

Loma Linda University  
**TheScholarsRepository@LLU: Digital Archive of Research,  
Scholarship & Creative Works**

---

Loma Linda University Electronic Theses, Dissertations & Projects

---

12-1-2011

# Neuropsychological, Psychosocial, and Mood Outcomes Following Mild Traumatic Brain Injury

Julia L. Evans  
*Loma Linda University*

Follow this and additional works at: <http://scholarsrepository.llu.edu/etd>

 Part of the [Clinical Psychology Commons](#)

---

## Recommended Citation

Evans, Julia L., "Neuropsychological, Psychosocial, and Mood Outcomes Following Mild Traumatic Brain Injury" (2011). *Loma Linda University Electronic Theses, Dissertations & Projects*. 29.  
<http://scholarsrepository.llu.edu/etd/29>

This Thesis is brought to you for free and open access by TheScholarsRepository@LLU: Digital Archive of Research, Scholarship & Creative Works. It has been accepted for inclusion in Loma Linda University Electronic Theses, Dissertations & Projects by an authorized administrator of TheScholarsRepository@LLU: Digital Archive of Research, Scholarship & Creative Works. For more information, please contact [scholarsrepository@llu.edu](mailto:scholarsrepository@llu.edu).

LOMA LINDA UNIVERSITY  
School of Science and Technology  
in conjunction with the  
Faculty of Graduate Studies

---

Neuropsychological, Psychosocial, and Mood Outcomes Following Mild  
Traumatic Brain Injury

by

Julia L. Evans

---

A Thesis submitted in partial satisfaction of  
the requirements for the degree of  
Master of Arts in Clinical Psychology

---

December 2011

© 2011

Julia L. Evans  
All Rights Reserved

Each person whose signature appears below certifies that this thesis in his/her opinion is adequate, in scope and quality, as a thesis for the degree Master of Arts.

---

Susan A. Ropacki, Associate Professor of Psychology

---

Brenda Bartnik-Olson, Professor of Radiology, School of Medicine

---

David A. Vermeersch, Professor of Psychology

## ACKNOWLEDGEMENTS

I would like to express thanks to Dr. Ropacki, my committee, and all who helped with data collection or served as a source of support during this project. I would also like to thank my husband, Jonathan Evans, my parents, Steve and Maryann Kroh, and my brother, Michael Kroh for constantly supporting me throughout my academic and professional career.

## CONTENT

Approval Page.....	iii
Acknowledgements.....	iv
List of Tables .....	vii
List of Abbreviations .....	ix
Abstract.....	xi
Chapter	
1. Introduction/Literature Review.....	1
Introduction.....	1
Traumatic Brain Injury: Description and Classifications .....	2
Neuropsychological Functioning Following Mild Traumatic Brain Injury.....	4
Predictive/ Mediating Factors of Outcome .....	8
TBI Severity.....	9
Demographic Factors .....	10
Cognitive Factors .....	14
Psychological Factors .....	14
Functional Outcomes .....	17
Objective.....	19
Hypotheses.....	20
2. Materials and Methods.....	21
Inclusion/ Exclusion Criteria .....	21
Materials .....	24
Neuropsychological Measures .....	24
Psychological and Life Satisfaction Measures .....	25
Security .....	27
Statistical Analysis.....	27
3. Results.....	30
Demographics .....	30

Description of sample .....	30
Confirmation of the Cognitive Effects of mTBI.....	31
Cognitive outcomes following mTBI .....	31
Coping Meditational Analysis .....	34
MTBI related to differences in coping.....	34
Coping style as a predictor of cognitive and mood outcomes .....	35
Coping as a Mediator of cognitive and mood outcomes.....	38
Quality of Life and Mood Meditational Analysis.....	38
MTBI related to differences in quality of life and mood .....	38
Quality of life as a predictor of cognitive and mood outcomes .....	44
Quality of life as a Mediator of cognitive and mood outcomes.....	52
4. Discussion.....	59
References.....	67

## TABLES

Tables	Page
1. Demographic Characteristics of the Sample .....	31
2. Neuropsychological Performances between groups.....	33
3. MTBI as a predictor of coping .....	34
4. Coping styles that are significantly predictive of neuropsychological and mood outcome .....	37
5. Mood and Quality of Life between groups.....	41
6. TBI as a predictor of neuropsychological outcome.....	42
7. TBI as a predictor of mood and quality of life .....	43
8. Overall QOL as a predictor of neuropsychological and psychological outcome .....	46
9. Physical QOL as a predictor of neuropsychological and psychological outcome .....	47
10. Psychological QOL as a predictor of neuropsychological and psychological outcome .....	48
11. Independence QOL as a predictor of neuropsychological and psychological outcome .....	49
12. Social QOL as a predictor of neuropsychological and psychological outcome .....	50
13. Environmental QOL as a predictor of neuropsychological and psychological outcome .....	51
14. Mediation models of digit span as an outcome measure following TBI .....	54
15. Mediation models of digit span forward as an outcome measure following TBI .....	55
16. Mediation models using QOL as mediators of attention and immediate memory for contextually related information following TBI.....	56



17. Mediational models using QOL measures as mediators of anxiety following TBI .....	57
18. Mediational models using QOL measures as mediators of depression following TBI .....	58

## ABBREVIATIONS

ADHD	Attention Deficit Hyperactivity Disorder
ANOVA	Analysis of Variance
BAI	Beck Anxiety Inventory
BDI-II	Beck Depression Inventory, Second Edition
BYI-II	Beck Youth Inventories, Second Edition
CPT-II	Conners' Continuous Performance Task – II, computer version
DKEF-S	Delis-Kaplan Executive Function System
DS	Digit Span
DSC-PWI	Dynamic susceptibility contrast perfusion weighted MRI
FSIQ	Full Scale Intelligence Quotient
LLU	Loma Linda University
LMI	Logical Memory I
LMII	Logical Memory I
MRI	Magnetic Resonance Imaging
MRSI	Magnetic resonance spectroscopic imaging
mTBI	Mild Traumatic Brain Injury
PIQ	Performance Intelligence Quotient
QOL	Quality of Life
RAVLT	Rey Auditory Verbal Learning Test
RCFT	Rey Complex Figure Test
TBI	Traumatic Brain Injury
TMSE	Transactional Model of Stress and Emotion

VIQ	Verbal Intelligence Quotient
WAIS-IV	Wechsler Adult Intelligence Scale, Fourth Edition
WAYS	Ways of Coping Questionnaire
WCST	Wisconsin Card Sort Test
WCST-64	Wisconsin Card Sort Test – 64 card version
WHO	World Health Organization
WHOQOL-100	World Health Organization Quality of Life Measure
WISC-IV	Wechsler Intelligence Scale for Children, Fourth Edition
WMS-III	Wechsler Memory Scale, Third Edition
WTAR	Wechsler Test of Adult Reading

## ABSTRACT OF THE THESIS

### Neuropsychological, Psychosocial, and Mood Outcomes Following Mild Traumatic Brain Injury

by

Julia L. Kroh

Master of Arts, Graduate Program in Clinical Psychology  
Loma Linda University, December 2011  
Dr. Susan A. Ropacki, Chairperson

Traumatic brain injury (TBI) in adolescents and adults can result in cognitive, emotional, behavioral and neurological deficits that can persist more than a year after an injury. The aim of the current preliminary study was to use a comprehensive neuropsychological assessment to determine the nature of cognitive impairments and their relationship with specific psychosocial factors, including coping skills and perceived quality of life, following mild TBI (mTBI). Neuropsychological tests administered measured intelligence, pre-morbid intelligence, executive functioning, verbal memory, complex visual construction and non-verbal memory, sustained attention distractibility, and vigilance, verbal learning and memory, fine motor speed, and novel problem solving and executive functioning. Psychological and life satisfaction measures assessed perceived quality of life, coping style, anxiety, and depression. MTBI subjects showed decreased attention, verbal and non-verbal memory, quality of life, and increased depression and anxiety when compared with healthy controls. Additionally, it was found that quality of life mediated the relationship between head injury and depression, anxiety, and attention. These findings may suggest that psychotherapy interventions may be able to improve quality of life and aspects of cognition following TBI.

**CHAPTER ONE**  
**INTRODUCTION/ LITERATURE REVIEW**

**Introduction**

Traumatic brain injury (TBI) represents a significant public health and fiscal challenge, as approximately 1.5 million brain injuries occur each year in the United States and approximately 5 million Americans are living with disabilities related to those injuries (Xiong, Mahmood, Chopp, 2010). The annual cost of TBI in the United States exceeds \$56 billion (Xiong, et. al., 2010). The majority of these brain injury cases (70-80%) are mild in both initial severity and outcome, and many experience a complete resolution of symptoms (Arciniegas et. al., 2005). The cognitive sequelae following mild TBI (mTBI) is commonly more subtle and less often recognized than in the moderate or severe TBI population (Arciniegas et. al., 2005).

The mTBI patient may be overlooked by health care providers, educators and researchers due to the mild nature of the injury and symptomatology when compared to the more complex impairments following a moderate or severe brain injury. Up to 20% of mTBI individuals are left with chronic post-concussive syndrome, with related cognitive, emotional, behavioral and neurological deficits that will persist more than a year following the injury (Arciniegas et. al., 2005). Post-concussive syndrome describes a set of symptoms including cognitive, physical, and emotional/ behavioral dysfunction that result from TBI (Arciniegas et. al., 2005). As noted by Arciniegas (2005), typical acute and/or chronic post-concussive symptoms include cognitive problems such as attention, memory and executive dysfunction. Additionally,

emotional and behavioral problems were noted including increased irritability, anxiety, depression, affective lability, apathy and impulsivity (Arciniegas et. al., 2005). There is a body of literature devoted to understanding the cognitive changes following mild to severe TBI and the resultant deficits. However, psychological dysfunction and its correlation to cognition following TBI it is not as clearly understood and the question of why individuals with similar injuries experience different neuropsychological deficits remains unanswered.

### **Traumatic Brain Injury: Description and Classifications**

Approximately 1.4 million individuals sustain a TBI each year in the US (Tsushima et. al., 2009). Within this patient population, males are about twice as likely as females to suffer from a TBI, although it has been reported that female mortality rates are 1.28 times greater than males (Tsushima et. al., 2009). The incidence of TBI occurs most often in young adulthood and in old age; there is significant evidence that age negatively correlates with poorer prognostic outcomes (Stapert et. al., 2006). Falls are the primary cause of TBI in children and elderly, and it is estimated that 64% of TBIs suffered by infants are a direct result of child abuse (Williamson et. al., 1996). Elderly patients are more likely than young TBI patients to develop traumatic mass lesions, including subdural hematomas and intra-cerebral hemorrhage from mild to moderate TBI (Stapert et. al., 2006).

A formal definition of mild TBI is given by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (Kwok et. al., 2008). According to this definition,

mild TBI implies that a patient has a traumatically induced physiological disruption of brain function which is marked by at least one of the following: (1) loss of consciousness of approximately 30 minutes or less; (2) after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and (3) post-traumatic amnesia (PTA) not longer than 24 hours (Kwok, 2008). The GCS assesses neurological domains including verbal response, eye opening, and motor response following injury and has been found useful for predicting neurobehavioral outcome (Lucas et. al., 2006). PTA is the period following the TBI that is characterized by disorientation, confusion, and retrograde and anterograde amnesia (McGhee et. al., 2006). Anterograde amnesia and disorientation are typically assessed over a period of several days following the injury and may consist of evaluations of orientation and memory. The Westmead Post-Traumatic Amnesia Scale (WPTA) is a measure of anterograde amnesia and disorientation that is frequently used to assess PTA.

TBI may result in focal, multifocal, or diffuse cerebral dysfunction and typically involves structures and systems beyond the initial site of impact (Lucas et. al., 2006). Brain damage that is the result of closed head injury typically occurs in two stages, a primary injury followed by a secondary injury. Primary injuries result from initial damage whereas secondary injuries typically occur in response to the cascade of events that follow a primary injury. The primary injury in mTBI is most typically diffuse axonal injury (DAI), in which axons are damaged or destroyed by acceleration and deceleration forces acting on axonal bundles and blood vessels, resulting in damage to the white matter (Kwok, 2008). The disruption of consciousness following TBI seems to be related to the extent of DAI (Williamson et. al., 1996).

In addition to DAI, brain contusions, lacerations, and disruption of vasculature can occur as primary injuries (Lucas et. al., 2006). Bruising is often seen at the original site of damage and is often referred to as a coup lesion. The pressure experienced at impact often causes the brain to rebound and hit the skull opposite the initial blow, causing an even larger lesion, known as the contre-coup lesion (Lucas et. al., 2006). Secondary injuries include ischemia, edema, hypoxia, epilepsy, increased intracranial pressure, and neurotransmitter and metabolic changes associated with damage to neurons (Lucas et. al., 2006).

### **Neuropsychological Functioning Following Mild Traumatic**

#### **Brain Injury**

Long-term neuropsychological outcomes following mTBI are reasonably understood and are important to consider. Specifically, reduced capacity for learning, slowed information processing, and disruption in complex integrative functions have been found to be resultant of mTBI (Millis et. al., 2001). One meta-analytic study reviewed 28 publications that summarized injury severity and time post injury as they related to neurocognitive domains in the pediatric population (Babikian & Asarnow, 2009). This meta analysis revealed that longitudinal studies of neurocognitive outcomes following mTBI in pediatric populations do not show changes in verbal skills as measured by the Verbal Intelligence Quotient (VIQ), Full Scale Intelligence Quotient (FSIQ), attention, working memory, or visual perceptual functioning over time (Babikian & Asarnow, 2009). However, within this meta-analytic study, there are no studies that assessed fluency, memory, or inhibition across time. Another interesting finding within



this study by Babikian and Asarnow (2009) is that the mTBI group appeared to make significant gains in nonverbal/performance-based skills as measured by the Performance Intelligence Quotient (PIQ) and processing speed, which was unexpected as these domains are not typically improved with practice (Babikian & Asarnow, 2009). Specifically, it was found that small to moderate effects were found for VIQ, PIQ, processing speed, and visual perceptual functioning when subjects were assessed at three time points: 0-5 months post injury, 6-23 months post injury, and 24+ months post injury (Babikian & Asarnow, 2009). Of note, significant improvements in immediate visual memory were only observed 0-5 months post injury.

Additionally, it is reported in the literature that the basic components of attention, including vigilance and sustained attention, as well as the superordinate components of attention control, including selective attention, inhibition, shifting, and divided attention are impaired following severe TBI (Galbiati et. al., 2009). According to various studies, attentional impairments observed following mild to severe TBI may be the result of reduced rate or capacity of controlled processing, or dysfunctional higher- level processes (Ziino et. al., 2006). Research utilizing tests measuring focused attention, mental speed and control, and forced choice reaction time revealed that severe TBI patients are generally able to cope with interference caused by distracting stimuli, although they tend to require more time (Bate et. al., 2001). Another study found slowed processing speed associated with mild TBI as well as greater variability in processing performance, suggesting impairment and insufficient capacity to complete speed-related tasks (Meyerson et. al., 2009). This literature suggests that impairments in divided and focused attention may result from decreased speed of processing rather than insufficient cognitive

capacity. However, it is important to note that pre-injury ADHD and behavioral problems are seen at higher rates in children who experience TBI; these problems are seen at the highest rates in children with severe TBI (Babikian & Asarnow, 2009). Thus post-injury testing in this population may reflect a pre-existing attentional problem. Kwok, Lee, Leung, and Poon (2008) report that in mTBI patients, divided attention was significantly poorer than in healthy controls immediately post-injury but recovered in one month and returned to normal within 3 months post- injury. However, this same group found that sustained attention remained impaired for the extent of the study, which was 3 months post-injury. Additionally, Chan (2005) found that patients with mTBI performed significantly worse on measures of sustained attention when tested at an average of 25 months post-injury. It is currently thought that the extent of attentional deficits a patient experiences post-TBI is correlated with the patient's age as well as severity of the injury. The frontal and temporal regions in the child and adolescent brain are immature, and continue to develop anatomically and functionally beyond adolescence and may be more vulnerable to trauma. A focal lesion in these areas can cause structural and functional changes, thus interfering with the development of these important attentional processing areas (Galbiati et. al., 2009). Further research outlining the implications of mTBI on the developing adolescent brain as it relates to attention and processing deficits is necessary to understand differing cognitive outcomes following mTBI.

There is evidence that suggests that language capacity, including semantic and phonemic fluency and confrontation naming abilities, may be impaired following mTBI. King, Hough, Vos, Walker, and Givens (2006) assessed the word-finding and word-retrieval capacity of mTBI patients when compared to non-injured control subjects. It

was revealed that mTBI patients were significantly slower and less accurate than controls when naming nouns (King et. al., 2006). Additionally, mTBI patients were significantly faster at completing sentences with nouns than with verbs. King and colleagues (2006) suggest that this performance discrepancy may be explained by the fact that noun naming in sentence tasks is easier than verbal naming tasks. Kwok, Lee, Leung, and Poon (2008) reported that immediately post-injury, mTBI patients' verbal fluency, specifically semantic fluency, was significantly poorer than that of healthy controls. At 1-month post-injury mTBI patients' verbal fluency ability was significantly improved, but was still significantly different than the performance of healthy controls. This further highlights the potential short and long-term complications of mTBI and the importance of researching language impairments following brain injuries. It is an aim of the current study to investigate what factors may mediate this relationship between mTBI and impact on language functions.

Additional cognitive impairments have been revealed in empirical studies with mTBI patients. Visuospatial functioning and visuoconstructional skill, for example, is shown to decrease following mTBI. Specifically, it is reported that symptomatic mTBI patients show deficits in complex visual information processing as assessed by Event-Related Potentials (ERPs) (Lachapelle, Bolduc-Teasdale, Ptito, & McKerral, 2008). Memory and executive functioning declines have also been found following mTBI. For example, Belanger, Spiegel, and Vanderploeg (2010) revealed that patients who presented with multiple occurrences of mTBI performed poorer on measures of delayed memory and executive functioning than patients who presented with only one occurrence of mTBI. Moreover, severity has been found to inversely correlate with executive

functioning in children, including planning, goal setting and problem solving (Anderson & Catroppa, 2005). Additionally, children with severe TBI demonstrated slowed and significantly less accurate performance on cognitive flexibility tasks that were mentally demanding (Anderson & Catroppa, 2005).

While a significant amount of research has contributed to our current understanding of TBI, there is clearly a lack of research on factors that may act as possible mediators to cognitive outcome following mTBI. There is currently a dearth of research exploring the possibility of psychological factors, such as coping style and perceived quality of life, as mediating influences on cognition following mTBI. Additionally, more research is needed to better understand neuropsychological outcomes following mTBI, including language abilities, verbal and nonverbal memory, and executive functioning. A significant amount of current literature compares mTBI patients to moderate and severe TBI patients, which oftentimes underestimates the neuropsychological deficits and overestimates the cognitive capacity of mTBI patients. It is thus essential to examine psychological mediating factors and to compare mild TBI patients with healthy controls in order to add depth to the current body of research on the nature and outcome of cognitive functioning after mTBI and the possible impact of psychological factors on these outcomes.

### **Predictive/ Mediating Factors of Outcome**

Prognostic outcome following TBI can be described as the ability to predict a patient's function both psychologically and cognitively on a time continuum. This prognosis is valuable and can be utilized to develop expectations and treatment strategies

post-TBI. The ability to statistically correlate psychological factors with cognitive benchmarks may offer the patient and caregiver a better understanding of cognitive potential or deficits based on neuropsychological evaluation.

### ***TBI Severity***

As previously discussed, TBI severity is predictive of resultant deficits in cognitive sequelae, including attention, processing speed, and executive functioning. Injury severity is highly predictive of neuropsychological outcomes and is an important predictor of the extent of cognitive deficits following TBI. Babikian and Asarnow (2009) report longitudinal studies of mild, moderate, and severe TBI patients that were assessed at 3 time points: 0-5 months post-injury, 6- 23 months post-injury, and 24+ months post-injury. Specifically, it is found that in the pediatric population, mild TBI patients generally demonstrate few impairments in general intelligence, attention and executive skills, and memory, and tend to show some recovery in these domains two years post injury (Babikian & Asarnow, 2009). Within the moderate pediatric TBI population, it is found that post-injury neurocognitive impairments involve several domains, including general intellectual functioning, executive skills, processing speed, attention, verbal fluency, inhibition, and problem solving (Babikian & Asarnow, 2009). In contrast, the authors reported that in the moderate TBI group, working memory, memory and visual perceptual skills were not statistically different from non-injured controls. Additionally, Babikian and Asarnow (2009) reported statistically significant improvements in FSIQ, PIQ, processing speed, attention, problems solving, and visual perceptual functioning within the first 2 years following moderate TBI (Babikian & Asarnow, 2009). No

cognitive changes were observable after two years post injury in the pediatric moderate TBI group (Babikian & Asarnow, 2009). The severe TBI pediatric patients showed significant impairments in nearly all neurocognitive domains at two years post-injury. When severe TBI patients were compared to non-injured controls, as well as mild and moderate TBI patients, the severe TBI patients demonstrated significantly more cognitive deficits across time points. Specifically, deficits were noted within general intellectual functioning, verbal memory, visual perceptual skills, executive functioning, verbal fluency, processing speed, attention, problem solving, and working memory domains (Babikian & Asarnow, 2009). At 6-23 months post-injury, it was found that moderate to large improvements were observed in general intellectual functioning (FSIQ), performance IQ (PIQ), processing speed, and visual perceptual functioning. Interestingly, no neurocognitive changes were observed after 23 months.

### ***Demographic Factors***

Demographic factors including age, gender, education, and ethnicity have also been implicated as important predictors of long-term neurocognitive outcomes. One longitudinal study found that five years after injury, a substantial portion of individuals with moderate to severe TBI continue to show impairments in learning, memory, complex attention, and processing speed (Millis et. al., 2001). Age was the only significant predictor of these cognitive changes following injury. Specifically, for every increase of 10 years of age at the time of injury, the risk of subsequent neuropsychological decline went up 4.97 times (Millis et. al., 2001).

Education and cognitive reserve have also been studied as a possible predictors of outcome following TBI. Shames et. al (2007) found that higher education levels were positively correlated with an individual's likelihood of returning to work following mild to severe TBI. Cognitive reserve is an important aspect of an individual's cognitive potential and likely has important implications in predicting functional and neuropsychological outcomes following TBI. Kesler, Adams, and Bigler (2003) investigated cognitive reserve in 25 TBI patients by examining the relationships between total intracranial volume, education, and post-injury cognitive outcomes. The authors concluded that larger pre-morbid brain volume and higher education level may decrease vulnerability to cognitive deficits following TBI. The WTAR has been considered to be an important assessment tool in measuring cognitive reserve (Hank et. al., 2008). Hank and colleagues (2008) reported the WTAR to be predictive of 1-year outcomes following TBI, including prediction of handicap, functional independence, and employability.

Gender has also been analyzed as a possible predictor of outcome following TBI. The predictive role of gender was identified in a study by Brewster and colleagues (2009). These researchers found that women performed significantly better on the Short Category Test, which measures executive functions, and the Trail Making Test, which assesses processing speed, following mTBI. At fifteen months following injury, the women showed better executive processing than the men. Donders and Woodward (2003) studied gender as a moderator of memory following mild to severe pediatric TBI. The authors found that boys with TBI performed worse than girls with TBI and worse than healthy controls on a measure of memory. Donders and Woodward (2003) concluded that the effect of TBI on children's memory appeared to be moderated by

gender. Conversely, in another study of moderate to severe adult TBI patients, it was found that gender had no significant influence on mortality or unfavorable outcomes (Leitgeb, Mauritz, Branzinova, Janciak, Majdan, Wilbacher, & Rusnak, 2011). Overall, the authors concluded that female gender was not an independent risk factor for inpatient mortality post-TBI. In a study by Morrison, Arbelaez, Fackler, De Maio, and Paidas (2004) it was found that there were no statistically significant differences between boys and girls in total hospitalization length of stay or functional outcome following mild to severe pediatric TBI. Specific outcome variables assessed included vision, hearing, speech, feeding, bathing, dressing, walking, toileting, cognition, and behavior. This group concluded that girls do not have a better outcome following pediatric TBI than boys and for every outcome measure there was a trend toward girls performing worse than boys (Morrison et. al., 2004). Overall, the research evaluating the predictive nature of gender on outcomes measures following TBI is inconsistent with a dearth of research paying specific attention to the mild TBI population.

Ethnicity may have important implications in cognitive outcomes following TBI. In a meta-analytic study by Gary and colleagues (2009) it was found that prior to mild to severe TBI, African Americans and Hispanics were generally younger, male, more likely to be unemployed and unmarried, earned less money and were less likely to have health insurance than Caucasians. This same study found that African Americans and Hispanics were 3-4 times more likely than Caucasians to acquire TBIs through acts of violence. Additionally, patients who were less acculturated, espousing more traditional cultural values and beliefs, and scored lower than Caucasians on a composite measure of overall neuropsychological test performance (Gary et. al., 2009). Specifically, poorer



neuropsychological functioning was observed on tests of attention, orientation, language, visuomotor/processing speed, visuospatial/constructional skills and memory. Of note, this group of less acculturated individuals performed poorer than Caucasians even after controlling for injury severity, time since injury, age, sex, years of formal education, and socioeconomic status (Gary et. al., 2009). Overall, Gary and colleagues (2009) indicated ethnicity may be related to differences in functional outcomes, community integration and quality of life following TBI. In contrast, Proctor and Zhang (2008) researched the performance of European Americans, African Americans, and Latino/a Americans on tests of executive function following TBI and found no statistically significant impact of ethnicity on the Wisconsin Card Sort Test (WCST), a measure of cognitive flexibility and novel problem solving. In consideration of ethnicity as it relates to TBI, while there is research on ethnicity and some aspects of outcome, there is little research on ethnicity as a predictor of neuropsychological outcomes following mTBI.

The current preliminary study considered age, gender, education, and ethnicity as possible predictors of neuropsychological outcomes. It was a goal of the study to add important information to the current TBI literature about individual factors that may contribute to prognostic outcomes. There are numerous studies that have investigated the ways in which demographic factors including, age, gender, education, and ethnicity affect long-term neurocognitive outcomes following TBI. However, there is a lack of research investigating the possible interaction between these demographic factors and psychological functioning post mTBI. Currently there is a gap in the literature examining the possibility that demographic factors could be indirect markers of differences in psychological outcomes. Additionally, it is important to understand that research is

needed to evaluate the possible predictive nature of demographic factors on psychological and cognitive outcomes in the chronic post-concussive mTBI patient as research in this area is lacking. Establishing this possible relationship may provide insight into why some people who incur mTBI experience residual symptoms and other do not.

### ***Cognitive Factors***

Cognitive factors, such as premorbid intelligence and memory following the injury, may also play a critical role in predicting functional outcome following TBI, including return to work. O'Connell (2000) conducted a study involving 43 adult TBI patients in which the outcome variable was return to work and predictor variables included demographic, intellectual, and memory data. Specifically, independent variables included age, gender, race, education, occupation, Performance IQ, Verbal IQ, verbal and nonverbal memory. O'Connell (2000) found that age was negatively correlated with returning to work, whereas higher scores on measures of Performance IQ and verbal memory measures (indicating a higher level of cognitive capacity) were predictive of a greater likelihood of returning to work.

### ***Psychological Factors***

It is clearly established through research studies that an important relationship exists between psychological and cognitive functions. The literature in this area provides evidence that psychological factors can meaningfully impact cognitive functioning. For example, a study by Goodman, Knoll, Isakov, and Silver (2005) found a relationship between negative attitudes towards medication and decreased cognitive outcome,

specifically within working memory capacity, in schizophrenic patients. Yen, Cheng, Huang, Ko, Yen, and Chan (2009) studied the relationship between psychosocial adjustment and executive functioning in patients with bipolar disorder and schizophrenia in remission. The group indicates that poor psychosocial adjustment, as evidenced by unemployment, lacking reliable friends and leisure activities is associated with decreased quality of life (Yen et. al., 2009). The authors report that significant correlations exist between executive function, insight, and psychosocial adjustment among schizophrenic and bipolar patients (Yen et. al., 2009). Yen and colleagues (2009) also report a positive association between verbal memory and psychosocial function in bipolar patients. This study demonstrates the relationship between psychosocial function (an aspect of psychological well-being), and executive function and verbal memory (important neuro-cognitive tasks). The current literature supports the correlation between various psychological factors, including negative attitudes and psychosocial functioning, and cognitive factors, including working memory, verbal memory and executive functioning, in various mental health populations. However, there is currently a lack of research investigating the possible correlation between psychological factors, including coping process and perceived quality of life, in the mTBI population.

Psychological factors may mediate the relationship between well-being and cognition and predict long-term prognosis (outcome) following TBI. Studies have found that psychological factors, including coping process and perceived quality of life, impact functional outcomes, including return to pre-injury independent activities of daily living and cognitively dependent tasks such as work. Fontana and McLaughlin (1998) define coping as “the thoughts and acts that people use to manage the internal and external

demands posed by a stressful encounter.” Folkman and Lazarus (1984) have proposed the transactional model of stress and emotion (TMSE; Lazarus and Folkman, 1984) as a framework to better understand the process by which an individual copes with stressful external stimuli. Folkman and Lazarus (1988) explain that individuals make primary appraisals when initially faced with a stressor; the individual may appraise the stimuli as stressful, positive, controllable, challenging, or irrelevant. The individual will then assert a second appraisal of the situation; this appraisal typically evaluates the individual’s own coping resources and options available (Lazarus and Folkman, 1988). This secondary appraisal involves the individual’s ability to manage and ameliorate the problem. Keiffer and MacDonald (2011) state that within the TMSE model, coping is considered to be a “process of changing cognitive and behavioral efforts to manage either internal or external demands placed on an individual.”

In addition to coping style, perceived quality of life (QOL) may mediate the relationship between cognition and mTBI. Quality of life was defined by Awad and Voruganti (2000) as “feelings of well-being and satisfaction to issues related to standards of living such as housing, finances, and employment.” Quality of life has also been described as the gap between a patient’s expectations and achievements (Calman, 1984). The World Health Organization (WHO) defines quality of life as an individual’s perception of their position in life in the context of culture and value systems in which they live and in relation to their goals, expectations, standards, and concerns (World Health Organization, 1997). The WHO (1997) explains that QOL is an expansive concept encompassing a persons' physical health, psychological state, level of independence,

social relationships, personal beliefs and their relationships to salient features of the environment.

### ***Functional Outcomes***

Functional outcomes following TBI are critical to the patient's social, psychological, and economic welfare. Functional outcomes following injury may be defined as the level of an individual's ability to return to premorbid levels of daily functioning. Functional outcome following TBI may be measured as return to work (O'Connell, 2000), as well as self-care, locomotion, communication, and social cognition (Cullen, Park, & Bayley, 2008).

Tsaousides et. al. (2009) found that employment-related and general self-efficacy were strongly related to perceived quality of life. Specifically, TBI patients who reported greater confidence in their ability to meet the demands within the workplace and generally within their lives also reported higher levels of life satisfaction and perceived quality of life. A study by Brewster and colleagues (2009) found that following TBI, only psychological well-being predicted whether or not the patient returned to work, a high level cognitive activity. In another study which examined return to work as a functional outcome, it was found that greater injury severity was associated with decreased life satisfaction (Wood, 2006) and patients with more severe brain injury were the least likely to return to work (Fraser et. al., 2006). Another study found several important factors that were predictive of a mild TBI patient's eventual return to work (Guerin, Kennepohl, Leveille, Dominique, McKerral, 2006). This group found that the number of subjective complaints was significantly associated with the individual's

eventual return to work following TBI. Fraser et. al., 2006 reported that the group of TBI patients that was the most able to maintain complex professional work was more likely to have been female, had fewer alcohol problems, was less severely injured and demonstrated better neuropsychological functioning. Additionally, Shames et. al (2007) reported that patients with more social interaction and pre-injury occupations that included more decision-making capacity were more likely to return to work.

The research is varied with regard to the psychological deficits that follow TBI. Goldstein and Levin (2001) found that within a sample of individuals over the age of 50 who had experienced uncomplicated mild head injury, there were no persistent cognitive deficits. However, these researchers found that although the sample demonstrated normal cognitive functioning, mild TBI patients reported significantly more depressive complaints, somatic concerns, and anxiety than non-injured control subjects. These psychological factors may seriously impact an individual's ability to return to pre-morbid levels of cognitive functioning in terms of critical thinking and ability to work. Another study, conducted in Quebec, Canada, found several important factors that were predictive of a mild TBI patient's eventual return to work (Guerin, Kennepohl, Leveille, Dominique, and McKerral, 2006). The group found that increased age, number of subjective complaints and the presence of public insurance significantly correlated with the individual's eventual return to work following TBI. Public insurance in Canada reportedly provides patients salary replacement and access to special medical services following an injury (Guerin et. al., 2006). Additionally, the group found there was no correlation between a post-TBI diagnosis of a mood or anxiety disorder and likelihood of returning to work. However, it should be noted that the individuals enrolled in this study

were actively engaged in an intervention program, which provided psychological support. Therefore, it is unclear whether or not psychological factors, including depression and anxiety, mediate the relationship between cognitive deficits following TBI and return to functionality, as measured by return to work.

### **Objective**

This inter-departmental study is part of a larger study, which aims to use 3D magnetic resonance spectroscopic imaging (MRSI) and dynamic susceptibility contrast perfusion weighted MRI (DSC-PWI) to determine if 1) prolonged cerebral metabolic alterations occur in children, adolescents, and adults with persistent neurocognitive deficits following a mild TBI and 2) if regions of altered cerebral metabolism are associated with changes in tissue perfusion. The aim of the current preliminary study is to use a comprehensive neuropsychological assessment to determine the nature of cognitive impairments and their relationship with specific psychosocial factors, including coping skills and perceived quality of life, following mild TBI. Understanding this possible relationship is necessary to establish effective treatment strategies, which may involve a focus on psychological factors. If it is determined that psychological qualities, such as coping skills and perceived quality of life, do mediate the relationship between injury and neurocognitive outcome, then psychotherapy interventions may be able to improve cognitive outcome following TBI.

## **Hypotheses**

The hypothesis of this study was that psychological factors, including coping style and perceived quality of life, would be predictive of better cognitive and mood outcomes following mild TBI. Specifically, it was predicted that the use of problem-focused coping processes (confrontive coping, planful problem-solving) would be associated with better performance on neuro-cognitive tests following mTBI. With regard to emotion-focused coping, it was hypothesized that distancing and wishful thinking, which can be considered an avoidant mechanism, would be associated with poorer overall performance on neuropsychological measures. It was predicted that seeking social support and positive reappraisal, which are associated with a positive evaluation of emotions, would be associated with better overall neuropsychological performance. It was hypothesized that coping styles would mediate the relationship between mTBI and neuropsychological and mood outcomes. It was also hypothesized that mTBI subjects would report poorer quality of life when compared to healthy controls and that quality of life would mediate the relationship between mTBI and neuropsychological and mood outcomes



## **CHAPTER TWO**

### **MATERIALS AND METHODS**

Patients were identified either through the LLU Behavioral Health Institute Intake Department or through the LLU department of Neurology. Once a potential candidate had been identified, the potential candidate and/or family members were interviewed, screened for inclusion/exclusion criteria, and enrolled by obtaining the properly signed informed written consent. If the patient's injury occurred within three months prior to testing the injury was considered recent; if the TBI occurred more than three months prior to testing, the injury was considered remote. If the patient was a minor, written consent was obtained from the parent or legal guardian and verbal assent was obtained from the patient. If a patient failed to meet necessary criteria for inclusion into the MRI portion of the study, the patient was still eligible to receive neuropsychological testing, providing that necessary inclusion criteria for neuropsychological assessment were met. This study included 18 pediatric and adult TBI subjects and 12 adult control subjects that meet the inclusion and exclusion criteria.

#### **Inclusion/ Exclusion Criteria**

The inclusion criteria for TBI subjects are:

- Patients were at least 10 years of age without gender or ethnic restrictions. There was an upper age limit of 65.
- Diagnosis of post-concussive syndrome or mild traumatic brain injury, and suspected cognitive change following head injury as determined by the referring physician or supervising neuropsychologist.

- Eligibility for MRI per routine screening checklist in order to confirm that the patient is physically able to undergo an MRI, as determined by the referring neurologist or radiologist.

The MRI exclusion criteria are:

- History of a known neurological disorder prior to qualifying injury.
- Renal insufficiency or known history of kidney disease.
- Previous allergic reaction to gadolinium MR contrast.

The neuropsychological assessment exclusion criteria are:

- History of psychiatric disorder.

Twelve age-matched normal volunteers were targeted for recruitment as control subjects. Control subjects were recruited from Loma Linda University and/or Medical Center staff, student or resident populations as well as from family members of recruited TBI subjects. The final sample included 18 mTBI subjects and 12 healthy controls. The mean age of mTBI subjects was 29.22 and the mean age of controls was 29.58. The mTBI group consisted of 12 males and 6 females, while the control group included 10 males and 2 females. Additionally, the mTBI group had an average of 12 years of education and the control group had an average of 13.75 years of education. The estimated premorbid IQ (as measured by the WTAR) for the mTBI group was 103.5 and 110.58 for the control group.

Control Subject Inclusion Criteria:

- At least 10 years of age without gender or ethnic restrictions. There was an upper age limit of 65.

- Eligibility for MRI per routine screening checklist.

Control Subject Exclusion Criteria:

- MRI Department staff or subordinate of project Investigator.
- History of neurosurgical intervention, excluding the placement of ventriculostomy shunt.
- History of a prior known brain injury with associated loss of consciousness.
- History of a known neurological disorder.
- History of psychiatric disorder.
- Renal insufficiency or known history of kidney disease.
- Previous allergic reaction to gadolinium MR contrast.
- History of known claustrophobia.

Review of the medical record was performed to obtain patient characteristics such as age, gender, date of birth, medical history, date of injury, Glasgow coma score (GCS; initial, admission, and lowest post-resuscitation), Abbreviated Injury Score (AIS), pupillary reaction at admission, presence of associated injuries, length of patient's unconsciousness, length of post-traumatic amnesia (PTA), evidence of hypoxia, duration of ventilatory support, time to follow commands, medication regimen, and duration of stay in the ICU. In addition, the results of any outpatient neurological or neuropsychological tests prior to involvement in this study were noted. Relevant demographic information was collected from the control subjects through the administration of a medical history form at the time the patient was consented. All TBI

and control subjects were administered an assessment by a trained member of the research team.

## **Materials**

Subjects were administered a variety of neuropsychological and life satisfaction measures.

### *Neuropsychological Measures*

The Wechsler Adult Intelligence Scale, Fourth Edition (WAIS-IV) was used to measure intelligence in adult participants ages 16 and older (Wechsler, 2008). The Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV) was used to measure intelligence in participants ages 10-15 (Wechsler, 2003). Prorated estimates of verbal comprehension, perceptual reasoning, working memory, and processing speed were measured using select subtests. The WAIS-IV and WISC-IV subtests that were employed in this study include: Symbol Search, Digit Span subtest (forward and backward), Information, Matrix Reasoning, Similarities, Block Design, and Arithmetic. Selected subtests from the Delis-Kaplan Executive Function System (DKEF-S) were given to measure aspects of executive functioning (Delis, Kapan, & Kramer, 2001). Specifically, the Trails Subtest assessed processing speed, motor speed, and mental flexibility and the Verbal Fluency Subtest measured semantic fluency, phonemic fluency, and category switching( aspects of mental flexibility). The Logical Memory subtest (I and II) from the Wechsler Memory Scale, Third Edition (WMS-III; Wechsler, 1997) was utilized to assess immediate and delayed memory for contextual information. The

Wechsler Test of Adult Reading (WTAR) was employed to estimate the subject's level of intellectual functioning before the onset of injury (Wechsler, 2001). The WTAR is a test of single-word reading that has been found to be a reliable measure of pre-morbid cognitive functioning in addition to outcomes following TBI (Hanks, Millis, Ricker, Giacino, Nakese-Richardson, Frol, Novack, Kalmar, Sherer, & Gordon, 2008). Visuoconstruction with executive, memory, and recognition components was measured through the use of the Rey Complex Figure Test (RCFT) (Meyers & Meyers, 1995). The Conners' Continuous Performance Task – II, computer version (CPT-II) (Conners, 2000) was given to test sustained attention, distractibility, and vigilance. Verbal learning and memory was assessed using the Rey Auditory Verbal Learning Test (RAVLT-II) (Schmidt, 1996). Fine motor speed was tested by way of the Grooved Pegboard (Trites, 2002). Novel problem solving was measured with the Wisconsin Card Sort Test – 64 card version (WCST-64) (Grant & Berg, 2000).

### ***Psychological and Life Satisfaction Measures***

Perceived quality of life was measured with the use of the World Health Organization Quality of Life Measure (WHOQOL-100) (World Health Organization, 1997). The WHO, in collaboration with 15 centers around the world, has developed the World Health Organization Quality of Life Instrument (WHOQOL-100), a standardized measure of quality of life. The instrument assesses an individual's subjective overall QOL, physical health, psychological state, level of independence, social relationships, personal beliefs, and their relationship to their environment. The WHOQOL-100 Overall QOL Domain assesses a person's overall QOL, health and well-being. The WHOQOL-

100 Physical Domain assesses an individual's perceived pain and discomfort, energy and fatigue, and sleep and rest. The WHOQOL-100 Psychological Domain measures positive feelings, thinking, learning, memory, concentration, self-esteem, body-image and appearance, and negative feelings. The WHOQOL-100 Level of Independence Domain examines a person's mobility, activities of daily living, dependence on medication or treatments, and working capacity. The WHOQOL-100 Social Relationships Domain includes an assessment of personal relationships, social support, and sexual activity. The WHOQOL-100 Environment Domain includes questions about physical safety and security, home environment, financial resources, health and social care availability and quality, opportunities for acquiring new information and skills, participation in and opportunities for recreation and leisure, physical environment, and transport. Finally, the WHOQOL-100 Spirituality/Religion/Personal Beliefs Domain examines the person's personal beliefs and how they affect quality of life.

The Ways of Coping Questionnaire (WAYS) is a process measure containing a range of thoughts and acts employed by people when dealing with internally or externally stressful situations (Keiffer and MacDonald, 2011). The WAYS (Folkman & Lazarus, 2003) was given to understand the subject's coping style, including the thoughts and actions he or she uses to handle stressful encounters. The WAYS measures 8 different coping factors. Measured coping factors include "confrontative coping," which describes aggressive efforts to alter the situation, "distancing," involving cognitive efforts to detach oneself and to minimize the significance of the situation, and "self-controlling," which describes efforts employed to regulate one's feelings and actions (Folkman & Lazarus, 2003). Additional factors include "seeking social support," which describes one's efforts

to seek informational, tangible, and emotional support, “accepting responsibility,” whereby one acknowledges one’s own role in the problem and efforts to make it right, and “escape avoidance,” which describes wishful thinking and behavioral efforts to escape or avoid the problem. Final coping factors include “planful problem solving,” describing the deliberate problem-focused efforts used to alter the situation, coupled with an analytic approach to problem-solving, and “positive reappraisal,” or efforts employed to create positive meaning by focusing on personal growth (Folkman & Lazarus, 2003). Finally, psychological factors, including anxiety and depression in adult participants, were assessed by way of the Beck Anxiety Inventory (BAI) (Beck, 1993) and Beck Depression Inventory, Second Edition (BDI-II) (Beck, 1996), respectively. Participants under the age of 16 were given the Beck Youth Inventories, Second Edition (BYI-II) (Beck, Beck, and Jolly, 2005) depression and anxiety scales as subjective measures of depression and anxiety.

### **Security**

The study investigator kept all information obtained from the medical record review in a locked filing cabinet and password protected database. A study number replaced subject names and the PHI was removed.

### **Statistical Analysis**

A total of 18 TBI subjects and 12 healthy controls were included in analyses and all subjects met the study’s inclusion criteria. An a priori power analysis was completed using G\*Power 3.1 in order to assess the sufficiency of the sample size (Faul, Erdfelder,

Buchner, & Lang, 2009). Effect sizes ( $f^2$ ) for multiple regression are often defined by scores of 0.02, 0.15, and 0.35 which are termed small, medium, and large, respectively (Cohen, 1988).  $f^2$  is calculated with the  $R^2$  [Equation:  $f^2 = R^2/(1-R^2)$ ]. Using a conservative effect size estimate ( $f^2 = 0.15$ ), this study needs approximately 68 subjects to achieve a liberal power of 0.80. Using a liberal effect size estimate ( $f^2 = 0.35$ ) this study needs approximately 31 subjects in order to achieve a liberal power of 0.80. Baron and Kenny's (1986) model for testing mediation was used. Baron and Kenny (1986) have defined 4 steps in establishing mediation. Step 1 shows that the initial variable is correlated with the outcome; therefore establishing that there is an effect that may be mediated. Step 2 shows that the initial variable is correlated with the mediator, treating the mediator as though it were an outcome variable. Step 3 shows that the mediator affects the outcome variable; step 4 evaluates complete versus partial mediation.

The data analysis emphasized description and graphical statistics. Descriptive statistics included the mean, minimum/maximum values and associated 95% confidence intervals. Data was reported as mean (SD or range). For all tests, an alpha level of  $P < 0.05$  was taken to indicate significance. Differences in the nature and extent of cognitive deficits among TBI and control groups was analyzed using univariate regressions. Univariate regressions were also used to assess whether mTBI is a predictor of neuropsychological and mood outcomes, coping style, and QOL. Univariate regressions were also be used to evaluate whether coping style is predictive of neuropsychological and mood outcomes, and QOL. Additionally, univariate regressions were used to determine whether QOL is predictive of neuropsychological and mood outcomes. Multivariate regressions were utilized to determine whether coping style



mediates the relationship between TBI and neuropsychological and mood outcomes.

Multivariate regressions were also used to determine whether quality of life mediates the relationship between TBI and neuropsychological and mood outcomes.

## CHAPTER THREE

### RESULTS

#### Demographics

##### *Description of Sample*

The mTBI and control groups did not significantly differ in age, premorbid intelligence, or education (Table 1). The final sample included eighteen mTBI subjects and twelve control subjects, twelve male and six female mTBI subjects and ten male and two female control subjects; no difference was noted in distribution of gender between groups  $\chi^2(1) = 1.02, p = \text{n.s.}$  Two subjects in the mTBI groups are missing data for the WTAR VIQ; one subject discontinued the test as the result of significant frustration and one subject was tested by a clinician at the LLU Behavioral Health Institute who failed to administer the WTAR. With regard to checks for statistical assumptions, descriptive statistics were analyzed for each measure, including distribution, skewness, kurtosis, and assessment of outliers. All variables in the current analysis had normal distributions with normal skewness and kurtosis. Pairwise deletion was used in the current analyses due to the fact the current data was preliminary and the maximum amount of power was needed for all analyses. Thus, subtle differences will be noted in number of subjects within each analysis.

Table 1

*Demographic Characteristics of the Sample*

	N	Mean (SD)	F
Age			2.944
mTBI	18	29.22 (17.56)	
Control	12	29.58 (13.69)	
Gender			
mTBI			
Male	12		
Female	6		
Control			
Male	10		
Female	2		
WTAR VIQ			1.921
mTBI	16	103.50 (14.05)	
Control	12	110.58 (9.07)	
Education			.176
mTBI	18	12.00 (3.94)	
Control	12	13.75 (2.83)	

**Confirmation of the Cognitive Effects of mTBI***Cognitive Outcomes Following mTBI*

The mTBI performed significantly worse than healthy controls on a number of neuropsychological measures (Table 2). Specifically, the mTBI group (WAIS-IV/WISC-IV DS  $M=8.71$ ; WAIS-IV/WISC-IV DS For  $M=6.35$ ; CPT-II Omiss  $M=14$ ) performed worse than controls ( $M=12.50$ ; WAIS-IV/WISC-IV DS For  $M=8.00$ ; CPT-II Omiss  $M=6$ ) on measures of attention (WAIS-IV/WISC-IV DS,  $p < .01$ ;  $F=.525$ ; WAIS-IV/WISC-IV DS For,  $p < .01$ ;  $F=1.08$ ; CPT-II Omiss,  $p < .01$ ;  $F=2.44$ ). It is important to note that 4 of the 18 mTBI were not administered the CPT-II; 1 of these subjects was not tested at LLU, therefore did not have access to the computer containing the CPT-II; 1 of the mTBI

subjects had a history of having a seizure and therefore was not given the CPT-II. The remaining 2 subjects were not given the CPT-II due to technical difficulties at the time of testing. Additionally, 6 of the control subjects were not given the CPT-II due to the fact that they were tested off the LLU campus and therefore did not have access to the computer. Additionally, the mTBI group (RCFT 3 min  $M=34.94$ ; RCFT 30 min  $M=36.41$ ) performed significantly worse than controls (RCFT 3 min  $M=48.83$ ; RCFT 30 min  $M=48.08$ ) on measures of immediate and delayed non-verbal memory (RCFT 3 min,  $p < .01$ ;  $F=3.63$ ; RCFT 30 min,  $p < .01$ ;  $F=.92$ ). Finally, the mTBI group (WMS-III LMI  $M=7.94$ ) performed significantly worse than healthy controls (WMS-III LMI  $M=11.45$ ) on a measure of immediate verbal memory for contextually related information (WMS-III LMI,  $p < .05$ ;  $F=3.15$ ).

Table 2

*Neuropsychological Performances between groups*

	N	Mean	F(df)
WAIS-IV/ WISC-IV DS Total			.525(1,27)**
mTBI	17	8.71	
Control	12	12.50	
WAIS-IV/ WISC-IV DS Forward			1.08(1,27)**
mTBI	17	6.35	
Control	12	8.00	
WAIS-IV/ WISC-IV DS Backward			1.58(1,27)
mTBI	17	5.18	
Control	12	6.00	
CPT-II Omissions			2.44(1,18)*
mTBI	14	47.41	
Control	6	36.79	
CPT-II Commissions			.034(1,18)
mTBI	14	54.19	
Control	6	47.86	
CPT-II Hit Rate			.180(1,18)
mTBI	14	46.56	
Control	6	43.12	
RCFT 3 minute delay			3.63(1,27)**
mTBI	17	34.94	
Control	12	48.83	
RCFT 30 minute delay			.92(1,27)**
mTBI	17	36.41	
Control	12	48.08	
WMS-III LM 1			3.15(1,25)*
mTBI	16	7.94	
Control	11	11.45	
WMS-III LM II			1.61(1,25)
mTBI	16	8.56	
Control	11	11.64	

\* significant at &lt;.05

\*\* significant at &lt;.01

## Coping Meditational Analysis

### *MTBI Related to Differences in Coping*

No significant differences were found in coping styles between the mTBI and the control group (Table 3). Therefore, coping style cannot mediate the relationship between mTBI and neuropsychological outcomes.

Table 3

*MTBI as a predictor of coping*

	N	F(df)	R <sup>2</sup>	β (TBI)
Group(IV)*Confrontive(DV)		.342(1,26)	.013	.114
mTBI	17			
Control	12			
Group(IV)*Distancing(DV)		.006(1,26)	.000	.015
mTBI	17			
Control	12			
Group(IV)*Self-Controlling(DV)		.004(1,26)	.000	-.013
mTBI	17			
Control	12			
Group(IV)*Seeking Social Support(DV)		.107(1,26)	.004	-.064
mTBI	17			
Control	12			
Group(IV)*Accepting Responsibility(DV)		.000(1,26)	.000	-.004
mTBI	17			
Control	12			
Group(IV)*Escape Avoidance(DV)		2.222(1,26)	.079	-.281
mTBI	17			
Control	12			
Group(IV)*Planful Problem Solving(DV)		2.849(1,26)	.064	.314
mTBI	17			
Control	12			
Group(IV)*Positive Reappraisal(DV)		.824(1,26)	.031	-.175
mTBI	17			
Control	12			

\* significant at <.05

\*\* significant at <.01

### *Coping Style as a Predictor of Cognitive and Mood Outcomes*

The hypothesis that the use of problem-focused coping processes (confrontive coping, planful problem-solving) would be associated with better performance on neuro-cognitive tests following mTBI was confirmed (Table 4). Specifically, subjects utilizing higher levels of confrontive coping demonstrated better performance on attention measures than subjects with lower levels of confrontive coping (CPT-II omissions:  $F(1,16)= 7.155, p<.05, \beta =-.556$ ; CPT-II Hit RT:  $F(1,16)= 5.132, p<.05, \beta =.493$ ). Subjects endorsing increased planful problem-solving demonstrated better immediate and delayed non-verbal memory for complex information than subjects who were less likely to utilize a planful problem-solving coping style (RCFT 3 min:  $F(1,26)= 8.288, p<.01, \beta =.492$ ; RCFT 30 min:  $F(1,26)= 7.458, p<.05, \beta =.472$ ).

The hypothesis that distancing would be associated with poorer overall performance on neuropsychological measures was not confirmed (see Table 4). Specifically, individuals who endorsed more distancing, as a coping style, did not demonstrate significant differences on neuropsychological measures.

The hypothesis that wishful thinking, as measured by the escape avoidance coping style, would be associated with poorer overall performance on neuropsychological measures was confirmed (Table 4). Specifically, the escape avoidance coping style was significantly related to poorer performance on measures of simple attention (WAIS-IV DS:  $F(1,25)= 4.262, p<.05, \beta =-.382$ ; WAIS-IV DS For:  $F(1,26)= 9.234, p<.01, \beta =-.512$ ), delayed non-verbal memory for complex information (RCFT 30 min:  $F(1,26)= 5.031, p<.05, \beta =-.403$ ), and was significantly related to increased depression (BDI-II:  $F(1,19)= 6.015, p<.05, \beta =.490$ ).

The hypothesis that seeking social support and positive reappraisal would be associated with better neuropsychological performance was confirmed (Table 4). Specifically seeking social support was positively correlated with performance on measures of simple attention (WAIS-IV DS For:  $F(1,26)= 15.527, p<.01, \beta =.570$ ; WAIS-IV DS Back:  $F(1,26)= 7.968, p<.01, \beta =.484$ ). Additionally, the positive reappraisal coping style was positively associated with recognition memory for verbal information (WMS-III LM Rec:  $F(1,19)= 5.188, p<.05, \beta =-.463$ ). It is important to note that there is missing WAYS data for one mTBI subject due to the fact that the subject was tested as a clinical patient at the LLU Behavioral Health Institute and the clinician failed to administer the test.



Table 4

*Coping styles that are significantly predictive of neuropsychological and mood outcome*

	N	F(df)	R <sup>2</sup>	β (TBI)
WAYS Confrontive (IV)*CPT-II omiss(DV)		7.155(1,16)	.309	-.556*
mTBI	14			
mControl		6		
WAYS Confrontive (IV)*CPT Hit RT(DV)		5.132(1,16)	.243	.493*
mTBI	14			
mControl		6		
WAYS Seeking social support (IV)*DS For(DV)		15.527(1,26)	.325	.570**
mTBI	17			
Control	12			
WAYS Seeking social support (IV)*DS back(DV)		7.968(1,26)	.235	.484**
mTBI	17			
Control	12			
WAYS Accepting Responsibility (IV)*CPT-II Hit RT(DV)		6.635(1,16)	.293	-.541*
mTBI	14			
Control	6			
WAYS Escape Avoidance (IV)*DS(DV)		4.262(1,25)	.146	-.382*
mTBI	17			
Control	12			
WAYS Escape Avoidance (IV)*DS for(DV)		9.234(1,26)	.262	-.512**
mTBI	17			
Control	12			
WAYS Escape Avoidance (IV)*RCFT 30(DV)		5.031(1,26)	.162	-.403*
mTBI	17			
Control	12			
WAYS Escape Avoidance (IV)*BDI-II(DV)		6.015(1,19)	.240	.490*
mTBI	16			
Control	11			
WAYS Planful problem solving (IV)*RCFT 3 min(DV)		8.288(1,26)	.242	.492**
mTBI	17			
Control	12			
WAYS Planful problem solving (IV)*RCFT 30 min(DV)		7.458(1,26)	.223	.472*
mTBI	17			
Control	12			
WAYS Positive reappraisal(IV)*LM Rec(DV)		5.188(1,19)	.214	-.463*
mTBI	16			
Control	11			

\* significant at <.05

\*\* significant at <.01

### *Coping as a Mediator of Cognitive and Mood Outcomes*

The hypothesis that coping styles would mediate the relationship between mild TBI and neuropsychological mood outcomes was not confirmed. According to Baron and Kenny (1986), in order to assume mediation, the initial variable must correlate with the mediator. The initial variable, or the TBI group, did not correlate with the mediator, which was coping style (Table 3). Therefore, it is assumed that coping style does not mediate the relationship between mTBI and neuropsychological and mood outcomes.

### **Quality of Life and Mood Meditational Analyses**

#### *mTBI Related to Differences in Quality of Life and Mood*

The mTBI group performed significantly worse than healthy controls on a number of mood and quality of life measures (Table 5). Specifically, the mTBI group (BAI  $M=13.00$ ; BDI-II  $M=14.00$ ), when compared to controls (BAI  $M=3.18$ ; BDI-II  $M=4.73$ ), endorsed more symptoms of anxiety and depression (BAI,  $p<.01$ ;  $F=15.76$ ; BDI-II,  $p<.01$ ;  $F=3.00$ ). The mTBI group's average BAI and BDI-II scores were in the mildly anxious and depressed ranges, respectively, whereas the normal control group's average BAI and BDI-II scores were in the minimally anxious and depressed ranges.

Additionally, the mTBI group (WHO Overall  $M=56.25$ ; WHO Psych  $M=61.67$ ; WHO Ind.  $M=51.77$ ; WHO Phys.  $M=54.85$ ; WHO Social  $M=61.40$ ; WHO Environ  $M=64.75$ ) when compared to controls (WHO Overall  $M=85.42$ ; WHO Psych  $M=75.55$ ; WHO Ind.  $M=92.45$ ; WHO Phys.  $M=79.72$ ; WHO Social  $M=80.21$ ; WHO Environ  $M=85.68$ ) endorsed significantly worse overall, psychological, independence, physical, social, and environmental quality of life (WHO Overall,  $p<.01$ ;  $F=1.34$ ; WHO Psych,  $p<.05$ ;  $F=.83$ ;

WHO Ind.,  $p < .01$ ;  $F=6.00$ ; WHO Phys.  $p < .01$ ;  $F=.01$ ; WHO Social  $p < .01$ ;  $F=.27$ ; WHO Environ,  $p < .01$ ;  $F=2.75$ ). One mTBI subject is missing data for the WHO-QOL as a result of failure of the student clinician to administer the questionnaire.

MTBI was found to be a statistically significant predictor of poorer neuropsychological outcomes (Table 6). Specifically, when compared to controls, mTBI predicted poorer performances on attentional measures (WAIS-IV DS:  $F(1,27)=9.82$ ,  $R^2=.267$ ,  $p < .01$ ,  $\beta = .516$ ; WAIS-IV DS For:  $F(1,27)=6.45$ ,  $R^2=.193$ ,  $p < .01$ ,  $\beta = .439$ ; CPT-II omiss:  $F(1,18)=5.81$ ,  $R^2=.244$ ,  $p < .01$ ,  $\beta = -.494$ ). Additionally, mTBI was significantly predictive of poorer performances on measures of immediate and delayed non-verbal memory (RCFT 3 min:  $F(1,27)=11.99$ ,  $R^2=.308$ ,  $p < .01$ ,  $\beta = .55$ ; RCFT 30 min:  $F(1,27)=6.87$ ,  $R^2=.209$ ,  $p < .01$ ,  $\beta = .45$ ). MTBI was also found to be predictive of poorer performance on a measure of immediate memory for contextually related information (WMS-III LM I:  $F(1,25)=4.85$ ,  $R^2=.162$ ,  $p < .05$ ,  $\beta = .40$ ).

MTBI was found to be a statistically significant predictor of mood and perceived QOL (Table 7). Specifically, MTBI was predictive of increased anxiety and depression (BAI:  $F(1,22)=10.21$ ,  $R^2=.317$ ,  $p < .01$ ,  $\beta = -.563$ ; BDI-II:  $F(1,21)=11.17$ ,  $R^2=.347$ ,  $p < .01$ ,  $\beta = -.589$ ). Within the mTBI group, 5 subjects were not given the BAI and 6 subjects were not given the BDI-II due to the fact that the participant was either a child or was tested by a student clinician who failed to administer the test. BAI and BDI-II data only included data from adult subjects due to the fact that BYI (BAI and BDI) data was not directly comparable. Within the control group, one child subject was given the BYI and not the BDI-II or BAI. Additionally, TBI was predictive of poorer overall, psychological, independence, physical, social, and environmental QOL (WHO Overall:

F(1,27)= 12.66,  $R^2=.319$ ,  $p<.01$ ,  $\beta =.565$ ; WHO Psych: F(1,27)= 4.26,  $R^2=.136$ ,  $p<.05$ ,  $\beta =.37$ ; WHO Ind: F(1,27)= 31.16,  $R^2=.536$ ,  $p<.01$ ,  $\beta =.73$ ; WHO Phys: F(1,27)= 13.61,  $R^2=.335$ ,  $p<.01$ ,  $\beta =.58$ ; WHO Social: F(1,27)= 10.96,  $R^2=.289$ ,  $p<.01$ ,  $\beta =.54$ ; WHO Environ: F(1,27)= 14.09,  $R^2=.343$ ,  $p<.01$ ,  $\beta =.59$ ).

Table 5

*Mood and Quality of Life between groups*

	N	Mean	F(df)
BAI			15.76(1-22)**
mTBI	13	13.00	
Control	11	3.18	
BDI-II			3.00(1-21)**
mTBI	12	14.00	
Control	11	4.73	
WHO Overall			1.34(1-27)**
mTBI	17	56.25	
Control	12	85.42	
WHO Psychological			.83(1-27)*
mTBI	17	61.67	
Control	12	75.55	
WHO Independence			6.00(1-27)**
mTBI	17	51.77	
Control	12	92.45	
WHO Physical			.01(1-27)**
mTBI	17	54.85	
Control	12	79.72	
WHO Social			.27(1-27)**
mTBI	17	61.40	
Control	12	80.21	
WHO Environmental			2.75(1-27)**
mTBI	17	64.75	
Control	12	85.68	
WHO Spirituality			.15(1-27)
mTBI	17	69.49	
Control	12	80.21	

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 6

*TBI as a predictor of neuropsychological outcome*

	N	F(df)	R <sup>2</sup>	β (TBI)
Group(IV)*DS(DV)		9.82(1-27)	.267	.516**
mTBI	17			
Control	12			
Group(IV)*DS For(DV)		6.45(1-27)	.193	.439**
mTBI	17			
Control	12			
Group(IV)*CPT-II Omissions(DV)		5.81(1-18)	.244	-.494*
mTBI	14			
Control	6			
Group(IV)*RCFT 3 min(DV)		11.99(1-27)	.308	.55**
mTBI	17			
Control	12			
Group(IV)*RCFT 30 min(DV)		6.87(1-27)	.209	.45**
mTBI	17			
Control	12			
Group(IV)*LMI(DV)		4.85(1-25)	.162	.40*
mTBI	16			
Control	11			

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 7

*TBI as a predictor of mood and quality of life*

	N	F(df)	R <sup>2</sup>	β (TBI)
Group(IV)*BAI(DV)		10.21(1-22)	.317	-.563**
mTBI	13			
Control	11			
Group(IV)*BDI-II(DV)		11.17(1-21)	.347	-.589**
mTBI	12			
Control	11			
Group(IV)*WHO Overall(DV)		12.66(1-27)	.319	.565**
mTBI	17			
Control	12			
Group(IV)*WHO Psychological(DV)		4.26(1-27)	.136	.37*
mTBI	17			
Control	12			
Group(IV)*WHO Independence(DV)		31.16(1-27)	.536	.73**
mTBI	17			
Control	12			
Group(IV)*WHO Physical(DV)		13.61(1-27)	.335	.58**
mTBI	17			
Control	12			
Group(IV)*WHO Social(DV)		10.96(1-27)	.289	.54**
mTBI	17			
Control	12			
Group(IV)*WHO Environmental(DV)		14.09(1-27)	.343	.59**
mTBI	17			
Control	12			

\* significant at &lt;.05

\*\* significant at &lt;.01

### *Quality of Life as a Predictor of Cognitive and Mood Outcomes*

Quality of life was found to be a significant predictor of neuropsychological and psychological outcomes following mTBI (Tables 8, 9, 10, 11, 12, 13). Overall QOL was a significant predictor of simple attention (WAIS-IV DS:  $F(1,27)= 7.03$ ,  $R^2=.207$ ,  $p<.05$ ,  $\beta =.46$ ), anxiety (BAI:  $F(1,21)= 18.55$ ,  $R^2=.469$ ,  $p<.01$ ,  $\beta =-.69$ ), and depression (BDI-II:  $F(1,20)= 37.83$ ,  $R^2=.654$ ,  $p<.01$ ,  $\beta =-.81$ ). Physical QOL was found to be a significant predictor of attention (WAIS-IV DS:  $F(1,26)= 11.06$ ,  $R^2=.298$ ,  $p<.01$ ,  $\beta =.546$ ; WAIS-IV DS For.:  $F(1,27)= 13.04$ ,  $R^2=.326$ ,  $p<.01$ ,  $\beta =.57$ ; CPT-II Omis:  $F(1,17)= 4.69$ ,  $R^2=.216$ ,  $p<.05$ ,  $\beta =-.465$ ), anxiety (BAI:  $F(1,21)= 14.88$ ,  $R^2=.415$ ,  $p<.01$ ,  $\beta =-.64$ ), and depression (BDI-II:  $F(1,20)= 39.65$ ,  $R^2=.665$ ,  $p<.01$ ,  $\beta =-.815$ ). Psychological QOL was found to significantly predict attention (WAIS-IV DS:  $F(1,26)= 4.531$ ,  $R^2=.148$ ,  $p<.05$ ,  $\beta =.385$ ; WAIS-IV DS For:  $F(1,27)= 5.423$ ,  $R^2=.167$ ,  $p<.05$ ,  $\beta =.409$ ), immediate memory for contextually related information (LM I:  $F(1,24)= 4.82$ ,  $R^2=.167$ ,  $p<.05$ ,  $\beta =.409$ ), anxiety (BAI:  $F(1,21)= 10.32$ ,  $R^2=.330$ ,  $p<.01$ ,  $\beta =-.574$ ), and depression (BDI-II:  $F(1,20)= 24.708$ ,  $R^2=.553$ ,  $p<.01$ ,  $\beta =-.743$ ). Level of independence QOL was found to significantly predict attention (WAIS-IV DS:  $F(1,26)= 6.31$ ,  $R^2=.195$ ,  $p<.05$ ,  $\beta =.442$ ; WAIS-IV DS For:  $F(1,27)= 10.596$ ,  $R^2=.282$ ,  $p<.01$ ,  $\beta =.531$ ), anxiety (BAI:  $F(1,21)= 18.67$ ,  $R^2=.471$ ,  $p<.01$ ,  $\beta =-.686$ ), and depression (BDI-II:  $F(1,20)= 53.097$ ,  $R^2=.726$ ,  $p<.01$ ,  $\beta =-.852$ ). Social QOL was found to significantly predict attention (WAIS-IV DS:  $F(1,26)= 8.826$ ,  $R^2=.253$ ,  $p<.01$ ,  $\beta =.503$ ; WAIS-IV DS For:  $F(1,27)= 13.949$ ,  $R^2=.316$ ,  $p<.01$ ,  $\beta =.584$ ), anxiety (BAI:  $F(1,21)= 10.162$ ,  $R^2=.326$ ,  $p<.01$ ,  $\beta =-.571$ ), and depression (BDI-II:  $F(1,20)= 12.083$ ,  $R^2=.377$ ,  $p<.01$ ,  $\beta =-.614$ ). Finally, environmental



QOL was found to significantly predict attention (WAIS-IV DS:  $F(1,26)= 7.024$ ,  $R^2=.213$ ,  $p<.05$ ,  $\beta =.461$ ; WAIS-IV DS For:  $F(1,27)= 11.830$ ,  $R^2=.305$ ,  $p<.01$ ,  $\beta =.552$ ), anxiety (BAI:  $F(1,21)= 19.779$ ,  $R^2=.85$ ,  $p<.01$ ,  $\beta =-.696$ ), and depression (BDI-II:  $F(1,20)= 32.250$ ,  $R^2=.617$ ,  $p<.01$ ,  $\beta =-.786$ ).

Table 8

*Overall QOL as a predictor of neuropsychological and psychological outcome*

	N	F(df)	R <sup>2</sup>	β (TBI)
Overall QOL(IV)*DS(DV)		7.03(1-26)	.099	.314
mTBI	17			
Control	12			
Overall QOL(IV)*DS For(DV)		7.03(1-27)	.207	.46*
mTBI	17			
Control	12			
Overall QOL(IV)*CPT-II Omiss(DV)		.787(1-17)	.044	-.21
mTBI	14			
Control	6			
Overall QOL(IV)*RCFT 3 min(DV)		1.86(1-27)	.007	.083
mTBI	17			
Control	12			
Overall QOL(IV)*RCFT 30 min(DV)		.042(1-27)	.002	.039
mTBI	17			
Control	12			
Overall QOL(IV)*LM I(DV)		1.87(1-24)	.072	.27
mTBI	16			
Control	11			
Overall QOL(IV)*BAI(DV)		18.55(1-21)	.469	-.69**
mTBI	13			
Control	11			
Overall QOL(IV)*BDI-II(DV)		37.83(1-20)	.654	-.81**
mTBI	12			
Control	11			

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 9

*Physical QOL as a predictor of neuropsychological and psychological outcome*

	N	F(df)	R <sup>2</sup>	β (TBI)
Physical QOL(IV)*DS(DV)		11.06(1-26)	.298	.546**
mTBI	17			
Control	12			
Physical QOL(IV)*DS For(DV)		13.04(1-27)	.326	.57**
mTBI	17			
Control	12			
Physical QOL(IV)*CPT-II Omiss(DV)		4.69(1-17)	.216	-.465*
mTBI	14			
Control	6			
Physical QOL(IV)*RCFT 3 min(DV)		1.73(1-27)	.060	.245
mTBI	17			
Control	12			
Physical QOL(IV)*RCFT 30 min(DV)		1.13(1-27)	.040	.20
mTBI	17			
Control	12			
Physical QOL(IV)*LM I(DV)		3.17(1-24)	.117	.342
mTBI	16			
Control	11			
Physical QOL(IV)*BAI(DV)		14.88(1-21)	.415	-.64**
mTBI	13			
Control	11			
Physical QOL(IV)*BDI-II(DV)		39.65(1-20)	.665	-.815**
mTBI	12			
Control	11			

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 10

*Psychological QOL as a predictor of neuropsychological and psychological outcome*

	N	F(df)	R <sup>2</sup>	β (TBI)
Psychological QOL(IV)*DS(DV)		4.531(1-26)	.148	.385*
mTBI	17			
Control	12			
Psychological QOL(IV)*DS For(DV)		5.423(1-27)	.167	.409*
mTBI	17			
Control	12			
Psychological QOL(IV)*CPT-II Omiss(DV)		1.04(1-17)	.058	-.240
mTBI	14			
Control	6			
Psychological QOL(IV)*RCFT 3 min(DV)		.498(1-27)	.018	.135
mTBI	17			
Control	12			
Psychological QOL(IV)*RCFT 30 min(DV)		.464(1-27)	.017	.13
mTBI	17			
Control	12			
Psychological QOL(IV)*LM I(DV)		4.82(1-24)	.167	.409*
mTBI	16			
Control	11			
Psychological QOL(IV)*BAI(DV)		10.32(1-21)	.330	-.574**
mTBI	13			
Control	11			
Psychological QOL(IV)*BDI-II(DV)		24.708(1-20)	.553	-.743**
mTBI	12			
Control	11			

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 11

*Independence QOL as a predictor of neuropsychological and psychological outcome*

	N	F(df)	R <sup>2</sup>	β (TBI)
Independence QOL(IV)*DS(DV)		6.31(1-26)	.195	.442*
mTBI	17			
Control	12			
Independence QOL(IV)*DS For(DV)		10.596(1-27)	.282	.531**
mTBI	17			
Control	12			
Independence QOL(IV)*CPT-II Omiss(DV)		3.79(1-17)	.183	-.427
mTBI	14			
Control	6			
Independence QOL(IV)*RCFT 3 min(DV)		1.872(1-27)	.065	.255
mTBI	17			
Control	12			
Independence QOL(IV)*RCFT 30 min(DV)		.888(1-27)	.032	.178
mTBI	17			
Control	12			
Independence QOL(IV)*LM I(DV)		2.35(1-24)	.089	.299
mTBI	16			
Control	11			
Independence QOL(IV)*BAI(DV)		18.67(1-21)	.471	-.686**
mTBI	13			
Control	11			
Independence QOL(IV)*BDI-II(DV)		53.097(1-20)	.726	-.852**
mTBI	12			
Control	11			

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 12

*Social QOL as a predictor of neuropsychological and psychological outcome*

	N	F(df)	R <sup>2</sup>	β (TBI)
Social QOL(IV)*DS(DV)		8.826(1-26)	.253	.503**
mTBI	17			
Control	12			
Social QOL(IV)*DS For(DV)		13.949(1-27)	.316	.584**
mTBI	17			
Control	12			
Social QOL(IV)*CPT-II Omiss(DV)		.046(1-17)	.003	.052
mTBI	14			
Control	6			
Social QOL(IV)*RCFT 3 min(DV)		1.331(1-27)	.047	.217
mTBI	17			
Control	12			
Social QOL(IV)*RCFT 30 min(DV)		.652(1-27)	.024	.154
mTBI	17			
Control	12			
Social QOL(IV)*LM I(DV)		3.118(1-24)	.115	.339
mTBI	16			
Control	11			
Social QOL(IV)*BAI(DV)		10.162(1-21)	.326	-.571**
mTBI	13			
Control	11			
Social QOL(IV)*BDI-II(DV)		12.083(1-20)	.377	-.614**
mTBI	12			
Control	11			

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 13

*Environmental QOL as a predictor of neuropsychological and psychological outcome*

	N	F(df)	R <sup>2</sup>	β (TBI)
Environmental QOL(IV)*DS(DV)		7.024(1-26)	.213	.461*
mTBI	17			
Control	12			
Environmental QOL(IV)*DS For(DV)		11.830(1-27)	.305	.552**
mTBI	17			
Control	12			
Environmental QOL(IV)*CPT-II Omiss(DV)		1.242(1-17)	.068	-.261
mTBI	14			
Control	6			
Environmental QOL(IV)*RCFT 3 min(DV)		.525(1-27)	.019	.138
mTBI	17			
Control	12			
Environmental QOL(IV)*RCFT 30 min(DV)		.170(1-27)	.006	.079
mTBI	17			
Control	12			
Environmental QOL(IV)*LM I(DV)		1.889(1-24)	.073	.270
mTBI	16			
Control	11			
Environmental QOL(IV)*BAI(DV)		19.779(1-21)	.485	-.696**
mTBI	13			
Control	11			
Environmental QOL(IV)*BDI-II(DV)		32.250(1-20)	.617	-.786**
mTBI	12			
Control	11			

\* significant at &lt;.05

\*\* significant at &lt;.01

### *Quality of Life as a Mediator Between Cognitive and Mood Outcomes*

Quality of life was found to significantly mediate the relationship between mTBI and various neuropsychological and psychological outcomes. It was found that QOL did not mediate the relationship between mTBI and Digit Span or CPT-II Omissions performance, which are measures of attention (Tables 14 and 16). However, physical QOL (physical QOL:  $F(1,26)= 6.802$ ,  $R^2=.343$ ,  $p=n.s.$ ,  $\beta_{group} = .164$ ,  $p<.05$ ,  $\beta_{model} = .476$ ), social QOL (social QOL:  $F(1,26)= 7.402$ ,  $R^2=.363$ ,  $p=n.s.$ ,  $\beta_{group} = .176$ ,  $p<.05$ ,  $\beta_{model} = .489$ ), and Environmental QOL (environ QOL:  $F(1,26)= 6.262$ ,  $R^2=.325$ ,  $p=n.s.$ ,  $\beta_{group} = .176$ ,  $p<.05$ ,  $\beta_{model} = .449$ ) did mediate the relationship between mTBI and Digit Span Forward, which is another measure of simple attention (Table 15). QOL did not significantly mediate the relationship between mTBI and immediate memory for contextually related information (Table 16).

Overall QOL (overall QOL:  $F(1,20)= 10.304$ ,  $R^2=.507$ ,  $p=n.s.$ ,  $\beta_{group} = -.247$ ,  $p<.05$ ,  $\beta_{model} = -.535$ ), physical QOL (physical QOL:  $F(1,20)= 9.066$ ,  $R^2=.476$ ,  $p=n.s.$ ,  $\beta_{group} = -.301$ ,  $p<.05$ ,  $\beta_{model} = -.471$ ), independence QOL (independence QOL:  $F(1,20)= 9.149$ ,  $R^2=.478$ ,  $p=n.s.$ ,  $\beta_{group} = -.128$ ,  $p<.05$ ,  $\beta_{model} = -.590$ ), and environmental QOL (environ QOL:  $F(1,20)= 11.172$ ,  $R^2=.528$ ,  $p=n.s.$ ,  $\beta_{group} = -.253$ ,  $p<.01$ ,  $\beta_{model} = -.550$ ) significantly mediated the relationship between mTBI and anxiety (Table 15). Psychological QOL (psychological QOL:  $F(1,20)= 9.379$ ,  $R^2=.484$ ,  $p<.05$ ,  $\beta_{group} = -.420$ ,  $p<.05$ ,  $\beta_{model} = -.425$ ) and social QOL (social QOL:  $F(1,20)= 7.937$ ,  $R^2=.443$ ,  $p<.05$ ,  $\beta_{group} = -.388$ ,  $p<.05$ ,  $\beta_{model} = -.387$ ) were found to partially mediate the relationship between mTBI and anxiety (Table 17).



Overall QOL (overall QOL:  $F(1,19)= 21.003$ ,  $R^2=.689$ ,  $p=n.s.$ ,  $\beta_{group} =-.229$ ,  $p<.01$ ,  $\beta_{model} =-.674$ ), physical QOL (physical QOL:  $F(1,19)= 23.134$ ,  $R^2=.709$ ,  $p=n.s.$ ,  $\beta_{group} =-.252$ ,  $p<.01$ ,  $\beta_{model} =-.676$ ), independence QOL (independence QOL:  $F(1,19)= 25.244$ ,  $R^2=.727$ ,  $p=n.s.$ ,  $\beta_{group} =.020$ ,  $p<.01$ ,  $\beta_{model} =-.867$ ), and environmental QOL (environ QOL:  $F(1,19)= 18.89$ ,  $R^2=.665$ ,  $p=n.s.$ ,  $\beta_{group} =-.266$ ,  $p<.01$ ,  $\beta_{model} =-.635$ ) significantly mediated the relationship between mTBI and depression (Table 16). Psychological QOL (psychological QOL:  $F(1,19)= 24.290$ ,  $R^2=.719$ ,  $p<.01$ ,  $\beta_{group} =-.431$ ,  $p<.01$ ,  $\beta_{model} =-.605$ ) and social QOL (social QOL:  $F(1,19)= 10.467$ ,  $R^2=.524$ ,  $p<.05$ ,  $\beta_{group} =-.434$ ,  $p<.05$ ,  $\beta_{model} =-.413$ ) were found to partially mediate the relationship between mTBI and depression (Table 18).

Table 14

*Mediational models of digit span as an outcome measure following mTBI*

	N	F(df)	R <sup>2</sup>	β (group)	β (model)
Group(IV)+Physical QOL(IV)=DS(DV)					
		6.755(1-25)	.351	.278	.388
mTBI	17				
Control	12				
Group(IV)+Psychological QOL(IV)=DS(DV)					
		5.283(1-25)	.297	.414*	.235
mTBI	17				
Control	12				
Group(IV)+Independence QOL(IV)=DS(DV)					
		4.457(1-25)	.263	.377	.169
mTBI	17				
Control	12				
Group(IV)+Social QOL(IV)=DS(DV)					
		6.111(1-25)	.328	.323	.332
mTBI	17				
Control	12				
Group(IV)+Environmental QOL(IV)=DS(DV)					
		5.218(1-25)	.295	.350	.260
mTBI	17				
Control	12				

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 15

*Mediational models of digit span forward as an outcome measure following mTBI*

	N	F(df)	R <sup>2</sup>	β (group)	β (model)
Group(IV)+Physical QOL(IV)=DS For(DV)					
		6.802(1-26)	.343	.164	.476*
mTBI	17				
Control	12				
Group(IV)+Psychological QOL(IV)=DS For(DV)					
		4.649(1-26)	.263	.334	.286
mTBI	17				
Control	12				
Group(IV)+Independence QOL(IV)=DS For(DV)					
		5.242(1-26)	.287	.109	.451
mTBI	17				
Control	12				
Group(IV)+Social QOL(IV)=DS For(DV)					
		7.402(1-26)	.363	.176	.489*
mTBI	17				
Control	12				
Group(IV)+Environmental QOL(IV)=DS For(DV)					
		6.262(1-26)	.325	.176	.449*
mTBI	17				
Control	12				

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 16

*Mediational models using QOL as mediators of attention and immediate memory for contextually related information following mTBI*

	N	F(df)	R <sup>2</sup>	β (group)	β (model)
Group(IV)+Physical QOL(IV)=CPT-II Omiss(DV)					
		2.734(1-16)	.255	-.299	-.239
mTBI	14				
Control	6				
Group(IV)+Physical QOL(IV)=LMI(DV)					
		2.401(1-23)	.173	.286	.181
mTBI	16				
Control	11				

\* significant at <.05

\*\* significant at <.01

Table 17

*Mediational models using QOL measures as mediators of anxiety following TBI*

	N	F(df)	R <sup>2</sup>	β (group)	β (model)
Group(IV)+Physical QOL(IV)=BAI(DV)					
		9.066(1-20)	.476	-.301	-.471*
mTBI	13				
Control	11				
Group(IV)+Psychological QOL(IV)=BAI(DV)					
		9.379(1-20)	.484	-.420*	-.425*
mTBI	13				
Control	11				
Group(IV)+Independence QOL(IV)=BAI(DV)					
		9.149(1-20)	.478	-.128	-.590*
mTBI	13				
Control	11				
Group(IV)+Social QOL(IV)=BAI(DV)					
		7.937(1-20)	.443	-.388*	-.387*
mTBI	13				
Control	11				
Group(IV)+Environmental QOL(IV)=BAI(DV)					
		11.172(1-20)	.528	-.253	-.550**
mTBI	13				
Control	11				
Group(IV)+Overall QOL(IV)=BAI(DV)					
		10.304(1-20)	.507	-.247	-.535*
mTBI	13				
Control	11				

\* significant at &lt;.05

\*\* significant at &lt;.01

Table 18

*Mediational models using QOL measures as mediators of depression following mTBI*

	N	F(df)	R <sup>2</sup>	β (group)	β (model)
Group(IV)+Physical QOL(IV)=BDI-II(DV)					
		23.134(1-19)	.709	-.252	-.676**
mTBI	12				
Control	11				
Group(IV)+Psychological QOL(IV)=BDI-II(DV)					
		24.290(1-19)	.719	-.431**	-.605**
mTBI	12				
Control	11				
Group(IV)+Independence QOL(IV)=BDI-II(DV)					
		25.244(1-19)	.727	.020	-.867**
mTBI	12				
Control	11				
Group(IV)+Social QOL(IV)=BDI-II(DV)					
		10.467(1-19)	.524	-.434*	-.413*
mTBI	12				
Control	11				
Group(IV)+Environmental QOL(IV)=BDI-II(DV)					
		18.891(1-19)	.665	-.266	-.635**
mTBI	12				
Control	11				
Group(IV)+Overall QOL(IV)=BDI-II(DV)					
		21.003(1-19)	.689	-.229	-.674**
mTBI	13				
Control	11				

\* significant at &lt;.05

\*\* significant at &lt;.01

## CHAPTER FOUR

### DISCUSSION

Overall the results from this study demonstrated that the mTBI group performed significantly worse than healthy controls on a number of neuropsychological measures. Specifically, the mTBI group performed worse than controls on measures of attention, immediate and delayed non-verbal memory, and immediate verbal memory for contextually related information. MTBI was found to be a statistically significant predictor of poorer neuropsychological outcomes. Specifically, when compared to controls, mTBI predicted poorer performances on attentional measures, immediate and delayed non-verbal memory, and immediate memory for contextually related information. This confirms established research, which has shown that mTBI negatively affects cognition.

No significant differences were found in coping styles between the mTBI and the control group. This was not expected, however this finding may be explained by the fact that coping style may be more of an intrinsic character quality than a psychological outcome measure. Therefore in future research, coping style may be best viewed as an independent variable rather than a dependent, or outcome variable. However, coping style was significantly related to neuropsychological and psychological outcomes. Specifically, confrontive coping was related to better performance on attention measures. Subjects endorsing increased planful problem-solving demonstrated better immediate and delayed non-verbal memory for complex information. The escape avoidance coping style was significantly related to poorer performance on measures of simple attention, delayed non-verbal memory for complex information, and was significantly related to increased

depression. Seeking social support was associated with better performance on measures of simple attention. Additionally, the positive reappraisal coping style was positively related with recognition memory for verbal information. Coping styles did not mediate the relationship between mild TBI and neuropsychological mood outcomes. Due to the fact that the initial variable, or the TBI group, did not correlate with the mediator, which was coping style, coping style cannot be considered a mediator of mTBI and neuropsychological and mood outcomes.

Overall, the results from the coping style analyses confirm that different coping styles may predict differences in neuropsychological and psychological outcomes, as was predicted. However, the current findings also suggest that mild head injury does not produce changes in coping style. Although, it might be argued that with increased severity of injury and/ or frontal lobe damage, an individual might demonstrate diminished executive functioning, including compromised problem- solving, flexibility, and inhibition, which may impact coping style. The current study only evaluated mild TBI patients; therefore conclusions cannot be drawn regarding the possibility of using coping style as an outcome measure in a more severe TBI population. Research in this area would be useful and provide information as to whether or not head injury across severity contributes to changes in the way an individual copes with life's stressors.

Another possible explanation of why group differences were not observed within coping style may be that the majority of the current subjects were from the Loma Linda area, which is a predominantly Seventh Day Adventist community. It is possible that this group of people is more religious and/or spiritual than members of other communities. This increased spirituality may in turn predict better overall ability to cope with life's



stressors. An additional possible factor that may have led to insignificant findings within the coping style analyses between groups may be related to a limitation of the WAYS coping style measure. Specifically, this measure evaluates the most stressful situation that has occurred for the individual over the past week. Throughout testing, numerous individuals reported that they had not experienced anything significantly stressful over the past week. As a result, the subjects “most stressful situation” may have been an event that was minimally distressing. Conversely, other individuals responded to questions with a highly stressful situation in mind. Therefore, this measure may have inaccurately evaluated the true coping style of individuals who reported not having experienced a stressful situation over the past week. Specifically, it is unlikely that a minimally stressful situation would have evoked significant coping skills, therefore diminishing the potential for true coping style to be evaluated with a measure. Additionally, a possible explanation of why coping style did not significantly differ between groups may be related to the fact the some of the participants may have been involved in psychotherapy, thus likely focusing on developing effective coping skills within the therapeutic context. It is known that some of the participants were actively involved in therapy or had received therapy at some time following their head injury. Unfortunately, this information was not available for all subjects as it was not a formal variable being measured within the scope of the current study. However, it is an important fact to consider in future research. Overall, the current conclusion from the population studied is that while coping style varied within the two groups it did not vary between groups. Therefore, it is currently assumed that mTBI does not affect an individual’s coping style. However, more research is needed, with a larger sample size, in order to confirm the current findings.

Furthermore, results indicated that the mTBI participants performed significantly worse than healthy controls on a number of mood and quality of life measures. Specifically, mTBI was predictive of increased anxiety, depression, and poorer overall, psychological, independence, physical, social, and environmental QOL. These findings confirm previously established literature, which shows that mTBI contributes to increased depression and anxiety. However, the quality of life findings were surprising, as it was not expected that nearly all domains of quality of life assessment would indicate such significant decline in the mTBI group. All domains of QOL were found to be lower in the mTBI group than the control group, with the single exception being the Spirituality domain. This finding is interesting and may be conceptualized in a number of ways. For example, spirituality may be more truly understood as an inherent character quality that may not be as vulnerable to fluctuate following mTBI as other QOL domains. Another possible explanation for this finding may be the fact that the subject population was predominantly from the Loma Linda area, which is a largely Seventh Day Adventist community. There is a possibility that subjects from the current study were more spirituality homogenous in nature due to the fact that the sample was obtained within a highly spiritual community. However, more research is needed in this area to better understand whether religious affiliation impacts QOL and whether mTBI predicts changes in spiritual QOL.

Quality of life was found to be a significant predictor of neuropsychological and psychological outcomes following mTBI. Overall QOL was a significant predictor of simple attention, anxiety, and depression. Physical QOL was found to be a significant predictor of attention, anxiety, and depression. Psychological QOL was found to

significantly predict attention, immediate memory for contextually related information, anxiety, and depression. Level of independence QOL was found to significantly predict attention, anxiety, and depression. Social QOL was found to significantly predict attention, anxiety, and depression. Finally, environmental QOL was found to significantly predict attention, anxiety, and depression. It is feasible that the robust QOL differences seen between groups may even more significantly mediate the relationship between mTBI and neuropsychological outcomes with additional subjects.

Quality of life, which has been considered to be a dependent variable was found to significantly mediate the relationship between mTBI and various neuropsychological and psychological outcomes. It was found that physical QOL, social QOL, and Environmental QOL significantly mediated the relationship between mTBI and simple attention. Overall QOL, physical QOL, independence QOL, and environmental QOL significantly mediated the relationship between mTBI and anxiety. Psychological QOL and social QOL were found to partially mediate the relationship between mTBI and anxiety. Overall QOL, physical QOL, independence QOL, and environmental QOL significantly mediated the relationship between mTBI and depression. Psychological QOL and social QOL were found to partially mediate the relationship between mTBI and depression. These findings demonstrate an important finding and possibly provide an explanation to the basic question asking why some mTBI patients experience residual cognitive and psychological symptoms while others do not. Additionally, the current results fill a gap in the literature, which, to date, has not evaluated mediational affects of QOL on outcomes following mTBI.

In general, the study is underpowered, which requires more subjects to evaluate true effects. When using a conservative estimate (0.15) of the effect size, many of the measures (i.e., coping, quality of life) are more psychosocial in nature and therefore have less robust effects. However, in some cases the findings were rather robust and an effect was clearly seen. One should interpret non-significant findings with caution, as it is likely that type II errors have been made due to decreased power resulting from a low subject number.

It is important for future research to confirm the current findings in addition to exploring additional questions. Future studies should evaluate the differences in cognitive, mood, and quality of life outcomes between children, adolescents, and adults following mTBI. These findings may provide an important understanding of how mTBI affects cognition, mood, and quality of life across the lifespan. Another interesting question for future research is whether or not the type of injury has a significant impact on outcomes. Specific injuries that appear to be the most common include sports related injuries, motor vehicle accidents and falls. It may be useful to better understand whether or not these injuries differ from other head injuries incurred in different ways. This may provide insight into the clinical implications of various types of mTBI. Additionally, it is important for future research to consider the factors that may predict perceived QOL following mTBI. Specifically, research is needed to examine the possible relationship between neuroimaging findings and cognitive and psychological outcomes in patients with chronic post-concussive symptoms. Research questions may seek to determine if prolonged cerebral metabolic alterations occur in individuals with persistent neurocognitive deficits following a mild TBI and if regions of altered cerebral

metabolism are associated with changes in tissue perfusion. It is feasible that following mTBI, alterations to cerebral metabolism and perfusion will occur, which may correlate to post-concussive syndromes following mTBI. Patients with greater amounts of alterations of cerebral metabolism and tissue perfusion may demonstrate poorer performance on neuropsychological, mood, and quality of life measures. Additionally, alterations to cerebral metabolism and perfusion may mediate the relationship between head injury and neuropsychological, mood, and quality of life outcomes. Possible findings may provide an understanding of whether chronic metabolic changes mediate cognitive and psychological outcomes in mTBI patients with chronic post-concussive symptoms.

In summary, this preliminary study has shown that mTBI subjects, when compared to healthy controls, performed poorer on various neuropsychological measures, displayed increased levels of depression and anxiety and reported poorer quality of life. Additionally, quality of life was found to significantly mediate the relationship between mTBI and simple attention, anxiety, and depression. Using these findings as a guide, future studies should continue to assess additional participants in order to increase statistical power to the current findings. This study has been useful in filling a gap in the literature, which has failed to examine the potential mediating role of QOL on outcomes following mTBI. The current findings provide clarification about the nature and extent of cognitive and psychological outcomes following TBI. More importantly, this study has established a new understanding of the importance of perceived QOL following mTBI. Finally, these findings may suggest that treatment interventions focused on improving an individual's perceived quality of life may result in improved attention and amelioration of

depression and anxiety following mTBI. Specifically, given the great risk for mTBI patients to develop depression and anxiety it is important to identify the factors that may intercept this conversion. This study has demonstrated that perceived quality of life mediates the relationship between mTBI and depression and anxiety. This finding directly affects treatment and can be translated to specific therapeutic interventions. Specifically, the current findings provide evidence that working with an individual to improve his or her perceived quality of life will likely reduce depression and anxiety. Cognitive behavioral therapy (CBT), one of the most commonly used therapies, helps the patient identify negative beliefs and behaviors and replace them with healthy, positive ones. CBT encourages the individual to own his or her thoughts and change the way he or she thinks and behaves. This therapeutic modality, with a focus on changing one's thoughts and feelings with regard to quality of life, may prove to dramatically reduce mood symptoms. It is hopeful that the findings from the current findings will affect therapeutic interventions, improving the overall prognosis of the mTBI patient.

## REFERENCES

1. Anderson, V. V., & Catroppa, C. C. (2005). Recovery of executive skills following paediatric traumatic brain injury (TBI): A 2 year follow-up. *Brain Injury*, 19(6), 459-470. doi:10.1080/02699050400004823
2. Arciniegas DB, Anderson CA, Topkoff JL, et al. (2005) Mild traumatic brain injury: a neuropsychiatric approach to diagnosis, evaluation, and treatment. *Neuropsychiatr Dis Treat* 1:311-327.
3. Ashwal S, Holshouser BA, Shu S, et. al. (2000) Predictive value of proton magnetic resonance spectroscopy in pediatric closed head injury. *Pediatric Neurol* 23:114-125.
4. Babikian, T., & Asarnow, R. (2009). Neurocognitive outcomes and recovery after pediatric TBI: Meta-analytic review of the literature. *Neuropsychology*, 23(3), 283-296. doi:10.1037/a0015268
5. Babikian T, Freier MC, Ashwal S, Riggs ML, Burley T, Holshouser BA. (2006) MR spectroscopy: predicting long-term neuropsychological outcome following pediatric TBI. *J Magn Reson Imaging* 24:801-811.
6. Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51, 1173-1182.
7. Bate, A. J., Mathias, J. L., & Crawford, J. R. (2001). Performance on the Test of Everyday Attention and standard tests of attention following severe traumatic brain injury. *The Clinical Neuropsychologist*, 15(3), 405-422. doi:10.1076/clin.15.3.405.10279
8. Bergsneider M, Hovda DA, Lee SM, Kelly DF, McArthur DL, Vespa PM, Lee JH, Huang SC, Martin NA, Phelps ME, Becker DP (2000). Dissociation of cerebral glucose metabolism and level of consciousness during the period of metabolic depression following human traumatic brain injury. *J Neurotrauma*, 17:389-401.
9. Bergsneider M, Hovda DA, McArthur DL, Etchepare M, Huang SC, Sehati N, Satz P, Phelps ME, Becker DP (2001). Metabolic recovery following human traumatic brain injury based on FDG-PET: time course and relationship to neurological disability. *J Head Trauma Rehabil*, 16:135-148.
10. Binder LM, Rohling ML, & Larrabee GL (1997). A review of mild head trauma. Part I: meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology*, 19(3), 421-431.

11. Brewster, P. H., Lawson, K., & Ornstein, T. J. (2009). Gender Differences in Cognitive and Affective Function Following Mild TBI. 2. Retrieved from EBSCOhost.
12. Brooks WM, Friedman SD, Gasparovic C (2001) Magnetic resonance spectroscopy in traumatic brain injury. *J Head Trauma Rehabil*, 16:149-164.
13. Cecil KM, Hills EC, Sandel ME, et al. (1998) Proton magnetic resonance spectroscopy for detection of axonal injury in the splenium of the corpus callosum of brain-injured patients. *J Neurosurg*, 88:795-801.
14. Chan, R. K. (2005). Sustained attention in patients with mild traumatic brain injury. *Clinical Rehabilitation*, 19(2), 188-193.
15. Cicerone KD & Azulay J (2007). Perceived self-efficacy and life satisfaction after traumatic brain injury. *Journal of Head Trauma Rehabilitation*. 22(5), 257-66.
16. Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences*, Second ed. New Jersey: Lawrence Erlbaum Associates.
17. Conners, C.K. & MHS Staff. (Eds.) (2000) *Conners' Continuous Performance Test II: Computer Program for Windows Technical Guide and Software Manual*. North Tonawanda, NY: Mutli-Health Systems.
18. Cullen, N. K., Park, Y., & Bayley, M. T. (2008). Functional recovery following traumatic vs non-traumatic brain injury: A case-controlled study. *Brain Injury*, 22(13-14), 1013-1020.
19. Dawson, D. R., Schwartz, M. L., Winocur, G., & Stuss, D. T. (2007). Return to productivity following traumatic brain injury: Cognitive, psychological, physical, spiritual, and environmental correlates. *Disability and Rehabilitation: An International, Multidisciplinary Journal*, 29(4), 301-313.
20. Holshouser BA, Tong KA, Ashwal S, Oyoyo U, Ghamsary M, Saunders D, Shutter L. (2006) Prospective longitudinal proton magnetic resonance spectroscopic imaging in adult traumatic brain injury. *J Magn Reson Imaging*, 24, 33-40.
21. Fontana, A., & McLaughlin, M. (1998). Coping and appraisal of daily stressors predict heart rate and blood pressure levels in young women. *Behavioral Medicine*, 24(1), 5-16. doi:10.1080/08964289809596376
22. Fraser, R. R., Machamer, J. J., Temkin, N. N., Dikmen, S. S., & Doctor, J. J. (2006). Return to work in traumatic brain injury (TBI): A perspective on capacity for job complexity. *Journal of Vocational Rehabilitation*, 25(3), 141-148.
23. Galbiati, S., Recla, M., Pastore, V., Liscio, M., Bardoni, A., Castelli, E., & Strazzer,



- S. (2009). Attention remediation following traumatic brain injury in childhood and adolescence. *Neuropsychology*, 23(1), 40-49.
24. Gary, K., Arango-Lasprilla, J., & Stevens, L. (2009). Do racial/ethnic differences exist in post-injury outcomes after TBI? A comprehensive review of the literature. *Brain Injury*, 23(10), 775-789.
25. Goldstein, F. C., & Levin, H. S. (2001). Cognitive outcome after mild and moderate traumatic brain injury in older adults. *Journal of Clinical and Experimental Neuropsychology*, 23(6), 739-753.
26. Goodman C., Knoll G., Isakov V., & Silver H. (2005). Negative attitude towards medication is associated with working memory impairment in schizophrenia patients. *International Clinical Psychopharmacology*, 20(2), 93-6.
27. Guérin, F., Kennepohl, S., Léveillé, G., Dominique, A., & McKerral, M. (2006). Vocational outcome indicators in atypically recovering mild TBI: A post-intervention study. *NeuroRehabilitation*, 21(4), 295-303.
28. Hanks R.A., Millis S.R., Ricker J.H., Giacino J.T., Nakese-Richardson R., Frol A.B., Novack T.A., Kalmar K., Sherer M., & Gordon W.A. (2008). The predictive validity of a brief inpatient neuropsychologic battery for persons with traumatic brain injury. *Archives of Physical Medical Rehabilitation*. 89(5), 950-7.
29. Holcomb, Matthew J., Anya Mazur-mosiewicz, and Raymond S. Dean. 2009. Comorbidity of Secondary Diagnose in Children With TBI and ADHD. 3. *PsycEXTRA*, EBSCOhost.
30. Kesler, S.R., Adams, H.F., Blasey, C.M., and Bigler, E.D. (2003). Premorbid intellectual functioning, education, and brain size, in traumatic brain injury: an investigation of the cognitive reserve hypothesis. *Applied Neuropsychology*. 10(3): 153-62
31. King, K. A., Hough, M. S., Vos, P., Walker, M. M., & Givens, G. (2006). Word retrieval following mild TBI: Implications for categorical deficits. *Aphasiology*, 20(2-4), 233-245. doi:10.1080/02687030500473155
32. Kumar, R., Gupta, R. K., Husain, M., Chaudhry, C., Srivastava, A., Saksena, S., & Rathore, R. S. (2009). Comparative evaluation of corpus callosum DTI metrics in acute mild and moderate traumatic brain injury: Its correlation with neuropsychometric tests. *Brain Injury*, 23(7-8), 675-685.
33. Kwok, F. Y., Lee, T. C., Leung, C. S., & Poon, W. S. (2008). Changes of cognitive functioning following mild traumatic brain injury over a 3-month period. *Brain Injury*, 22(10), 740-751.

34. Lachapelle, J., Bolduc-Teasdale, J., Ptito, A., & McKerral, M. (2008). Deficits in complex visual information processing after mild TBI: Electrophysiological markers and vocational outcome prognosis. *Brain Injury*, 22(3), 265-274.
35. Leitgeb, J., Mauritz, W., Brazinova, A., Janciak, I., Majdan, M., Wilbacher, I. (2011). Effects of gender on outcomes after traumatic brain injury. *J Trauma: Injury, Infection and Critical Care*, XX(XXX).
36. Levine, B., Dawson, D., Boutet, I., Schwartz, M. L., & Stuss, D. T. (2000). Assessment of strategic self-regulation in traumatic brain injury: Its relationship to injury severity and psychosocial outcome. *Neuropsychology*, 14(4), 491-500.
37. Lucas, J. A., & Addeo, R. (2006). Traumatic Brain Injury and Postconcussion Syndrome. In P. J. Snyder, P. D. Nussbaum, D. L. Robins, P. J. Snyder, P. D. Nussbaum, D. L. Robins (Eds.) , *Clinical neuropsychology: A pocket handbook for assessment, 2nd ed* (pp. 351-380). Washington, DC US: American Psychological Association.
38. McGhee, H., Cornwell, P., Addis, P., & Jarman, C. (2006). Treating dysarthria following traumatic brain injury: Investigating the benefits of commencing treatment during post-traumatic amnesia in two participants. *Brain Injury*, 20(12), 1307-1319.
39. Meyerson, D. M., Sparadeo, F. R., & Meyerson, M. M. (2009). Utility of a Novel Processing Speed Measure in Assessing mTBI. 2. Retrieved from EBSCOhost.
40. Millis, S.R., Rosenthal, M, Novack, T.A., Sherer, M, Nick, T.G., Kreutzer, J.S., High, WM., Ricker, Joseph H. (2001). Long-Term Neuropsychological Outcome After Traumatic Brain Injury. *Journal of Head Trauma Rehabilitation*, 16(4), 343-355.
41. O'Connell, M.J. (2000). Prediction of Return to Work Following Traumatic Brain Injury: Intellectual, Memory, and Demographic Variables. *Rehabilitation Psychology*, 45(2), 212-217.
42. Proctor A., Zhang J. (2008.) Performance of three racial/ethnic groups on two tests of executive function: Clinical implications for traumatic brain injury (TBI). *NeuroRehabilitation*, 23, 529-536.
43. Shames, J., Treger I., Ring, H., & Giaquinto, S. (2007). Return to work following traumatic brain injury: Trends and challenges. *Disability and Rehabilitation*, 29(17), 1387-1395.
44. Stapert, S., Houx, P., De Kruijk, J., Ponds, R., & Jolles, J. (2006). Neurocognitive fitness in the sub-acute stage after mild TBI: The effect of age. *Brain Injury*, 20(2), 161-165.

45. Tsaousides, T., Ashman, T., & Seter, C. (2008). The psychological effects of employment after traumatic brain injury: Objective and subjective indicators. *Rehabilitation Psychology, 53*(4), 456-463.
46. Tsaousides, T., Warshowsky, A., Ashman, T. A., Cantor, J. B., Spielman, L., & Gordon, W. A. (2009). The relationship between employment-related self-efficacy and quality of life following traumatic brain injury. *Rehabilitation Psychology, 54*(3), 299-305.
47. Tsushima, W. T., Lum, M., & Geling, O. (2009). Sex differences in the long-term neuropsychological outcome of mild traumatic brain injury. *Brain Injury, 23*(10), 809-814.
48. Williamson, D. G., Scott, J. G., & Adams, R. L. (1996). Traumatic brain injury. In R. L. Adams, O. A. Parsons, J. L. Culbertson, S. Nixon, R. L. Adams, O. A. Parsons, S. Nixon (Eds.), *Neuropsychology for clinical practice: Etiology, assessment, and treatment of common neurological disorders* (pp. 9-64). Washington, DC US: American Psychological Association.
49. Wood, R. L., & Rutterford, N. A. (2006, December). Predictors of Long-Term Outcome Following Traumatic Brain Injury. *Clinician's Research Digest*. p. 2.
50. Xiong, Y., Mahmood, A., Chopp, M. (2010). Neurorestorative Treatments for Traumatic Brain Injury. *Discovery Medicine, 10*(54):434-42.
51. Yen, C., Chen, C., Cheng, C., Yen, C., Lin, H., Ko, C., & ... Chen, C. (2008). Comparisons of insight in schizophrenia, bipolar I disorder, and depressive disorders with and without comorbid alcohol use disorder. *Psychiatry and Clinical Neurosciences, 62*(6), 685-690.
52. Ziino, C., & Ponsford, J. (2006). Selective attention deficits and subjective fatigue following traumatic brain injury. *Neuropsychology, 20*(3), 383-390.

### **Related Articles**

53. Garnett MR, Blamir AM, Rajagopalan B, et. al (2002) Evidence for cellular damage in normal-appearing white matter correlates with injury severity in patients following traumatic brain injury: a magnetic resonance spectroscopy study. *Brain 123*:1403-1409.
54. Gladis, M. M., Gosch, E. A., Dishuk, N. M., & Crits-Christoph, P. (1999). Quality of life: Expanding the scope of clinical significance. *Journal of Consulting and Clinical Psychology, 67*(3), 320-331.
55. Hovda, D.A., (1996) Metabolic Dysfunction. *Neurotrauma*. Narayan RK, Wilberger JE,

and Povilshock (Eds.) McGraw Hill, Inc., New York.

56. Kervick, R. B., & Kaemingk, K. L. (2005). Cognitive appraisal accuracy moderates the relationship between injury severity and psychosocial outcomes in traumatic brain injury. *Brain Injury*, 19(11), 881-889.
57. Langfitt T.W., Obrist W.D., Alavi A., Grossman R.I., Zimmerman R., Jaggi J., Uzzell B., Reivich M., & Patton D.R. (1986). Computerized tomography, magnetic resonance imaging, and positron emission tomography in the study of brain trauma. Preliminary observations. *J Neurosurg*, 64:760-767.
58. Ruff R. (2005). Two decades of advances in understanding of mild traumatic brain injury. *Journal of Head Trauma Rehab*, 20, 5-18.
59. Shutter L., Tong K.A., Lee A., Holshouser B.A. (2006). Prognostic value of proton magnetic resonance spectroscopy in acute traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 21(4), 334-349.
60. Thurman DJ, Alverson C, Browne D, et al., (1999) Traumatic brain injury in the United States: a report to Congress. Center for Disease Control and Prevention.
61. Walz NC, Cecil KM, Wade SL, Michaud LJ (2008) Late proton magnetic resonance spectroscopy following traumatic brain injury during early childhood: relationship with neurobehavioral outcome. *J Neurotrauma* 25:94-103.
62. Wortzel H.S., Filley C.M., Anderson C.A., Oster T., Arciniegas D.B. (2008). Forensic applications of cerebral single photon emission computed tomography in mild traumatic brain injury. *J Am Acad Psychiatry Law*, 36, 310-322.
63. Yamaki T., Yoshino E., Fujimoto M., Ohmori Y., Imahori Y., Ueda S. (1996). Chronological positron emission tomographic study of severe diffuse traumatic brain injury in the chronic stage. *J Trauma*, 40:50-56.
64. Garnett M.R., Blamire A.M., Corkill R.G., Rajagopalan B., Young J.D., Cadoux-Hudson T.A., Styles P. (2001). Abnormal cerebral blood volume in regions of contused and normal appearing brain following traumatic brain injury using perfusion magnetic resonance imaging. *J Neurotrauma*, 18, 585-593.
65. Lewine J.D., Davis J.T., Bigler E.D., Thoma R., Hill D., Funke M., Sloan J.H., Hall S., Orrison W.W. (2007). Objective documentation of traumatic brain injury subsequent to mild head trauma: multimodal brain imaging with MEG, SPECT, and MRI. *J Head Trauma Rehabil* 22:141-155.
66. Bonne O., Gilboa A., Louzoun Y., Kempf-Sherf O., Katz M., Fishman Y., Ben-

Nahum Z., Krausz Y., Bocher M., Lester H., Chisin R., Lerer B. (2003) Cerebral blood flow in chronic symptomatic mild traumatic brain injury. *Psychiatry Res*, 124, 141-152.