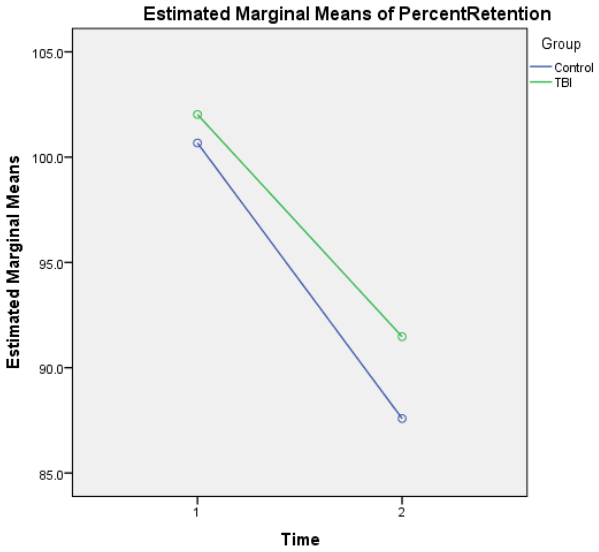

Figure 6: Estimated marginal means for percent retention. Pre-Test (Control: M=100.672, SE=3.087; TBI: M=102.026, SE=2.520) and Post-Test (Control: M=87.586, SE=6.182; TBI: M=91.473, SE=5.048).

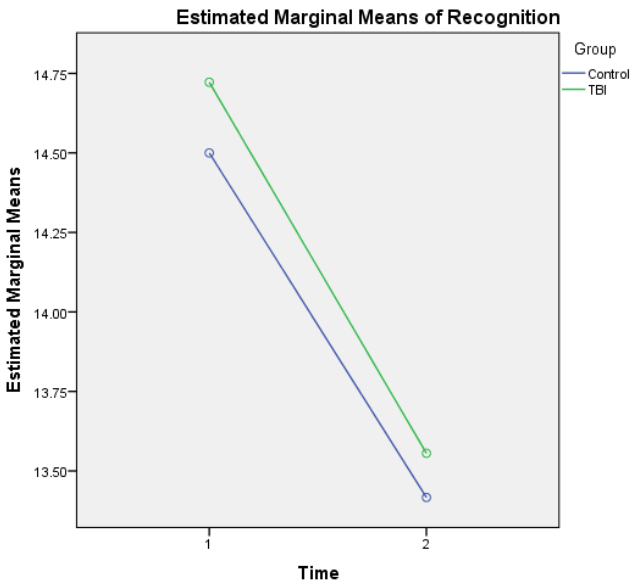

Figure 7: Estimated marginal means for recognition (hits). Pre-Test (Control: M=14.5, SE=.259; TBI: M=14.722, SE=.212) and Post-Test (Control: M=13.417, SE=.555; TBI: M=13.556, SE=.453).

Hypothesis Two

It was hypothesized that a higher number of mild TBIs would be associated with worse performance on attention and memory tasks following partial sleep deprivation compared to people with a single mild TBI. A meaningful statistical analysis could not be run to test this hypothesis due to an inadequate sample size, variability of sample size per cell, and insufficient power. Specifically, a total of 18 enrolled subjects reported one or more TBI (N=11 for a single mild TBI, N=4 for two TBI, and N=3 for three TBI). Overall, there was variability in the direction of each group with subtle differences in mean performance observed among the control group, the group with two TBI, and the group with three TBI. With the exception of encoding, the group with three TBI performed the worst following sleep restriction. There was improvement in performance for all three groups on attention and encoding tasks, and an overall

decline across all three groups on retention and recognition of true positives items. On the immediate recall task, the control group and two TBI group showed a slight improvement while the three TBI group evidenced a mild reduction following sleep restriction. On the delayed recall task, both the control and three TBI groups showed a slight decline whereas the two TBI group demonstrated a minor improvement during post-test. These differences are depicted in Figures 8 through 14.

Figure 8. Means for attention between control and multiple TBI groups.

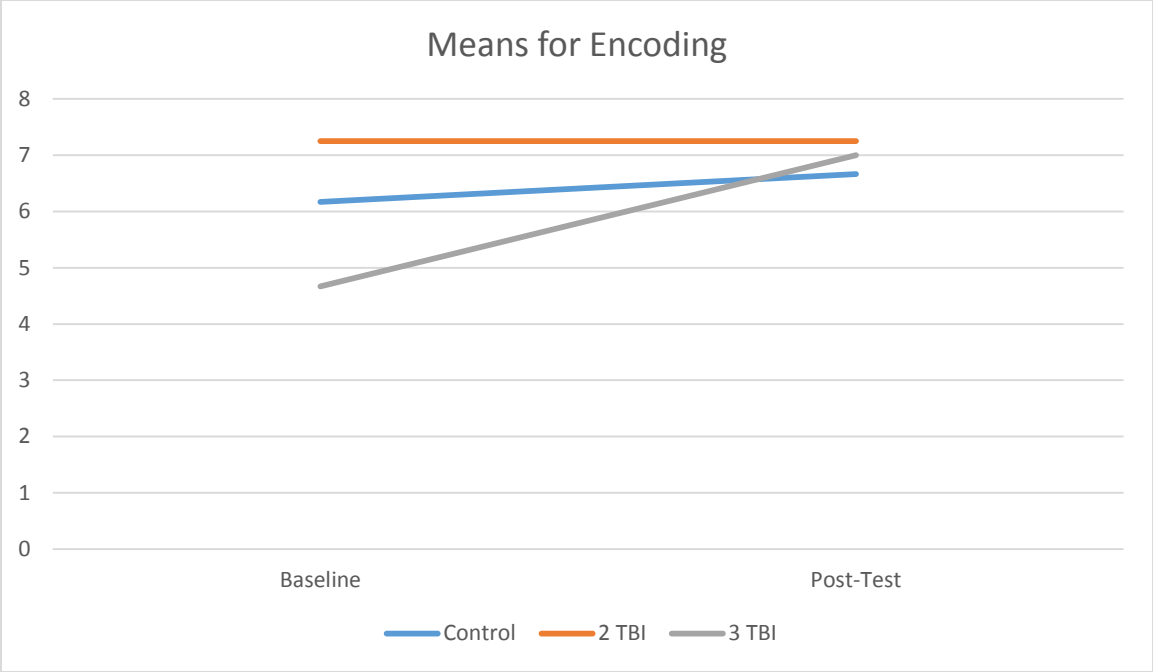

Figure 9. Means for encoding between control and multiple TBI groups.

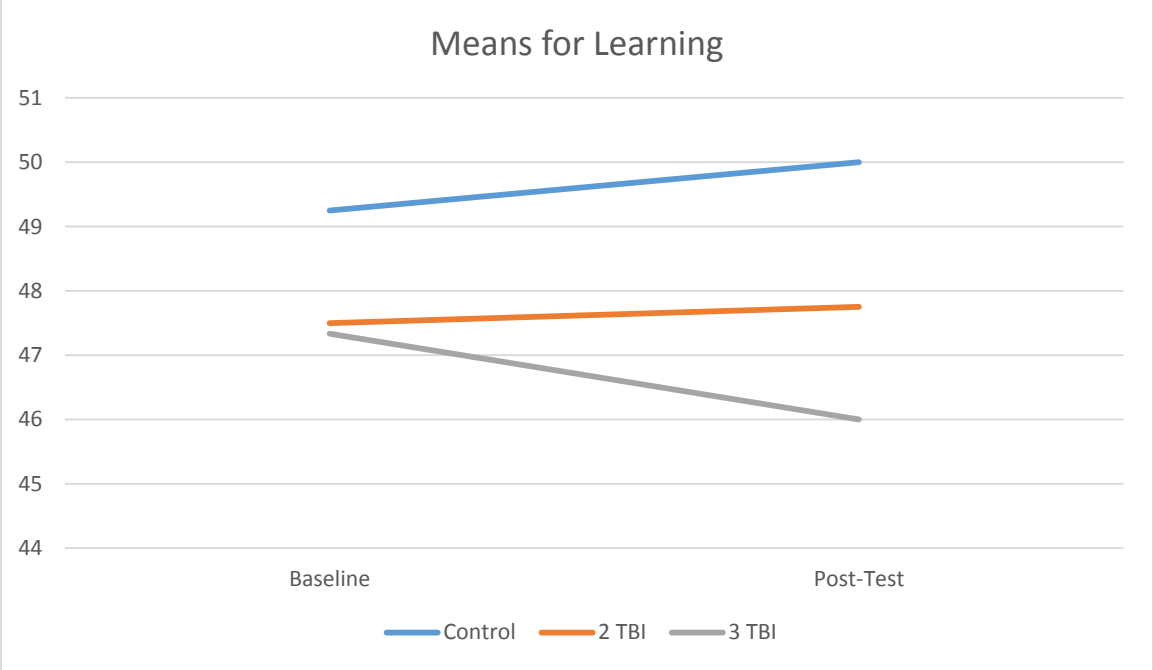

Figure 10. Means for learning between control and multiple TBI groups.

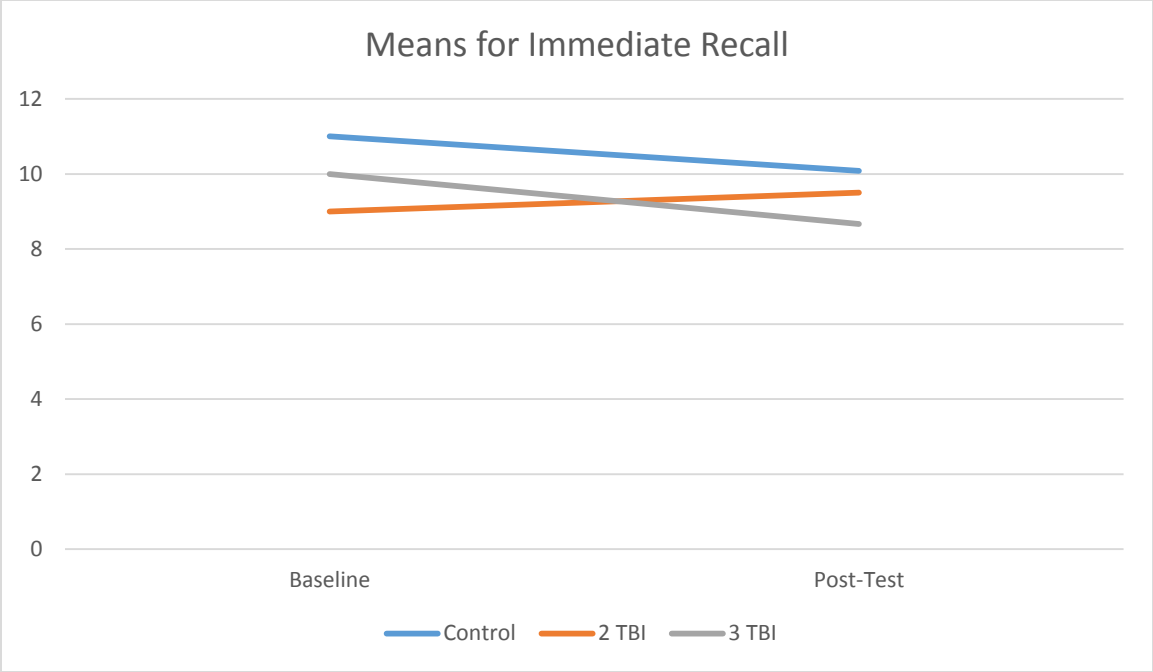

Figure 11. Means for immediate recall between control and multiple TBI groups.

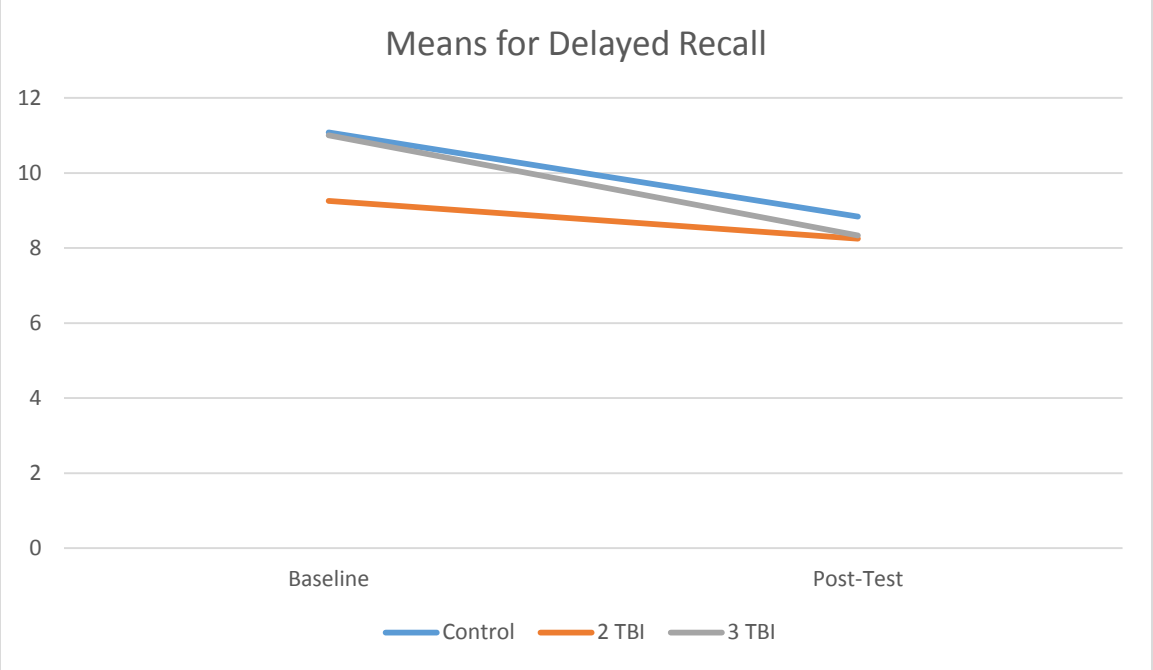

Figure 12. Means for delayed recall between control and multiple TBI groups.

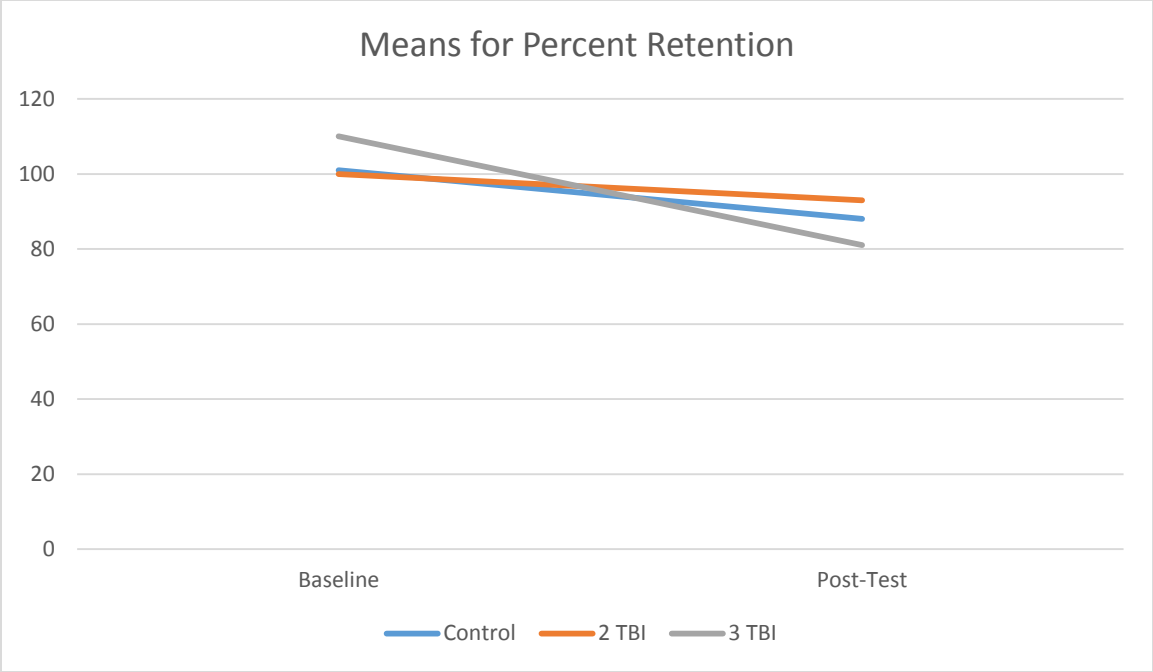

Figure 13. Means for percent retention between control and multiple TBI groups.

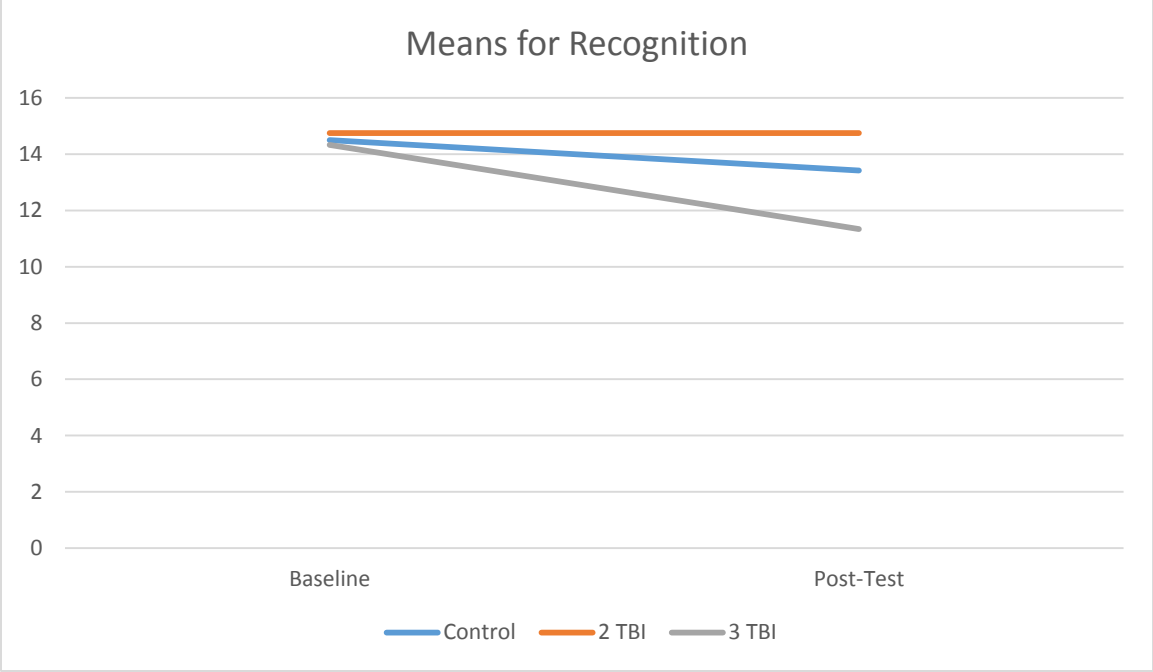

Figure 14. Means for recognition (hits) between control and multiple TBI groups.

When the TBI groups were collapsed, differences remained statistically non-significant. In summary, with the exception of sustained attention, both groups demonstrated a slight reduction in performance following sleep restriction. Regarding between-group differences, the multiple TBI group demonstrated subtly more reduced performance on memory outcomes (i.e., learning, immediate recall, delayed recall, and recognition) compared to the single TBI group. These differences are depicted in Figures 15 through 21.

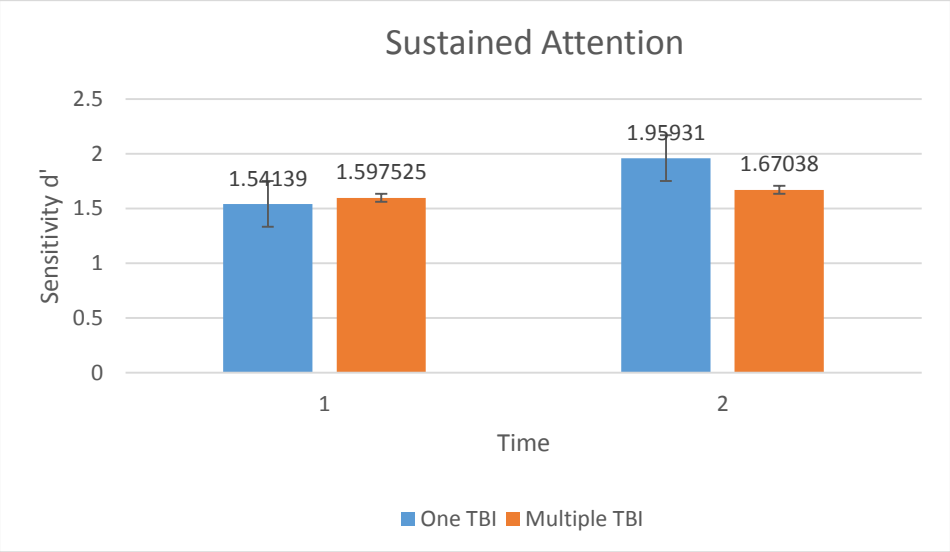

Figure 15. Means for sustained attention between single and multiple TBI groups.

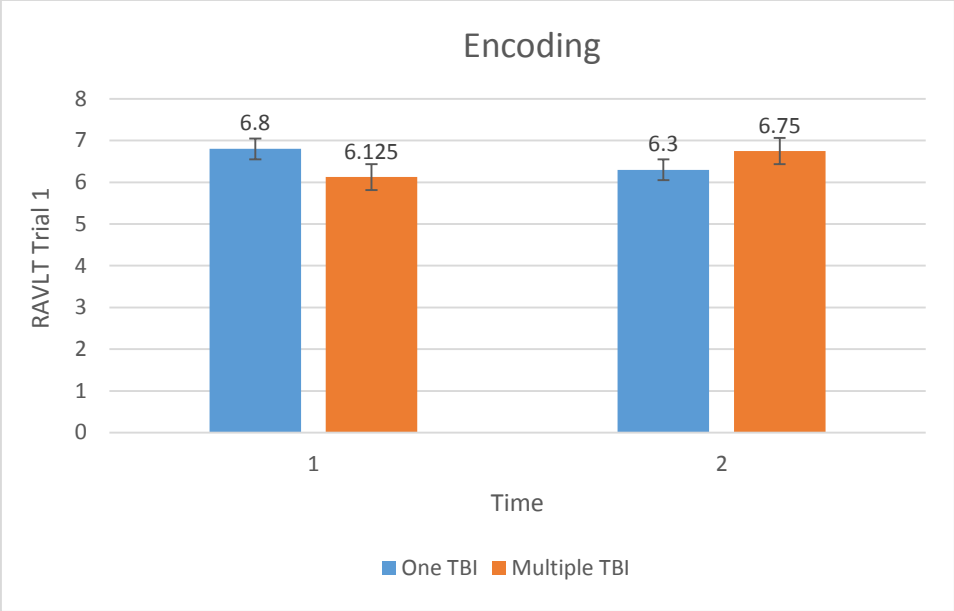

Figure 16. Means for encoding between single and multiple TBI groups.

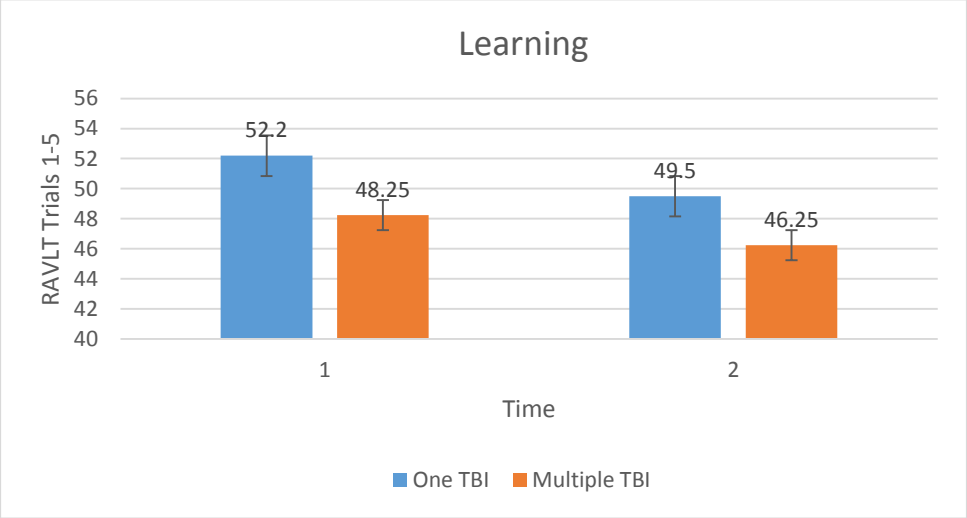

Figure 17. Means for learning between single and multiple TBI groups.

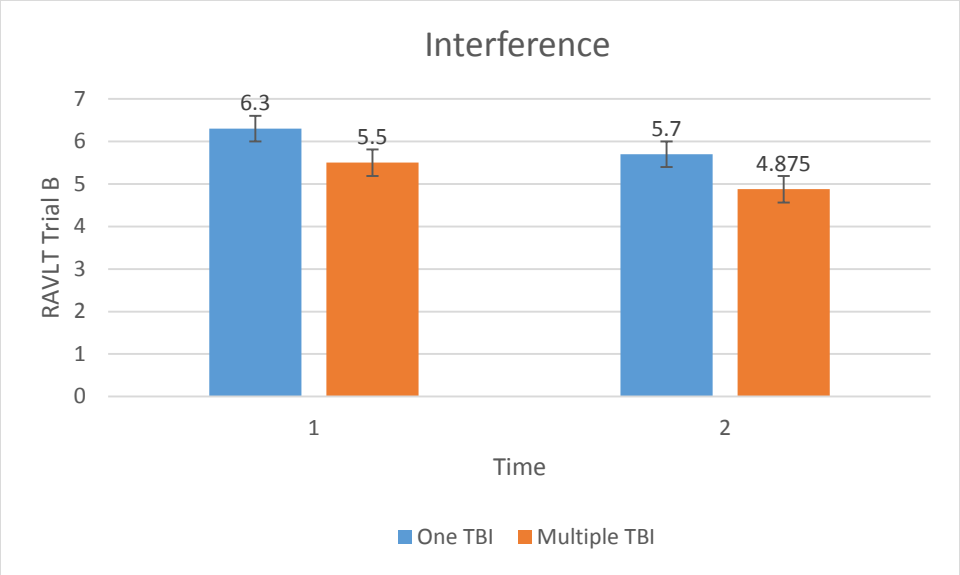

Figure 18. Means for interference between single and multiple TBI groups.

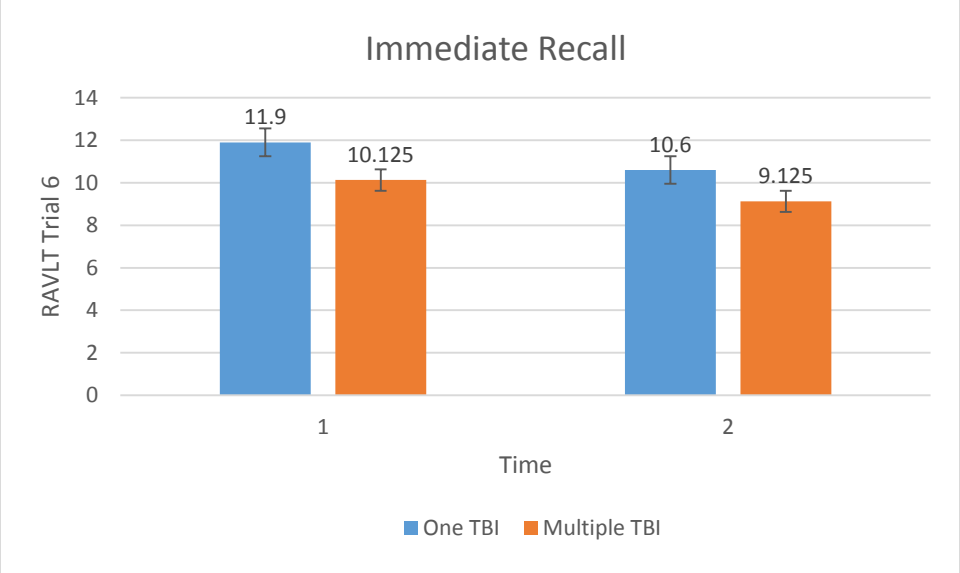

Figure 19. Means for immediate recall between single and multiple TBI groups.

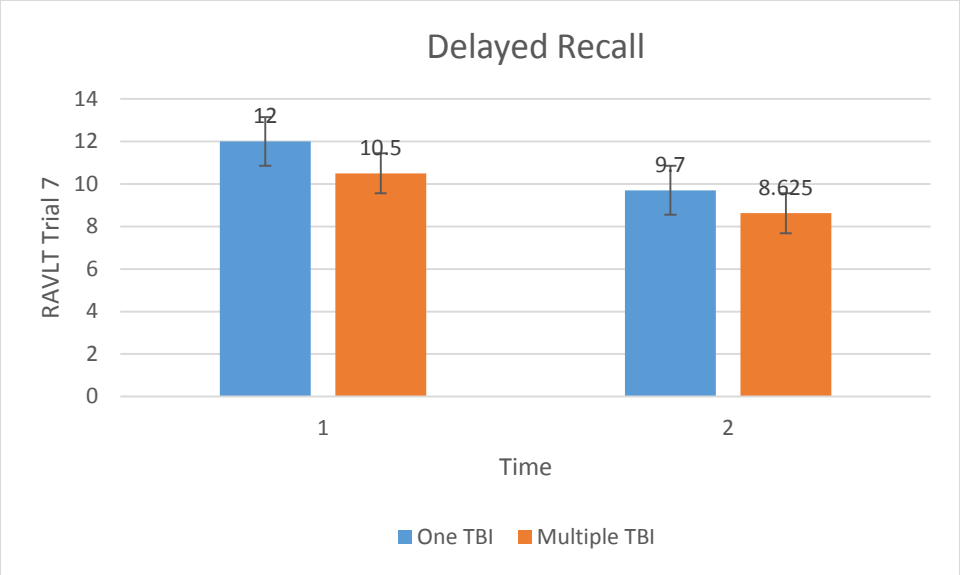

Figure 20. Means for delayed recall between single and multiple TBI groups.

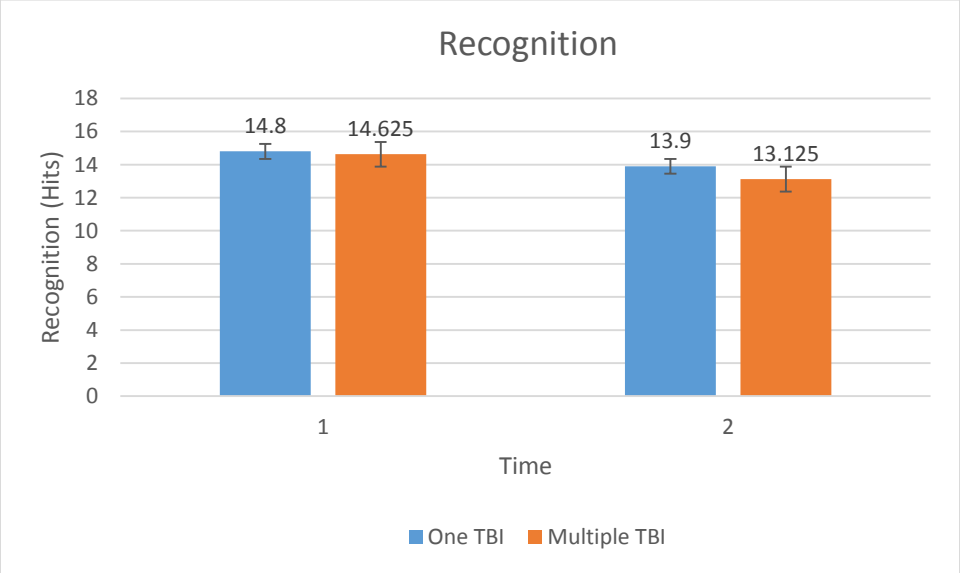

Figure 21. Means for recognition (hits) between single and multiple TBI groups.

CHAPTER 4

DISCUSSION

Previous research has indicated that being placed in an abnormally stressful environment may reveal cognitive deficits in people with previous TBI even when such deficits were not apparent in the absence of the stressor (Temme et al., 2013). In addition, research also suggests that TBI patients may need longer sleep durations than non-TBI patients (Sommerauer et al., 2013). This study sought to extend that research by investigating the potential presence of residual memory impairments in individuals with a history of one or more mild TBIs when they are exposed to the specific stressor of partial chronic sleep restriction. The design of the study was a 2 (mild TBI: none, at least one) x 2 (sleep: 8 hours, 6 hours) factorial MANOVA with brain injury as a between-subjects variable and sleep deprivation as a within-subjects variable. The dependent measures were sustained attention, encoding, learning, interference, immediate recall, delayed recall, and recognition. Due to the presence of two independent variables involving between-subjects and repeated measures combined with multiple dependent variables, a mixed MANOVA was run to analyze the data.

There were two original hypothesis for this study. First, it was hypothesized that there would be a significant difference in sustained attention and memory between the control group and the mild TBI group. It was also hypothesized that there would be a significant difference in the attention and memory variables between the single mild TBI group and the multiple TBI group. In summary, findings from this study did not support the first hypothesis. Results from running a mixed MANOVA revealed non-significant findings at $p < .05$. Furthermore, when univariate ANOVAs were run for each outcome measure after applying a Bonferroni correction to reduce inflation of type I error, findings remained statistically non-significant within and

between groups with very minimal effect sizes. The second hypothesis could not be analyzed due to a small sample size, great variability in cell size per group, and insufficient power.

Findings and Implications

The absence of a statistically significant difference between the control and TBI groups supports the longstanding theory that individuals who experience one or more mild TBI mostly recover from the head injury, and there is perhaps no latent deficit present to be unmasked. It is possible that different cognitive mechanisms were utilized during a controlled study versus during their daily activities. For instance, more attention may have been placed into encoding and retrieving information when they were directly instructed to do so whereas individuals with a history of mild TBI experience more difficulty with daily tasks that do not require effortful attention (e.g., searching for their car keys). As previously discussed, the dual consolidation theory suggested that different stages of sleep promoted consolidation of different information (i.e., SWS and declarative versus REM sleep and procedural). Given that this study only measured total hours of sleep and excluded information on stages of sleep, it is unknown whether or not there may have been differences in stages of sleep that may have potentially impacted cognitive performance.

One difference in administration of the procedure between sessions one and two was in the distractions during the delay. During the first session, subjects completed the ds-CPT and reviewed the instructions for utilizing the SleepTime application prior to the delayed recall task. In contrast, during the second session, subjects only completed the ds-CPT and were asked to take a seat in the waiting area where they were free to engage in their task of choice (e.g., checking their email, texting, or taking a restroom break). Although the distraction activities were different during the delay period, both the control and TBI groups were exposed to these

different procedures during sessions one and two, and are unlikely to have significantly contributed to the results.

The mild declines exhibited by the control group was unexpected given that sleep restriction to six hours each night for four nights was previously demonstrated to be insufficient to affect cognitive functioning due to a compensatory response from the brain (Van Dongen et al., 2003). Another unexpected outcome was that performance on the sustained attention task subtly improved following sleep restriction for both groups. This is in contrast to the current literature that supports the theory that sustained attention is most vulnerable to sleep deprivation compared to other cognitive domains and that impaired attention contributes to reductions in other areas of cognitive functioning. Given that the literature indicates that the DS-CPT is a more challenging task relative to other CPT tests, this unexpected outcome may be suggestive of a practice effect. A study to assess the test-retest reliability of the DS-CPT may be helpful for future studies.

Another unexpected observation was that the TBI group evidenced slightly better performance on the delayed recall and recognition tasks following sleep restriction despite the study design having controlled for potential confounding factors (e.g., medications, psychopathology, and heavy substance use). Although an attempt was made to control for practice effect of the RAVLT by administering an alternative version during the second session, it is possible that delayed recall and recognition tasks are more vulnerable to practice effects than the other memory outcome measures on the RAVLT (Lezak, Howieson, & Loring, 2004). Onset of sleep disturbances are sometimes reported as one of the symptoms following a mild TBI (Mathias & Alvaro, 2012). Given the variability in presentation of symptoms in people with mild TBI, individuals who do not experience sleep disturbance as one of their symptoms may not

necessarily experience cognitive reductions due to sufficient compensatory action taken by the brain similar to people without a history of a mild TBI. Although the subtly higher mean evidenced by the TBI group was unexpected, the difference was non-significant. Therefore, the implication that these subjects did not demonstrate a more reduced performance compared to the control group due to absence of sleep disturbances only helps to explain part of this unexpected outcome.

As noted above, a meaningful statistical analysis could not be conducted to test the second hypothesis of this study due to a small TBI sample size and the large difference in cell size per group. Specifically, a total of 18 enrolled subjects reported one or more TBI with N=11 for a single mild TBI, N=4 for two TBI, and N=3 for three TBI. However, taking into consideration the same caveat about statistical non-significance, the three TBI group demonstrated a subtle decrease in mean performance across sustained attention and most memory outcomes relative to the control and two TBI groups (i.e., learning, immediate recall, immediate recall, delayed recall, percent retention, and recognition). This may imply that individuals with a history of mild TBI who may be more vulnerable to cognitive weaknesses under stressful conditions may experience exacerbation of these reductions with an accumulation of subsequent mild TBI. This implication is also consistent with more recent TBI literature that suggests multiple concussions are correlated with more persistent cognitive deficits and a longer recovery time (i.e., >8 days following the TBI) compared to individuals with one or no previous TBI (Convassin, Moran, & Wilhelm, 2013). Even with a single mild TBI, individuals may continue to experience specifically cognitive weaknesses in the absence of other physical or emotional symptoms and are prone to sleep for longer durations (i.e., >9 hours per night) that is suggestive of active recovery mechanisms from the injury (Kostyun, Milewski, & Hafeez, 2015;

Thoma et al., 2015). In the absence of malingering, the effect of individual differences may have contributed to a statistical difference between groups. If multiple mild TBI are indeed more likely to be associated with residual memory deficits long after expected full recovery, it is imperative that attention be drawn to preventative measures and appropriate rehabilitative interventions to address potentially increased risk factors related to future functioning, such as neurodegenerative processes (e.g., Alzheimer's disease).

Limitations and Future Research

Internal validity was increased by utilizing a largely homogenous group of White, male undergraduate students within the age range of 18 through 22 with 12 to 13 years of education. Executive functioning is a cognitive domain that is largely associated with the frontal lobe, which is not fully developed until an adult reaches the average age of 25 years (Johnson, Blum, & Giedd, 2009). This may impact performance on memory tasks due to poor encoding (e.g., difficulty with organization or problem-solving when given a long list of words to memorize). Therefore, a more heterogeneous sample may help to account for unique differences within homogenous samples and to be better representative of the general population. Also, the literature suggests the presence of differences in sleep and memory when taking into consideration age as a variable. Although having a homogenous sample helped to improve internal validity, there were a number of limitations to the study as well.

Although new information was gleaned from this study, there were a number of limitations that should be taken into account in future research. One of the significant limitations was inadequate control over the sleep restriction condition. Given that there was no access to a sleep laboratory to directly monitor and ensure sleep restriction, this study utilized an alternative method of collecting objective sleep data by using a smartphone application in place of the

traditional sleep diaries. Although subjects who did not bring logged hours to their second session were excluded from data analysis given that the sleep literature suggested six hours of sleep over four nights was necessary to detect cognitive impairments, a sleep research laboratory would have allowed for better control over other factors, such as naps during the day or resumed sleep after turning off the mobile application.

Another limitation of this study was the sample size. Related to the limitation previously discussed, a total of 30 subjects who fully met the study requirements were included in data analysis. Although there were 12 subjects who also participated in the first part of the study, they were excluded from data analysis because they did not complete the sleep restriction requirement, which further decreased the sample size. In addition, given the large time commitment required of the subjects and the research assistants in addition to limited availability of testing rooms, not all subjects who met eligibility criteria were given the opportunity to participate in the study.

Lastly, a limitation of this study was the absence of a stand-alone performance validity measure. An embedded effort index was utilized in the form of the recognition task on the RAVLT with the cut-off score set at $\leq$9, which has been found to demonstrate sufficient sensitivity and specificity for predicting non-credible effort (Whitney & Davis, 2015). Although all of the eligible subjects' performance on this effort measure exceeded the cut-off during the first session, there was one subject whose performance on the same task during the second session fell below this cut-off. Statistical findings did not significantly change when including and excluding this subject. Therefore, it is unclear if the subject's performance during the second session is more suggestive of insufficient effort or an impairment in recognition of auditory information following sleep restriction. Inclusion of a stand-alone performance validity

measure may be helpful if this study is replicated with a larger sample size in order to take performance credibility into account and rule out another potential confounding factor.

Although there is extensive literature looking at cognitive functioning in individuals with mild TBI, this study is one of the few to investigate the presence of residual effects following one or more mild TBI when expected full recovery is approximately three months post-concussion. In this study, screening for mild TBI consisted of following the TBI severity criteria provided by the Department of Veterans Affairs and primarily assessed for duration of loss of consciousness and presence of posttraumatic amnesia. There may be complications or different neurologic sequelae following a mild TBI that cannot be detected using an isolated self-report, such as traumatic subarachnoid hemorrhages that affect recovery outcome (Deepika, Munivenkatappa, & Shukla, 2013). Currently, the literature on mild TBI commonly focuses on adolescents and sports-related injuries. Similarly, this study was comprised of a homogenous sample of college students who reported that the etiology of the injury was motor vehicular or sports-related. Given the various etiology of a mild TBI, the inclusion of neuroimaging in future research would be interesting to explain residual cognitive deficits from a neurological perspective following sleep deprivation.

In conclusion, this study did not find statistically significant findings related to differences in attention and memory abilities between people with no mild TBI and people with one or more mild TBI. The subtle differences found in these cognitive domains are non-significant but consistent with the literature that suggests that there may be more cognitive and functional difficulties following multiple mild TBI than previous research evidenced (Convassin, Moran, & Wilhelm, 2013). On the other hand, the absence of significant findings in this study also implies that perhaps there are other variables (e.g., age) that may help to identify the extent

to which mild TBI impairs memory after the expected acute recovery period of three months. The consequences of subsequent mild TBI combined with other risk factors (e.g., sleep deprivation and cognitive changes associated with increasing age) will continue to pose a significant public health concern. Therefore, continued exploration of these two growing public health issues will be necessary for preventative and treatment purposes.

References

Aben, B., Stapert, S., & Blokland, A. (2012). About the distinction between working memory and short-term memory. *Frontiers in Psychology, 3*, 301. doi:10.3389/fpsyg.2012.00301

Alexander, M. P. (1997). Minor traumatic brain injury: a review of physiogenesis and psychogenesis. *Seminars in Clinical Neuropsychiatry, 2*, 177-187. doi:10.1053/SCNP00200177

American Psychiatric Association (Ed.). (2000). *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR.* Washington, D.C.: American Psychiatric Association.

Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W., & Pollak, C. (2003). The role of actigraphy in the study of sleep and circadian rhythms. *Sleep, 26*(3), 342-392.

Baddeley, A. (2002). The psychology of memory. In A.D. Baddeley, M.D. Kopelman, & B.A. Wilson (Eds.), *The Handbook of Memory Disorders*. Chichester, UK: Wiley.

Banks, S., & Dinges, D. F. (2007). Behavioral and physiological consequences of sleep restriction. *Journal of Clinical Sleep Medicine, 3*(5), 519-528.

Banks, S., Van Dongen, H. P., Maislin, G., & Dinges, D. F. (2010). Neurobehavioral dynamics following chronic sleep restriction: dose-response effects of one night for recovery. *Sleep*, *33*(8), 1013.

Barulli, D., & Stern, Y. (2013). Efficiency, capacity, compensation, maintenance, plasticity: emerging concepts in cognitive reserve. *Trends in Cognitive Sciences, 17*(10), 502-509. doi:10.1016/j.tics.2013.08.012

Basner, M., & Dinges, D. F. (2011). Maximizing sensitivity of the psychomotor vigilance test (PVT) to sleep loss. *Sleep, 34*(5), 581-591.

Baumann, C. R., Stocker, R., Imhof, H. G., Trentz, O., Hersberger, M., Mignot, E., & Bassetti, C. L. (2005). Hypocretin-1 (orexin A) deficiency in acute traumatic brain injury. *Neurology, 65*(1), 147-149. doi:10.1212/01.wnl.0000167605.02541.f2

Baumann, C. R., Werth, E., Stocker, R., Ludwig, S., & Bassetti, C. L. (2007). Sleep–wake disturbances 6 months after traumatic brain injury: a prospective study. *Brain, 130*(7), 1873-1883. doi:10.1093/brain/awm109

Bay, E., & Covassin, T. (2012). Chronic stress, somatic and depressive symptoms following mild to moderate traumatic brain injury. *Archives of Psychiatric Nursing, 26*(6), 477-486. doi:10.1016/j.apnu.2012.06.002

Benington, J. H. (2000). Sleep homeostasis and the function of sleep. *Sleep, 23*(7), 959-966.

Bergman, K., & Bay, E. (2010). Mild traumatic brain injury/concussion: a review for ED nurses. *Journal of Emergency Nursing, 36*(3), 221-230. doi:10.1016/j.jen.2009.07.001

Boake, C., McCauley, S. R., Levin, H. S., Contant, C. F., Song, J. X., Brown, S. A., Goodman, H. S., Brundage, S. I., Diaz-Marchan, P. J., & Merritt, S. G. (2004). Limited agreement between criteria-based diagnoses of postconcussional syndrome. *Journal of Neuropsychiatry and Clinical Neuroscience, 16*(4), 493-499. doi:10.1176/jnp.16.4.493

Borbely, A. A., & Achermann, P. (1999). Sleep homeostasis and models of sleep regulation. *Journal of Biological Rhythms, 14*(6), 559-570. doi:10.1177/074873099129000894

Born, J., & Feld, G. B. (2012). Sleep to upscale, sleep to downscale: balancing homeostasis and plasticity. *Neuron, 75*(6), 933-935. doi:10.1016/j.neuron.2012.09.007

Brankačk, J., Platt, B., & Riedel, G. (2009). Sleep and hippocampus: do we search for the right things? *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 33*(5), 806-812. doi:10.1016/j.pnpbp.2009.03.027

Brankack, J., Scheffzuek, C., Kukushka, V. I., Vyssotski, A. L., Tort, A. B., & Draguhn, A. (2012). Distinct features of fast oscillations in phasic and tonic rapid eye movement sleep. *Journal of Sleep Research, 21*(6), 630-633. doi:10.1111/j.1365-2869.2012.01037.x

Brown, S. C. & Craik, F. I. (2000). *Encoding and Retrieval of Information.* New York, NY: Oxford University Press.

Buzsáki, G. (1989). Two-stage model of memory trace formation: a role for "noisy" brain states. *Neuroscience, 31*(3), 551-570. doi:10.1016/0306-4522(89)90423-5

Cabeza, R., Ciaramelli, E., Olson, I. R., & Moscovitch, M. (2008). The parietal cortex and episodic memory: an attentional account. *Nature Reviews Neuroscience, 9*(8), 613-625.

Chee, M. W. & Chuah, L. Y. (2008). Functional neuroimaging insights into how sleep and sleep deprivation affect memory and cognition. *Current Opinion in Neurology, 21*(4), 417-423. doi:10.1097/WCO.0b013e3283052cf7

Chen, Z., Lin, M., Chen, F., Lane, N. D., Cardone, G., Wang, R., Tianxing, L., Chen, Y., Choudhury, T., & Campbell, A. T. (2013). Unobtrusive sleep monitoring using smartphones. *Proceedings of Pervasive Health,* 145-152. doi:10.4108/pervasivehealth.2013.252148

Cirelli, C. (2012). Sleep and synaptic homeostasis. *Journal of Sleep Research, 21,* 113-113.

Clarke, L. A., Genat, R. C., & Anderson, J. F. (2012). Long-term cognitive complaint and post-concussive symptoms following mild traumatic brain injury: the role of cognitive and affective factors. *Brain Injury, 26*(3), 298-307. doi:10.3109/02699052.2012.654588

Clemens, Z., Fabo, D., & Halasz, P. (2005). Overnight verbal memory retention correlates with the number of sleep spindles. *Neuroscience, 132*(2), 529-535. doi:10.1016/j.neuroscience.2005.01.011

Cowan, N. (2008). What are the differences between long-term, short-term, and working memory? *Progress in Brain Research, 169*, 323–338. doi:10.1016/S0079-6123(07)00020-9

Crick, F., & Mitchinson, G. (1983). The function of dream sleep. *Nature, 304*, 111–114.

de Sousa Magalhães, S., Malloy-Diniz, L. F., & Hamdan, A. C. (2012). Validity convergent and reliability test-retest of the Rey Auditory Verbal Learning Test. *Clinical Neuropsychiatry, 9*(3): 129-137.

D'Esposito, M., Postle, B., Ballard, D., & Lease, J. (1999). Maintenance versus manipulation of information held in working memory: an event-related fMRI study. *Brain Cognition, 41*, 66–86. doi:10.1006/brcg.1999.1096

Department of Veterans Affairs & Department of Defense. (2009). *VA/DoD Clinical Practice Guideline for Management of Concussion/Mild Traumatic Brain Injury (mTBI).* Retrieved from http://www.healthquality.va.gov/mtbi/concussion_mtbi_full_1_0.pdf

Doran, S. M., Van Dongen, H. P., & Dinges, D. F. (2001). Sustained attention performance during sleep deprivation: evidence of state instability. *Archives Italiennes de Biologie, 139*(3), 253-267.

Drummond, S. P., Anderson, D. E., Straus, L. D., Vogel, E. K., & Perez, V. B. (2012). The effects of two types of sleep deprivation on visual working memory capacity and filtering efficiency. *PLoS One, 7*(4), e35653. doi:10.1371/journal.pone.0035653

Ertelt, D., Witt, K., Reetz, K., Frank, W., Junghanns, K., Backhaus, J., ... & Binkofski, F. (2012). Skill memory escaping from distraction by sleep—evidence from dual-task performance. *PLoS One, 7*(12), e50983. doi:10.1371/journal.pone.0050983

Fay, T. B., Yeates, K. O., Taylor, H. G., Bangert, B., Dietrich, A., Nuss, K. E., . . . Wright, M. (2010). Cognitive reserve as a moderator of postconcussive symptoms in children with

complicated and uncomplicated mild traumatic brain injury. *Journal of the International Neuropsychological Society, 16*(1), 94-105. doi:10.1017/S1355617709991007

Fogel, S. M., & Smith, C. T. (2011). The function of the sleep spindle: a physiological index of intelligence and a mechanism for sleep-dependent memory consolidation. *Neuroscience and Biobehavioral Reviews, 35*(5), 1154-1165. doi:10.1016/j.neubiorev.2010.12.003

Fogel, S. M., & Smith, C. T. (2006). Learning-dependent changes in sleep spindles and stage 2 sleep. *Journal of Sleep Research, 15*(3), 250-255. doi:10.1111/j.1365-2869.2006.00522.x

Frank, M. G. (2013). Why I Am Not SHY: A Reply to Tononi and Cirelli. *Neural Plasticity*. doi:10.1155/2013/394946

Goel, N., Rao, H., Durmer, J. S., & Dinges, D. F. (2009). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology, 29*(4), 320. doi:10.1055/s-0029-1237117

Gray, P. (2007). *Psychology*, 5th edition. New York: Worth Publishers.

Guskiewicz, K. M., McCrea, M., Marshall, S. W., Cantu, R. C., Randolph, C., Barr, W., Onate, J. A., & Kelly, J. P. (2003). Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA concussion study. *The Journal of the American Medical Association, 290*(19), 2549-2555. doi:10.1001/jama.290.19.2549

Heinsichs, R. W. (2001). *In Search of Madness: Schizophrenia and Neuroscience*. New York, NY: Oxford University Press.

Horne, J. A. (1988). *Why We Sleep*. New York, NY: Oxford University Press.

Horne, J. A., & Reyner, L. A. (1995). Sleep related vehicle accidents. *British Medical Journal, 310*(6979), 565-567. doi:10.1136/bmj.310.6979.565

Iverson, G. L., Brooks, B. L., Lovell, M. R., & Collins, M. W. (2006). No cumulative effects for one or two previous concussions. *British Journal of Sports Medicine, 40*(1), 72-75. doi:10.1136/bjsm.2005.020651

Iverson, G. L. (2006). Misdiagnosis of the persistent postconcussion syndrome in patients with depression. *Archives Clinical Neuropsychology, 21*(4), 303-310. doi:10.1016/j.acn.2005.12.008

Iverson, G. L., Lange, R. T., Waljas, M., Liimatainen, S., Dastidar, P., Hartikainen, K. M., . . . Ohman, J. (2012). Outcome from Complicated versus Uncomplicated Mild Traumatic Brain Injury. *Rehabilitation Research and Practice*, 415740. doi:10.1155/2012/415740

Jamora, C. W., Young, A., & Ruff, R. M. (2012). Comparison of subjective cognitive complaints with neuropsychological tests in individuals with mild vs more severe traumatic brain injuries. *Brain Injury, 26*(1), 36-47. doi:10.3109/02699052.2011.635352.

Johns, M. W. (1992). Reliability and factor analysis of the Epworth Sleepiness Scale. *Sleep, 15*(4), 376-381.

Johnson, B., Zhang, K., Gay, M., Horovitz, S., Hallett, M., Sebastianelli, W., & Slobounov, S. (2012). Alteration of brain default network in subacute phase of injury in concussed individuals: resting-state fMRI study. *Neuroimage, 59*(1), 511-518. doi:10.1016/j.neuroimage.2011.07.081

Johnstone, B., Callahan, C. D., Kapila, C. J., & Bouman, D. E. (1996). The comparability of the WRAT-R reading test and NAART as estimates of premorbid intelligence in neurologically impaired patients. *Archives of Clinical Neuropsychology, 11*(6), 513-519. doi:10.1093/arclin/11.6.513

Kashluba, S., Casey, J. E., & Paniak, C. (2006). Evaluating the utility of ICD-10 diagnostic criteria for postconcussion syndrome following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 12*(1), 111-118. doi:10.1017/S1355617706060036

Kogure, T., Shirakawa, S., Shimokawa M., & Hosokawa, Y. (2011). Automatic sleep/wake scoring from body motion in bed: validation of a newly developed sensor placed under a mattress. *Journal of Physiological Anthropology, 30*(3), 103-109. doi:10.2114/jpa2.30.103

Kontos, A. P., Kotwal, R. S., Elbin, R. J., Lutz, R. H., Forsten, R. D., Benson, P. J., & Guskiewicz, K. M. (2013). Residual effects of combat-related mild traumatic brain injury. *Journal of Neurotrauma, 30*(8), 680-686. doi:10.1089/neu.2012.2506

Lauderdale, D. S., Knutson, K. L., Yan, L. L., Liu, K., & Rathouz, P. J. (2008). Sleep duration: how well do self-reports reflect objective measures? The CARDIA sleep study. *Epidemiology, 19*(6), 838-845. doi:10.1097/EDE.0b013e318187a7b0

Lawson, S., Jamison-Powell, S., Garbett, A., Linehan, C., Kucharczyk, E., Verbaan, S., Rowland, D. A., & Morgan, K. (2013). Validating a mobile phone application for the everyday, unobtrusive, objective measurement of sleep. *CHI '13 SIGCHI Conference on Human Factors in Computing Systems, Paris, France.* doi:10.1145/2470654.2481345

Leger, D., Beck, F., Richard, J. B., & Godeau, E. (2012). Total sleep time severely drops during adolescence. *PLoS One, 7*(10), e45204. doi:10.1371/journal.pone.0045204

Lelkes, Z., Porkka-Heiskanen, T., & Stenberg, D. (2013). Cholinergic basal forebrain structures are involved in the mediation of the arousal effect of noradrenaline. *Journal of Sleep Research 22*(6), 721-736. doi:10.1111/jsr.12061

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological Assessment (4th ed.).* New York, NY: Oxford University Press.

Lim, J. & Dinges, D. F. (2008). Sleep deprivation and vigilant attention. *Annals of the New York Academy of Sciences, 1129,* 305-322. doi:10.1196/annals.1417.002

Lim, J., Tan, J. C., Parimal, S., Dinges, D. F., & Chee, M. W. (2010). Sleep deprivation impairs object-selective attention: a view from the ventral visual cortex. *PLoS One*, *5*(2), e9087. doi:10.1371/journal.pone.0009087

Lo, J. C., Groeger, J. A., Santhi, N., Arbon, E. L., Lazar, A. S., Hasan, S., ... & Dijk, D. J. (2012). Effects of partial and acute total sleep deprivation on performance across cognitive domains, individuals and circadian phase. *PLoS One*, *7*(9), e45987. doi:10.1371/journal.pone.0045987

Mallick, H. N. & Kumar, V. M. (2012). Basal forebrain thermoregulatory mechanism modulates auto-regulated sleep. *Frontiers in Neurology*, *3*, 102. doi:10.3389/fneur.2012.00102

Mass, R., Wolf, K., Wagner, M., & Haasen, C. (2000). Differential sustained attention/vigilance changes over time in schizophrenics and controls during a degraded stimulus Continuous Performance Test. *European Archives of Psychiatry and Clinical Neuroscience*, *250*(1), 24-30. doi:10.1007/PL00007535

Mathias, J. L., & Alvaro, P. K. (2012). Prevalence of sleep disturbances, disorders, and problems following traumatic brain injury: a meta-analysis. *Sleep Medicine, 13*(7), 898-905. doi:10.1016/j.sleep.2012.04.006

Mayes, A. R. (2000). The neuropsychology of memory. In G.E. Berrios & J.R. Hodges (Eds.), *Memory Disorders in Psychiatric Practice.* Cambridge, UK: Cambridge University Press.

McCrea, M., Guskiewicz, K. M., Marshall, S.W., Barr, W., Randolph, C., Cantu, R. C., Onate, J. A., Yang, J., & Kelly, J. P. (2003). Acute effects and recovery time following concussion in collegiate football players: the NCAA concussion study. *The Journal of the American Medical Association, 290*(19), 2556-2563. doi:10.1001/jama.290.19.2556

McGaugh, J. L. (2000). Memory--a century of consolidation. *Science, 287*(5451), 248-251. doi:10.1126/science.287.5451.248

McKnight-Eily, L. R., Liu, Y., Perry, G. S., Presley-Cantrell, L. R., Strine, T. W., Lu, H., & Croft, J. B. (2009). Perceived insufficient rest or sleep among adults-United States, 2008. *Morbidity and Mortality Weekly Report, 58*, 1175-1179.

National Institutes of Health. (2007). NIH Curriculum Supplement Series [Internet]. Bethesda (MD): National Institutes of Health (US). Information about Sleep. Retrieved from: http://www.ncbi.nlm.nih.gov/books/NBK20359/

National Lung, Heart, and Blood Institute. Retrieved from: http://www.nhlbi.nih.gov/news/spotlight/fact-sheet/sleep-disorders-insufficient-sleep-improving-health-through-research.html

O'Connor, M., & Verfaillie, M. (2002). The amnesic syndrome: Overview and subtypes. In A.D. Baddeley et al. (2002). *The Handbook of Memory Disorders*. Chichester, UK: Wiley.

Orme, D. R., Johnstone, B., Hanks, R., & Novack, T. (2004). The WRAT-3 reading subtest as a measure of premorbid intelligence among persons with brain injury. *Rehabilitation Psychology, 49*(3), 250-253. doi:10.1037/0090-5550.49.3.250

Ouellet, M. C., Savard, J., & Morin, C. M. (2004). Book Review: Insomnia following Traumatic Brain Injury: A Review. *Neurorehabilitation and Neural Repair, 18*(4), 187-198. doi:10.1177/1545968304271405

Palacios, E. M., Sala-Llonch, R., Junque, C., Fernandez-Espejo, D., Roig, T., Tormos, J. M., . . . Vendrell, P. (2013). Long-term declarative memory deficits in diffuse TBI: correlations with cortical thickness, white matter integrity and hippocampal volume. *Cortex, 49*(3), 646-657. doi:10.1016/j.cortex.2012.02.011

Parcell, D. L., Ponsford, J. L., Rajaratnam, S. M., Redman, J. R. (2006). Self-reported changes to nighttime sleep after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 87*, 278-285. doi:10.1016/j.apmr.2005.10.024

Payne, J. D., Chambers, A. M., & Kensinger, E. A. (2012). Sleep promotes lasting changes in selective memory for emotional scenes. *Frontiers in Integrative Neuroscience*, *6*, 108. doi:10.3389/fnint.2012.00108

Pellman, E. J., Lovell, M. R., Viano, D. C., Casson, I. R., & Tucker, A. M. (2004). Concussion in professional football: neuropsychological testing--part 6. *Neurosurgery, 55*(6), 1290-1303. doi:10.1227/01.NEU.0000149244.97560.91

Pollak, C. P., Tryon, W. W., Nagaraja, H., & Dzwonczyk, R. (2001). How accurately does wrist actigraphy identify the states of sleep and wakefulness?. *Sleep, 24*(8), 957-965.

Polusny, M. A., Kehle, S. M., Nelson, N.W., Erbes, C .R., Arbisi, P. A., & Thuras, P. (2011). Longitudinal effects of mild traumatic brain injury and posttraumatic stress disorder comorbidity on postdeployment outcomes in National Guard soldiers deployed to Iraq. *Archives of General Psychiatry, 68*(1), 79-89. doi:10.1001/archgenpsychiatry.2010.172

Pomplun, M., Silva, E. J., Ronda, J. M., Cain, S. W., Münch, M. Y., Czeisler, C. A., & Duffy, J. F. (2012). The effects of circadian phase, time awake, and imposed sleep restriction on performing complex visual tasks: Evidence from comparative visual search. *Journal of Vision, 12*(7), 1-19. doi:10.1167/12.7.14

Porkka-Heiskanen, T. (2013). Sleep homeostasis. *Current Opinion in Neurobiology, 23*(5), 799-805. doi:10.1016/j.conb.2013.02.010

Porte, H. S. (2005). Procedural replay: the anatomy and physics of the sleep spindle. *Behavioral and Brain Sciences, 28*(1), 79-80. doi:10.1017/S0140525X05370026

Rauchs, G., Feyers, D., Landeau, B., Bastin, C., Luxen, A., Maquet, P., & Collette, F. (2011). Sleep contributes to the strengthening of some memories over others, depending on hippocampal activity at learning. *The Journal of Neuroscience, 31*(7), 2563-2568. doi:10.1523/JNEUROSCI.3972-10.2011

Ramaligam, V., Chen, M. C., Saper, C. B., & Lu, J. (2013). Perspectives on the rapid eye movement sleep switch in rapid eye movement sleep behavior disorder. *Sleep Medicine, 14*(8), 707-713. doi:10.1016/j.sleep.2013.03.017

Rona, R. J. (2012). Long-term consequences of mild traumatic brain injury. *The British Journal of Psychiatry*, *201*(3), 172-174. doi:10.1192/bjp.bp.112.111492

Rona, R. J., Jones, M., Fear, N. T., Hull, L., Murphy, D., Machell, L., ... & Wessely, S. (2012). Mild traumatic brain injury in UK military personnel returning from Afghanistan and Iraq: cohort and cross-sectional analyses. *The Journal of Head Trauma Rehabilitation*, *27*(1), 33-44. doi:10.1097/HTR.0b013e318212f814

Sinclair, K. L., Ponsford, J. L., Rajaratnam, S. M., & Anderson, C. (2013). Sustained attention following traumatic brain injury: use of the psychomotor vigilance task. *Journal of*

Clinical and Experimental Neuropsychology, *35*(2), 210-224. doi:10.1080/13803395.2012.762340

Smith, C. (2001). Sleep states and memory processes in humans: procedural versus declarative memory systems. *Sleep Medicine Reviews, 5*(6), 491-506. doi:10.1053/smrv.2001.0164

Smith, C. T., Nixon, M. R., & Nader, R. S. (2004). Posttraining increases in REM sleep intensity implicate REM sleep in memory processing and provide a biological marker of learning potential. *Learning & Memory, 11*(6), 714-719. doi:10.1101/lm.74904

Sommerauer, M., Valko, P. O., Werth, E., & Baumann, C. R. (2013). Excessive sleep need following traumatic brain injury: a case–control study of 36 patients. *Journal of Sleep Research*. doi:10.1111/jsr.12068

Stevens, K. B. & Price, J. R. (1999). Adult reading assessment: Are we doing the best with what we have? *Applied Neuropsychology, 6*(2), 68-78. doi:10.1207/s15324826an0602_2

Stickgold, R., & Walker, M. P. (2005). Memory consolidation and reconsolidation: what is the role of sleep? *Trends in Neurosciences, 28*(8), 408-415. doi:10.1016/j.tins.2005.06.004

Temme, L., Bleiberg, J., Reeves, D., Still, D. L., Levinson, D., & Browning, R. (2012). Uncovering latent deficits due to mild traumatic brain injury by using normobaric hypoxia stress. *Frontiers in Neurology*, *4*, 41. doi:10.3389/fneur.2013.00041

Tononi, G., & Cirelli, C. (2012). Time to be SHY? Some comments on sleep and synaptic homeostasis. *Neural Plasticity*. doi:10.1155/2012/415250

Tryon, W. (2004). Issues of validity in actigraphic sleep assessment. *Sleep, 27*(1), 158-165.

Tulving, E. (1985). How many memory systems are there? *American Psychologist, 40*(4), 385. doi:10.1037/0003-066X.40.4.385

Uncapher, M. R. & Wagner, A. D. (2009). Posterior parietal cortex and episodic encoding: insights from fMRI subsequent memory effects and dual-attention theory. *Neurobiology of Learning and Memory, 91*(2), 139-154. doi:10.1016/j.nlm.2008.10.011

Van Dongen, H., Rogers, N. L., & Dinges, D. F. (2003). Sleep debt: theoretical and empirical issues. *Sleep and Biological Rhythms, 1*, 5-13. doi:10.1046/j.1446-9235.2003.00006

Vertes, R. P. & Eastman, K. E. (2000). The case against memory consolidation in REM sleep. *Behavioral and Brain Sciences, 23*(6), 867-876. doi:10.1017/S0140525X00004003

Wagner, A. D., Shannon, B. J., Kahn, I., & Buckner, R. L. (2005). Parietal lobe contributions to episodic memory retrieval. *Trends in Cognitive Sciences, 9*(9), 445-453. doi:10.1016/j.tics.2005.07.001

Waldron-Perrine, B., McGuire, A. P., Spencer, R. J., Drag, L. L., Pangilinan, P. H., & Bieliauskas, L. A. (2012). The influence of sleep and mood on cognitive functioning among veterans being evaluated for mild traumatic brain injury. *Military Medicine, 177*(11), 1293-1301. doi:10.7205/MILMED-D-12-00169

Walker, M. P. & Stickgold, R. (2004). Sleep-dependent learning and memory consolidation. *Neuron, 44*(1), 121-133. doi:10.1016/j.neuron.2004.08.031

Waters, F. & Bucks, R. S. (2011). Neuropsychological effects of sleep loss: implication for neuropsychologists. *Journal of the International Neuropsychological Society, 17*(4), 571. doi:10.1017/S1355617711000610

Watts, A., Gritton, H. J., Sweigart, J., & Poe, G. R. (2012). Antidepressant suppression of non-REM sleep spindles and REM sleep impairs hippocampus-dependent learning while augmenting striatum-dependent learning. *The Journal of Neuroscience, 32*(39), 13411-13420. doi:10.3109/02699052.2013.823663

Williams, R. M., Puetz, T. W., Giza, C. C., & Broglio, S. P. (2015). Concussion recovery time among high school and collegiate athletes: A systematic review and meta-analysis. *Sports Medicine, 45*(6), 893-903. doi:10.1007/s40279-015-0325-8

Wiseman-Hakes, M. B., Moineddin, R., Rochon, E., Cullen, N., Gargaro, J., & Colantonio, A. (2013). Evaluating the impact of treatment for sleep/wake disorders on recovery of cognition and communication in adults with chronic TBI. *Brain Injury, 27*(12), 1364-1376. doi:10.3109/02699052.2013.823663

Witt, S. T., Lovejoy, D. W., Pearlson, G. D., & Stevens, M. C. (2010). Decreased prefrontal cortex activity in mild traumatic brain injury during performance of an auditory oddball task. *Brain Imaging and Behavior, 4*(4), 232-247. doi:10.1007/s11682-010-9102-3

Wood, R.L. (2004). Understanding the 'miserable minority': a diasthesis-stress paradigm for post-concussional syndrome. *Brain Injury, 18*(11), 1135-1153. doi:10.1080/02699050410001675906

Zanini, G. D., Tufik, S., Andersen, M. L., da Silva, R., Bueno, O., Rodrigues, C. C., & Pompéia, S. (2012). Free recall of word lists under total sleep deprivation and after recovery sleep. *Sleep, 35*(2), 223-230. doi:10.5665/sleep.1626

Zhou, X., Ferguson, S. A., Matthews, R. W., Sargent, C., Darwent, D., Kennaway, D. J., & Roach, G. D. (2011) Sleep, wake and phase dependent changes in neurobehavioral function under forced desynchrony. *Sleep, 34*(7), 931–941. doi:10.5665/sleep.1130.

APPENDIX A
Pre-Screening Questionnaire

1. Is English your native language (i.e., was it the language you first learned as a child)?
a. Yes
b. No

2. Are you currently taking any psychotropic, anticonvulsant, antihistamine, opiate, or narcotic medication(s)?
a. Yes
b. No

3. Do you have a serious medical condition, such as sleep apnea, cardiovascular disease, or epilepsy?
a. Yes
b. No

4. Have you had injuries from any of the following? (circle all that apply)
a. Vehicular (any type of vehicle, including airplane)
b. Fall
c. Blast
d. Other

5. How long has it been since the injury(ies)?
    a. Less than one month
    b. More than one month

5. Did the injury(ies) result in any of the following?
    a. Being dazed, confused, or "seeing stars"
    b. Not remembering the injury
    c. Losing consciousness for less than a minute
    d. Losing consciousness for 1-20 minutes
    e. Losing consciousness for longer than 20 minutes

5. Are you currently experiencing any of the following problems that you think might be related to the head injury? (circle all that apply)
    a. Headaches
    b. Dizziness
    c. Memory problems
    d. Balance problems
    e. Ringing in the ears
    f. Irritability
    g. Sleep problems
    h. Other

APPENDIX B
Demographics Questionnaire

For descriptive purposes, please answer the following questions:

1. What is your age? _____

2. What is your gender?
   a. Male
   b. Female
   c. Transgender
   d. Not specified

3. What is your race/ethnicity?
   a. Black
   b. White
   c. Asian
   d. Hispanic
   e. Multiracial/multi-ethnic
   f. Other: _____

4. How many years of education have you completed? _____ years
(typical high school education = 12 years)

5. Do you consume alcohol?
   a. No
   b. Yes
      If yes, please describe how often and an average of how many drinks per occasion:

6. Do you use any drugs?
   a. No
   b. Yes
      If yes, please list the drug and describe how often:

7. Have you recently experienced a stressful event (e.g., exam, loss of a loved one, illness)?

APPENDIX C

Instructions for Sleep Time Application

You have downloaded the Sleep Time Application on your smartphone at this time. In order to obtain an accurate reading of your body movements overnight using the accelerometer feature on your phone, you will be expected to spend the night without any other people or pets sharing the bed. The following are instructions for how to use the Sleep Time Application and your responsibilities as a research subject in this study.

For the first two nights, you will be required to sleep 8 hours.

1. Turn off all alerts on your phone to avoid sleep disturbances (e.g., text messages, other application notifications).
2. Turn on the Sleep Time app once you are in bed, and use the "SET" dial to set the alarm time to 8:00 to indicate 8 hours. Press "START."

3. Place the phone face-down on top of your mattress out of the way of your pillow where it is unlikely to fall off the bed or disturb you while you sleep.
4. Hold the stop button for 3 seconds to stop the app from running after you wake up.
5. Repeat Steps 1-4 on the second night.

For the remaining two nights, you will be required to sleep only 6 hours.

1. Turn off all alerts on your phone to avoid sleep disturbances (e.g., phone calls, text messages, other application notifications).
2. Turn on the Sleep Time app once you are in bed, and use the "SET" dial to set the alarm time to 6:00 to indicate 6 hours. Press "START."

3. Place the phone face-down on top of your mattress out of the way of your pillow where it is unlikely to fall off the bed or disturb you while you sleep.
4. Hold the stop button for 3 seconds to stop the app from running after you wake up.
5. Repeat Steps 1-4 on the second night.

You will see the following data table after each night:

For the duration of the study,

- Please refrain from taking naps or returning to sleep after the alarm goes off
- Please abstain from consumption of alcohol and/or use of other substances

APPENDIX D
Informed Consent Form

(will insert IUP letterhead)

Informed Consent Form

You are invited to participate in this research study. The following information is provided in order to help you to make an informed decision about whether or not to participate. If you have any questions, please do not hesitate to ask. You are eligible to participate because you are a student enrolled in the Introduction to Psychology course and seeking to fulfill research participation requirements.

Description

The purpose of this study is to examine the effect of sleep on memory. Participation will take place over five consecutive days after which you will be given research credits to fulfill all of your research participation requirements for the Introduction to Psychology course.
You will be instructed to download a free mobile application called Sleep Time on your smartphone to obtain objective measures of light sleep, deep sleep, sleep duration, and sleep quality. You will place your smartphone on the corner of the mattress near the pillow prior to going to sleep at night. The Sleep Time mobile application will gather sleep data using body movements throughout the night. A handout with instructions on how to use the application and for how long will be provided to you at your first session. You will also participate in brief questionnaires and neuropsychological tests that look at your attention and memory after regular sleep and after partial sleep deprivation.

Voluntary Consent

Participation in this study is voluntary, and you are free to decide not to participate or to withdraw from this study at any time. If you choose to participate, you may withdraw at any time by notifying the student research. Upon your request to withdraw, all information pertaining to you will be destroyed.

Confidentiality

All of the information will be confidential. The information will be considered only in combination with that of other subjects. If you choose to withdraw, all information pertaining to you will be destroyed. The information obtained in the study may be published in scientific journals or presented at scientific meetings, but it will not be linked to identifiable information.

Risks and Benefits

There are no direct benefits from participating in this study. The literature indicates that partial sleep deprivation may affect people's ability to function. Therefore, it is advised that you do not consume alcohol, use other substances, or operate a motor vehicle during your participation in this study after you have been partially deprived of sleep over two nights. The contact information of the Principal Investigator and Faculty Sponsor are provided below regarding any issues that may arise during participation.

Student Researcher:
Stella Kim, M.A.
Clinical Psychology Doctoral Student
Uhler Hall, 1020 Oakland Ave.
Indiana, PA 15705
(724) 357-6228

Faculty Sponsor:
David LaPorte, Ph.D.
Professor, Director of Clinical Training
Uhler Hall, 1020 Oakland Ave.
Indiana, PA 15705
(724) 357-4524

Informed Consent Form (continued)

If you are willing to participate in this study, please sign the statement below and return it to the student researcher. Take the extra unsigned copy with you. If you choose not to participate, please give the unsigned copies to the student researcher.

VOLUNTARY CONSENT FORM:

I have read and understand the information on the form and I consent to volunteer to be a subject in this study. I understand that my responses are completely confidential and that I have the right to withdraw at any time. I have received an unsigned copy of this informed Consent Form to keep in my possession.

Name (PLEASE PRINT): _____

Signature: _____

Date: _____

I certify that I have explained to the above individual the nature and purpose, the potential benefits, and possible risks associated with participating in this research study, have answered any questions that have been raised, and have witnessed the above signature.

_____ _____

Date Investigator's Signature

APPENDIX E
Debriefing Form

Thank you for your participation in this study. This study sought to examine the effect of partial sleep deprivation on declarative memory in people with a history of at least one mild traumatic brain injury. The objective measures of sleep, subjective measures of sleepiness, and performance on the attention and memory tasks will be analyzed for significant differences between the normal population and mild traumatic brain injury population.

Research indicates that both sleep deprivation and mild traumatic brain injuries are growing public concerns that lead to chronic health problems, cognitive impairments, behavioral changes, and even mortality. Despite these consequences, sleep deprivation remains prevalent among Americans in the U.S., and mild traumatic brain injuries often go unreported. Understanding that a basic biological function such as sleep may hide or reveal residual deficits from a history of mild traumatic brain injuries (such as memory impairments) may encourage health professionals to conduct a more thorough evaluation and the public to take greater precaution to decrease the likelihood of future brain injuries.

If your concerns are such that you would now like to have your data withdrawn, please inform the research assistant and we will do so. Questions about your participation in the study may be directed to me at s.h.kim@iup.edu, or my faculty advisor, Dr. David LaPorte, at laporte@iup.edu. If you have questions about your rights as a research subject, you may contact Indiana University of Pennsylvania's Institutional Review Board at irb-research@iup.edu.

Thank you again for your participation.

Sincerely,

Stella Kim, M.A.
Student Researcher

David J. LaPorte, Ph.D.
Faculty Sponsor