Summer 7-2020

Subjective Cognitive Complaints, Affective Distress, and Objective Cognitive Performance in Mild Traumatic Brain Injury

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by

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DISSERTATION

Submitted in Partial Fulfillment of the
Requirements for the Degree of

Doctor of Philosophy
Psychology

The University of New Mexico
Albuquerque, New Mexico

July, 2020

ACKNOWLEDGMENTS

I would like to express my gratitude to my mentor, Dr. Ron Yeo, whose expertise, guidance, and patience has been invaluable. His willingness to give his time so generously and his encouragement made it possible to achieve this goal. My sincere thanks must also go to the other members of my thesis advisory and examination committee, Dr. Steven Verney, Dr. Richard Campbell, and Dr. James Cavanagh for the assistance they provided at all levels of the research project. I would like to thank my lab mates, Jessica Pommy and Rebecca Avila-Rieger for always being there for me, from late night phone calls to listening to me practice my presentations. I would like to thank Dr. Dan Matthews and Dr. Lorna Joachim for supporting me throughout my entire graduate career. I would like to acknowledge my supervisors at the New Mexico VA Medical Center, Dr. Shelley Leiphart, Dr. Jessica Madrigal-Bauguss, and Dr. Brian Kersh for fostering and supporting my interest in brain injury research and clinical practice. Finally, I must express my very profound gratitude to my parents, Susan and Ray Seaman for always believing in me even when I didn't believe in myself. Thank you for your unfailing support and continuous encouragement. Thank you to Kirk, Julie, Peter, Annie, Janet, and Michael for loving and supporting me.

SUBJECTIVE COGNITIVE COMPLAINTS, AFFECTIVE DISTERSS, AND OBJECTIVE COGNITIVE PERFORMANCE IN MILD TRAUMATIC BRAIN INJURY

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ABSTRACT

Background: Traumatic Brain Injury (TBI) is a leading cause of morbidity and mortality among adults in the US (Ma, Chan, & Carruthers, 2014; Raj et al., 2015). According to epidemiological estimates put forth by the CDC, of the 1.7 million TBIs that occur annually in the United States, 80% are mild TBI (mTBI) (Ma, Chan, & Carruthers, 2014). At the sub-acute stage, mTBI patients often report experiencing post-concussion symptoms that include somatic (nausea, headache, dizziness), cognitive (poor attention, memory, and executive function), and behavioral or emotional changes (irritability, depression, emotional lability, anxiety) following their injury (Levin & Diza-Arrastia, 2015).

Study Aims: The specific aims of the current study were to: 1) investigate the relationship between mood, subjective complaints of cognitive symptoms, and executive functioning (EF) performance in mTBI and control participants at the sub-acute time point; 2) evaluate the role of mood in understanding group differences in EF and subjective cognitive symptom complaints; 3) examine changes in mood and subjective cognitive symptom complaints in the mTBI group over time and determine if demographic factors, specifically ethnicity, impact mood, EF, and their relationship.

Participants and Methods: Participants were 52 individuals recruited from the Departments of Neurosurgery and Emergency Medicine from UNMHSC within two weeks following a mTBI. Control participants included 32 sex- and age- matched individuals from the Albuquerque, New Mexico community. Participants attended two assessment sessions; the first session was 3-14 days post injury and the second session was ~2 months post injury. Participants completed self-report measures of post-concussion symptoms (the Neurobehavioral Symptom Inventory, the Frontal Systems Behavior Scale, the Patient-Reported Measurement Information System, the Rivermead Post-Concussion Symptoms Questionnaire) and a depression measure (BDI-II), as well as an objective neuropsychological functioning measure (the Executive Abilities: Measure and Instruments for Neurobehavioral Evaluation and Research assessment battery (NIH-EXAMINER) and a measure of premorbid intelligence (the Test of Premorbid Intelligence).

Results: mTBI patients reported experiencing significantly worse mood and more subjective cognitive symptom complaints compared to healthy controls at two weeks post injury. While mTBI participants and healthy controls differed in estimates of premorbid intelligence, they did not differ in objectively measured EF. Across all self-report measures individuals with a mTBI did not demonstrate improvements in mood or symptoms between the first and second session. The current estimate of a small, non-significant effect size (d = .14) for group differences on the NIH-EXAMINER is consistent with reports from previous studies. Effect sizes for mood (d = .90) and subjective symptom reporting (d = .94) were much larger, and represent important targets for clinical intervention.

Conclusions: EF deficits were not present in the sub-acute time frame, but group differences in depressive mood and the number of subjective complaints were prominent. Depression appears to be a critical treatment target for improving quality of life in mTBI patients, in contrast to EF functioning.

Keywords: Mild Traumatic Brain Injury, mTBI, Brain Injury, Executive Function, Subjective Cognitive Complaints, NIH-EXAMINER

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INTRODUCTION

Traumatic Brain Injury (TBI) is a leading cause of morbidity and mortality in the United States affecting about 1.74 million people each year (Ma, Chan, & Carruthers, 2014; Raj et al., 2015). The public health burden of TBI is far-reaching, with TBI resulting in approximately 1.365 million emergency department visits, 275,000 hospitalizations, and 52,000 deaths yearly (Ma, Chan, & Carruthers, 2014). TBI is a significant public health burden and long-term disability figures are staggering, with a prevalence rate of 3.32 million individuals on long-term disability (Zaloshnja, Miller, Langlois, & Selassie, 2008). Among traditionally underserved populations, the prevalence and impact of a TBI is noteworthy. Blacks have the highest rate of TBI, with a reported rate of 78.7 per 100,000 individuals, followed by Whites, then American Indians and Alaskan Natives, Asians, or Pacific Islanders (Faul & Coronado, 2015). Furthermore, the Hispanic population has a higher than average incidence of TBI when compared to non-Hispanic White (NHW) individuals with reported rates of 262 per 100,000 individuals for Hispanics (Arango-Lasprilla et al., 2007). The estimated annual number of sustained TBIs is 998,176 for males and 693,329 for females, meaning that males sustain TBIs 1.4 times more often than their female counterparts (Faul, Xu, Wald, & Coronado, 2010).

The direct and indirect costs of TBIs affect multiple levels of the healthcare system including the government, the taxpayer, and the survivor. Direct medical costs alone have been estimated at $48.3 billion to $76.5 billion (Ma, Chan, & Carruthers, 2014). Indirect costs are appraised at $51.2 billion, due to missed work and lost productivity (Rutland-Brown, Langlois, & Thomas, 2006). In addition to the significant

monetary cost, there are many other severe consequences facing the TBI survivor. These often include difficulties with activities of daily living, financial independence, and social reintegration (Ma, Chan, & Carruthers, 2014).

Diagnostic Criteria. TBI is defined by the Center for Disease Control (CDC) as "an injury that disrupts the normal function of the brain. It can be caused by a bump, blow, or jolt to the head or a penetrating head injury" (CDC, 2014, p.2). Various approaches have been used to categorize TBIs based on severity, type of injury, and the presence or absence of specific symptoms (Friedland, 2013). Injury severity is based on the following criteria, loss of consciousness (LOC), post traumatic amnesia (PTA), Glasgow Coma Scale rating (GCS), and presence or absence of neuroimaging findings. LOC is defined as the presence or absence of LOC and the duration of time the individual is unconscious. LOC can range from seconds to weeks and varying lengths of LOC result in different determinations of TBI severity. PTA occurs during the period following a brain injury, usually after LOC, wherein the individual is conscious but is experiencing amnesia, meaning he/she is unable to maintain continuous memory. Of note, LOC does not have to occur for PTA to be present. To further assess injury severity, medical providers often administer the GCS in the acute setting following the injury (Friedland, 2013). The GCS assess coma and impaired level of consciousness and is considered the gold standard in assessment after brain injury (Teasdale & Jennett, 1974). Verbal performance, motor responsiveness and eye opening are independently measured to make an overall GCS score. Finally, in the event of a more severe injury, medical providers may preform neuroimaging studies to determine if brain abnormalities are present. The four components LOC, PTA, GCS, and neuroimaging results taken together provide

clinicians with an approximate measure of TBI severity categorized as: mild, moderate and severe. Relying on one indicator of injury severity can be problematic because medical interventions may complicate assessment. For example, facial injuries or intubation can limit the accuracy of GCS rating scores.

A mild TBI (mTBI) is defined by the following criteria: LOC that lasts for 30 minutes or less, PTA lasting for less than 24 hours, a GCS score between 13-15 and negative neuroimaging findings. A moderate TBI is defined by the following criteria: LOC that lasts for 1-24 hours, PTA lasting for 1-7 days, a GCS score between 9-12, and in some cases, abnormal brain imaging results. Severe TBI is defined by the following criteria: LOC that lasts for more than 24 hours, PTA lasting for more than 7 days, a GCS score between 3-8, and abnormal neuroimaging findings (Friedland, 2013).

Mild Traumatic Brain Injury

mTBI represents a major public health issue. Rates of hospitalized adults with mTBIs range from 100 to 300/100,000 each year (Carroll et al., 2004). According to epidemiological estimates put forth by the CDC, of the 1.7 million TBIs that occur annually in the United States, the majority, 80%, are mild (Ma, Chan, & Carruthers, 2014). This incidence rate most likely understates the true occurrence impact of mTBI in the United States as many mTBIs are treated in non-hospital medical settings, emergency departments, or go untreated. While an individual who sustains a mTBI may only experience brief symptoms, a significant minority of individuals experience persistent problems. Unfortunately, the term “mild” may be a misnomer and the patient or provider may fail to properly grasp its true impact. The consequences of mTBI on one’s life are not, in fact, always “mild”.

Typical Course of mTBI. In the typical course of a mTBI case, symptom and cognitive recovery is usually achieved three months post injury (Belanger et al., 2005). While many individuals follow the usual course of recovery, a nontrivial minority report experiencing persistent post-concussion symptoms following a mTBI. When symptoms persistent they are referred to as post-concussive syndrome (PCS) (Boake et al., 2005). Estimates of PCS occurrence range widely and the etiology of this syndrome continues to be debated in the literature (Ryan & Warden, 2009).

mTBI Symptoms

Individuals who sustain a mTBI, whether they recover as expected or are later diagnosed with PCS, commonly report experiencing a general cluster of post-concussion symptoms immediately following the mTBI. Post-concussion symptoms include somatic (nausea, headache, dizziness), cognitive (poor attention, memory, and executive function), and behavioral or emotional changes (irritability, depression, emotional lability, anxiety) following the injury (Levin & Diza-Arrastia, 2015). While clinicians expect these symptoms to resolve in the 12 weeks following the injury, and often do (Levin & Diaz-Arrastia, 2015), the area of most interest to medical and mental health providers is the population of individuals whose symptoms persist. Studies estimate 44% of individuals may continue to experience post-concussion symptoms one year after injury (Dikmen, Machamer, Fann, & Temkin, 2010) and 30% of patients with mTBI experience new onset or intensification of one or more post-concussion symptoms three months post injury (Meares et al., 2011). In looking at the types of symptoms mTBI survivors report, Ponsford and colleagues (2011) examined mTBI and control patients in the emergency department. Their results revealed that while both mTBI and control

patients recovered well physically at three months post injury, patients with mTBI reported more cognitive complaints (memory and concentration difficulties) than their control counterparts (Ponsford, Cameron, Fitzgerald, Grant, & Mikocka-Walus, 2011). At three months post injury, rates of post-concussion symptom prevalence have been reported at 64% for mild to moderate TBIs (Boake et al., 2005). Dikmen and colleagues (2010) found that as many as 44% report experiencing three or more symptoms one-year post injury (Dikmen, Machamer, Fann, & Temkin, 2010). While these estimates of post-concussion symptoms range considerably they nonetheless highlight the significant distress this population of mTBI survivors experiences and the negative impact these symptoms have on the lives of mTBI injury survivors.

Following discharge from the hospital, a significant proportion of mTBI survivors are not returning to stable employment. For those who are employed preinjury, 31% of individuals with a mTBI were found to be unemployed at one-year post-injury (Doctor et al., 2005). These findings contrast sharply with national expected unemployment rates ranging from 8% to 10% for the general population (Doctor et al., 2005). Prevalence rates of unemployment for mTBI survivors were found in one study to increase from 12% prior to the injury to 27% following the TBI, while for control participants, the unemployment rate increased from 5% to 16% (Edna & Cappelen, 1987). The impact of a TBI among individuals from traditionally underserved communities is particularly concerning. For example, Arango-Lasprilla et al., (2008) found that compared to non-Hispanic Whites, individuals from ethnic/racial minority groups (Black, Hispanic, Asian, Native American) were approximately twice as likely to be unemployed one-year post injury. After adjusting for gender, scores on the disability rating scale at discharge,

preinjury employment status, marital status, age, education, and cause of injury these results remained (Arango-Lasprilla et al., 2008). Given the high prevalence rate and persistence of symptoms following mTBI resulting in severe consequences such as unemployment, an examination of the symptoms following mTBI is warranted. In particular, this should be examined among traditionally underserved populations.

As previously discussed, typical acute and/or chronic symptoms following a mTBI include deficits and dysfunction in the domains of mood, cognition, and behavior. Mood symptoms include self-reported emotional or behavioral problems such as increased irritability, anxiety, depression, affective lability, apathy, and/or impulsivity (Bay & Donders, 2008). Cognitive impairments across multiple domains have been reported including deficits in attention, memory, and executive function (Chamelian & Feinstein, 2006). Finally, physical problems include self-reported pain, sleep disturbance, and sensory problems such as headaches, dizziness, and visual disturbances (Wickwire et al., 2016). The development of these symptoms can be the result of psychosocial factors, neurobiological consequences of the injury, or a combination of both (Ponsford, Cameron Fitzgerald, Grant, & Mikocka-Walus, 2011; Wood, 2004). Researchers note the deficits produced by an mTBI are often more subtle and not as well understood as those of more severe TBIs (Dikmen, Machamer, Miller, Doctor, & Temkin, 2001).

Mood Symptoms. Depression is not only one of the most common emotional changes reported by TBI survivors, it is also one of the most frequent psychiatric disorder diagnoses patients receive after sustaining a TBI (Bay & Donders, 2008; Jorge et al., 2004). Depression is characterized by feelings of sadness, hopelessness, worthlessness, and emptiness. Depression can manifest in the form of loss of interest in previously

enjoyed activities, fatigue, concentration problems, sleep disturbances, and changes in appetite. Thoughts about death or suicidal ideation may also be present (American Psychiatric Association, 2013). Estimates of depression following a TBI range from 10% to 77% and are often most prominent in the first year following a brain injury (Silver, McAllister, & Arciniegas, 2009). TBI point prevalence rates of depression have been reported to be 30% at one month post injury (Bombardier et al., 2010). In the year following the injury, prevalence rates between 13% and 53% have also been reported (Bombardier et al., 2010; Deb, Lyons, Koutzoukis, Ali, & McCarthy, 1999; Rogers & Reid, 2007). In contrast, rates of depression in the general population range from 8% to 10% (Guillamondegui et al., 2011).

Major depression in the acute period following a mTBI is associated with patients report of more post-concussion symptoms, psychosocial dysfunction, and psychological distress (Rapoport, McCullagh, Streiner, & Feinstein, 2003). Patients with depression self-report increases in the perceived severity and number of post-concussion symptoms (Silver, McAlister, & Arciniegas, 2009). Furthermore, the patient's perception of their daily functioning has been shown to exacerbate depressive symptoms in this population (Pagulyan, Hoffman, Temkin, Machamer, & Dikmen, 2008). Jorge and colleagues (2004) found that 33% of individuals with a mTBI were diagnosed with major depressive disorder (MDD) following their injury. Notably, of those who received an MDD diagnosis, more than half were diagnosed in the acute period (Jorge et al., 2004). Risk factors for depression such as prior history of depression and alcohol dependence may increase the likelihood of recurrent episodes of depression in TBI patients (Whelan-Goodinson, Ponsford, Schonberger & Johnston, 2010).

Depression is associated with poorer recovery following TBI, yet awareness of depression risk post-TBI among providers and patients is low. In examining symptom reporting of depression following mTBI, Lange and colleagues (2011) found that both depressed individuals with mTBI and depressed individuals without a history of mTBI endorsed greater post-concussive symptoms, and more severe symptoms than individuals with a mTBI without depression (Lange, Iverson, & Rose, 2011). Furthermore, the authors found that 95.7% of individuals in the mTBI-depressed group met ICD-10 criteria for PCS compared to only 48.6% in the mTBI-non-depressed group. The relationship between mood problems and reported perceived cognitive impairments is of particular interest as well. For instance, Jorge and colleagues (2004) found that patients with a TBI diagnosed with MDD following their injury exhibited significant impairment on tests of executive functioning (Jorge et al., 2004). Additionally, individuals in the depressed TBI group had smaller gray matter volume in the prefrontal cortex relative to the non-depressed TBI group (Jorge et al., 2004) suggesting there may be a relationship between depression, TBI, executive functioning, and pre-frontal cortex volume. In another study examining mild to moderate TBI, self-reported symptoms of depression, rather than cognitive complaints, were associated with poorer performance on measures of executive function (Schiehser et al., 2011). Though notably this study did not utilize controls and relied on participant self-report of pre and post-injury behaviors instead of using a longitudinal design. Therefore, they trusted their participants' ability to estimate their preinjury behavior which may not be an accurate reflection of their actual preinjury behavior (Schiehser et al., 2011).

Depression symptomatology is often measured using the Beck Depression Inventory II (BDI-II) (Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report measure of the intensity of depressive symptoms for individuals ages 13 to 80. Individuals are asked to respond to statements about their mood over the past two weeks on a 4-point scale, with the overall score obtained by summing the ratings for all items. Scores ranging between 0 and 13 are indicative of minimal depression; scores that fall between 14 and 19 are considered to reflect a mild level of depression; scores of 20 to 28 are considered moderate; and a score ranging from 29 to 63 is labeled severe (Beck Steer, & Brown, 1996).

Cognitive Symptoms. While changes in mood are common following mTBI, survivors often report experiencing a myriad of other symptoms that impact their quality of life and daily functioning including symptoms related to changes in cognition (Chamelian & Feinstein, 2006). Individuals with a mTBI may show deficits in a variety of cognitive domains including executive function, memory, attention, and processing speed (Arciniegas, Held, & Wagner, 2002; Frencham, Fox, & Maybery, 2005). Executive functioning (EF) comprises higher-order cognitive abilities such as cognitive flexibility, problem solving, concept formation, self-regulation, working memory, and attention. For mild, moderate, and severe TBI survivors, deficits in this domain are common (Frencham, Fox, & Maybery, 2005), and often result in difficulties engaging in inpatient rehabilitation and achieving social independence upon discharge (Wood & Worthington, 2017). EF deficits can be objectively quantified using neuropsychological test performance completed following the brain injury.

Results from neuropsychological assessments conducted on mTBI survivors reveal overall mild cognitive impairment following a mTBI with small effect sizes (Belanger et al., 2005). Jurick et al. (2018) demonstrated that poorer processing speed and attention as well as more severe psychological symptoms are associated with worse performance on EF aspects of inhibition and set-switching in veterans with a history of mTBI greater than three months. Further, this remained after accounting for injury variables (Jurick et al., 2018). Another recent study found that compared to healthy control veterans, veterans who sustained mTBIs performed significantly worse on EF measures even after controlling for combat exposure, depression, and age (Gaines, Soper, & Berenji, 2016).

Examining the effect of these EF impairments, a meta-analysis by Schretlen and Shapiro (2003) examined effect sizes of studies reporting on patients with mTBIs and patients with moderate to severe TBIs. Based on their review of 15 studies, the overall EF effect size (Cohen's d) for mTBI studies was d = .24 and the effect size for moderate to severe TBIs was d = .74. Cohen (1988) defined d's of 0.2, 0.4, and 0.8 as small, medium, and large effect sizes, respectively. A second meta-analysis reported similar overall effect sizes (d = .20) for EF deficits assessed in the first three months following a mTBI (Belanger et al., 2005). Overall these meta-analyses point to small effects sizes for EF deficits.

EF Assessment Measures

A variety of different measures have been used to assess EF impairments in mTBI including: Delis-Kaplan Executive Function System (D-KEFS) verbal fluency and color-word interference (Jurick et al., 2018), Trail Making Test (Gaines, Soper, & Berenji,

2016), Rey-Osterrieth Complex Figure Test (Gaines, Soper, & Berenji, 2016), Controlled Oral Word Association Test (COWA-FAS) (Prince & Bruhns, 2017), Wisconsin Card Sorting Test (WCST) (Prince & Bruhns, 2017). The Executive Abilities: Measure and Instruments for Neurobehavioral Evaluation and Research (NIH-EXAMINER) (Kramer et al., 2014) is a more recent neuropsychological assessment that aims to assess EF deficits in a variety of neurobehavioral conditions that is efficient, modifiable, and modular. The test is suitable for a broad range of ages and ability levels, available in Spanish and English, and notably includes individuals with TBI in its diagnostic cohort. The test is separated into domains of working memory, inhibition, set shifting, fluency, planning, insight, and social cognition and behavior. Regarding the psychometric properties of the NIH-EXAMINER, confirmatory factor analysis supports a one-factor and a three-factor model which are the basis for the Executive Composite score as well as subscale scores of Fluency, Working Memory, and Cognitive Control. Test-retest reliabilities for these scales range from .78 to .93 (Kramer et al., 2014).

Given that the NIH-EXAMINER is a more recent neuropsychological assessment measure there is a dearth of literature on its use in TBI and mTBI populations. Possin and colleagues (2014) examined the validity and neuroanatomical correlates of the Executive Composite score for the NIH-EXAMINER. The Executive Composite score includes measures of set-shifting, inhibition, fluency, and working memory. The study sample consisted of 225 mixed neurological patients and healthy controls, of which 16 has sustained a TBI. The study did not specify if the TBIs were mild, moderate, or severe. For their analyses, the Executive Composite score was considered to have good ecological validity if it was found to be a significant predictor of the Frontal Systems

Behavior Scale (FrSBe) in regression models. The results indicated the Executive Composite score on the NIH-EXAMINER accounted for 28% of the variance in FrSBe scores beyond age. Furthermore, the Executive Composite remained a significant predictor after including two commonly used EF tests, the Stroop test and Trails B as covariates (Possin, LaMarre, Wood, Mungus, & Kramer, 2014). In another study, Gardner and colleagues (2017) examined subjective and objective cognitive functioning in older adults with a history of TBI and concluded that their results, which showed a lack of cognitive impairment in mTBI compared with controls, was due to poor sensitivity of the cognitive battery chosen for the study. The study did not utilize measures that designed to assess EF and processing speed and therefore they recommended using the NIH-EXAMINER for future studies (Gardner, Langa, & Yaffe, 2017).

A review of the literature identifies only one study that utilized the NIH-EXAMINER specifically in a TBI population. Kaup and colleagues (2017) used the NIH-Examiner to assess the neuropsychological profile of TBI in older veterans. All study participants had sustained TBIs at least one year prior, with most having sustained their injury five years prior. Specifically, they analyzed factor scores for working memory, fluency and cognitive control in their analyses. Overall, they found the neuropsychological profile of older veterans with a lifetime history of TBI to be characterized by executive dysfunction (f^2 = .08) and slowed processing speed (f^2 = .09). Further, deficits associated with TBIs were most often present for individuals who had sustained multiple mTBIs or individuals who had sustained moderate to severe TBIs.

These EF and working memory deficits were not clearly present for veterans who sustained only a single mTBI (Kaup et al., 2017).

Subjective Cognitive Complaints

The ability of patients to accurately report cognitive deficits following a brain injury is of considerable clinical importance. Medical and mental health providers are often uncertain of the best way to interpret and utilize patient self-report, as this information, while extremely valuable, is often fraught with inconsistences. Subjective cognitive complaints following TBI can include self-reported impairments in memory, attention, and EF (Ponsford, Cameron, Fitzgerald, Grant, & Mikocka-Walus, 2011). These complaints are extremely distressing as they may lead to TBI survivors experiencing disability and prolong use of medical services. It is important to distinguish subjective cognitive complaints from objectively measured cognitive deficits. Subjective cognitive complaints following TBI are often only weakly related to injury severity and cognitive complaints do not necessarily imply impairment of cognitive abilities (Chamelian & Feinstein, 2006; Stulemeijer, Vos, Bleijenberg, & van der Werf, 2007). Physical limitations, fatigue, and affective distress have been shown to be strongly associated with cognitive symptom reporting (Trahan, Ross, & Trahan, 1999; Karzmark, Hall, & Englander, 1995).

A large proportion of the variance in subjective cognitive complaint reporting appears to be accounted for by factors that are not related to the injury. For example, depressed mood, as previously reported, is strongly related to the presence of subjective cognitive complaints in individuals with a mTBI (Trahan, Ross, & Trahan, 1999). Stulemijuer and colleagues (2007) examined individuals who sustained a mTBI with and

without cognitive complaints six months' post injury. Thirty-nine percent of individuals with a mTBI in their sample reported cognitive complaints. Within this group, cognitive complaints were strongly related to emotional distress, personality, lower education level, and poorer physical functioning but not to injury characteristics. Further, severity of self-reported cognitive complaints was unrelated to performance on neuropsychological assessment (Stulemijuer et al., 2007). These findings raise the strong possibility that subjective cognitive complaints may be more related to sub-acute emotional functioning than to neuropsychological test performance. This result has implications for treatment of mTBI using mood therapies as these findings imply that treatment of mood complaints may lead to a reduction in subjective cognitive complaints in this population.

At present there a few measures designed for TBI populations that are used in the reporting of subjective cognitive complaints. These measures are the Neurobehavioral Symptom Inventory (Cicerone & Kalmar, 1995), the Frontal Systems Behavior Scale (Grace & Malloy, 2001), and the Rivermead Post-Concussion Symptoms Questionnaire (King, Crawford, Wenden, Moss, & Wade, 1995).

Neurobehavioral Symptom Inventory. A common measure for assessing post concussive symptoms following TBI is the Neurobehavioral Symptom Inventory (NSI). The NSI is a 22-item symptom checklist and each item is rated on a 5-point Likert scale from 0 (none) to 4 (very severe) wherein the patient rates how much the symptoms have impacted the patient in the past two weeks (Cicerone & Kalmar, 1995). Cronbach's alpha for the comprehensive scale was 0.95, which indicates a high degree of internal consistency. High alphas for each of the three subscales, Somatic, Affective, and Cognitive, are observed as well (0.88, 0.91, 0.92 respectively). There is a high degree of

correspondence among most NSI items and the NSI total score, particularly anxiety, forgetfulness, difficulty concentrating, and poor frustration tolerance (r = 0.80 to 0.83) (King et al., 2012).

Frontal Systems Behavior Scale. The FrSBe is a behavior rating-scale comprised of 46 items, designed to measure dysfunctions regarding behaviors associated with the frontal regions of the brain (Grace & Malloy, 2001). Each item is rated on a 5-point Likert scale from 1 (almost never) to 5 (almost always) wherein the participants rates how they believe they were functioning before the injury and at present (after the injury). The FrSBe yields four scores for each time point (i.e. before the injury and after the injury): a total score, and three subscores (Apathy, Disinhibition, and Executive Dysfunction, respectively consisting of 14, 15, and 17 items). Higher scores are indicative of poorer self-reported functioning (Grace & Malloy, 2001). The total score and the three subscales have shown high internal consistency, satisfactory test-retest reliability in multiple studies Malloy & Grace, 2005). This suggests that the scales have strong construct, discriminant, convergent, and ecological validity (Malloy & Grace, 2005). Specifically, high internal consistency has been shown in the FrSBe manual which reports α coefficients of 0.88, 0.72, 0.75, and 0.79 for the Total, Apathy, Disinhibition, and Executive Dysfunction scales for the self-report form in a normative sample respectively (Grace & Malloy, 2001). Factor-analyses of the FrSBe for clinical use in a population of patients with traumatic brain injury indicated that the total-score is an appropriate measure when considering its psychometric properties (Niemeier, Perrin, Holcomb, Nersessova, & Rolston, 2013). Reid-Arndt and colleagues (2007) found the FrSBe to be a significant predictor of community integration outcomes following

traumatic brain injury. Specifically, their results showed more executive complaints (FrSBe Executive Dysfunction) predicted lower community integration Total Scores. Further, they found that increased apathy (FrSBe Apathy) was associated with reduced community integration productivity (Reid-Arndt, Nehl and Hinkebein, 2007). Normative data for the FrSBe comes from the FrSBe Professional Manual and the normative sample is comprised of 436 men and women ranging in age from 18 to 95 years and in education from 10 years to doctoral level. The normative tables stratified for gender, age, and education provide *T* scores (Malloy & Grace, 2005).

Rivermead Post-Concussion Symptoms Questionnaire. Another measure that is often used to quantify TBI severity is the Post-Head Injury Symptoms Questionnaire, an adaptation of the Rivermead Post-Concussion Symptoms Questionnaire (RPQ) (King, Crawford, Wenden, Moss, & Wade., 1995). In this measure, TBI post-concussive symptom severity is defined via self-report wherein individuals are asked to rate 16 post-concussive symptoms on a scale of 0 (not experienced at all) to 4 (severe problem) (Guise et al., 2015). The 16 symptoms are divided into three subscales: cognitive (poor concentration, poor memory, taking longer to think), emotional (depression, frustration, irritability, restlessness), and somatic (dizziness, nausea, headache, noise sensitivity, fatigue, sleep disturbance, blurred vision, double vision, light sensitivity). Similarly, factor analysis of the RPQ finds a three-factor model of cognitive, emotional, and somatic factors, with a high degree of covariation overall. The two-factor model combined somatic and emotional factors into one factor and resulted in a similar goodness-of-fit to the data (Potter, Leigh, Wade, & Fleminger, 2006).

The test is divided into the RPQ-13 and the RPQ-3, with the RPQ-13 items assessing mood, cognition, sleep, and other physical symptoms that are later symptoms of post-concussion syndrome. The RPQ-3 items assess dizziness, headaches, nausea and/or vomiting and are early concussion symptoms. It is recommended that analyses are conducted separately for the RPQ-13 and the RPQ-3 because individually they demonstrate good construct validity and test-retest reliability, which is lost when they are combined (Eyers, Carey, Gilworth, Neumann, &Tennant, 2005). In the present study, per recommendations from the literature, the RPQ-13 will be used to assess mTBI severity.

Sleep. Sleep disturbance is among the most common complaint individuals report following a TBI (Wickwire et al., 2016). Alterations in sleep can develop acutely following injury (Ponsford, Parcell, & Sinclair, 2013) or present at a later point during the recovery process (Mantua, Mahan, Henry, & Spencer, 2015). Individuals with milder injuries often report greater impairments in subjective and objective measures of sleep compared to those with moderate or severe TBIs (Parcell, Ponsford, Rajaratnam, & Redmond, 2006; Shekleton et al., 2010). Objective recordings of sleep obtained via polysomnography (PSG) demonstrate longer sleep latency (Mantua, Henry, Garskovas, & Spencer, 2017), hypersomnia (Imbach et al., 2016), decreased sleep efficiency (a measure of time asleep compared to time in bed) (Shekleton et al., 2010), and increased awakenings (Parcell, Ponsford, Redman, & Rajaratnam, 2008).

Present Study

Consequently, a question is raised, how does a clinician conceptualize and treat patients who present with subjective cognitive complaints despite no evidence of cognitive impairment on objective neuropsychological testing? As discussed previously,

mood, or more specifically depressed mood, appears to be one factor that could potentially moderate this relationship. Individuals with depression are generally expected to have cognitive complaints, as perceived cognitive impairment is a hallmark of depression (Channon & Green, 1999; Zakzains, Leach & Kaplan, 1998). Similarly, depressed mood is very common following a mTBI. Individuals with depressed mood commonly view themselves as being more emotionally, cognitively, and physically disabled compared with their non-depressed counterparts. Clinically, this is relevant as it has implications for treatment including when intervention is necessary following injury. Furthermore, the ability to predict if survivors of mTBI will experience PCS or improve would be a vital tool for clinicians.

Understanding the nature of subjective complaints in the mTBI population may be useful for accurate diagnosis and enable providers to design treatment interventions for individuals who experience persistent post-concussion symptoms. It is imperative we examine the relationship between subjective cognitive complaints and objective EF performance while taking into account depressive symptoms to better understand how best to help this population.

The purpose of this study was to clarify the relationship among self-reported behavioral and cognitive symptom complaints with objectively measured EF in mTBI in a sub-acute and post-acute stages of recovery. We employed symptom (NSI, FrSBe) and depression measures (BDI-II) in addition to a measure of mTBI symptom severity (RPQ) and objective neuropsychological functioning measures (NIH-EXAMINER assessment battery).

Specific Aims and Hypotheses

Specific Aim 1: To investigate the relationship between mood, subjective cognitive symptom complaints, and EF performance in mTBI and control participants at the sub-acute time point.

Hypothesis 1.1: Participants with a mTBI will report more mood complaints, more subjective cognitive symptom complaints, and perform worse on EF measures compared to control participants.

Hypothesis 1.2: We hypothesize presence of subjective cognitive symptom complaints will negatively predict EF performance in both mTBI and control participants.

Hypothesis 1.3: Within the mTBI group only, symptom severity, as measured by the RPQ-13, will be positively correlated with subjective cognitive symptom complaints and negatively correlated with EF.

Specific Aim 2: To evaluate the role of mood in understanding group differences in EF and subjective cognitive symptom complaints.

Hypothesis 2.1: The group differences in EF and subjective cognitive symptom complaints are due to mood problems.

Specific Aim 3: Examine changes in mood and subjective cognitive symptom complaints in the mTBI group over time and determine if demographic factors account for some of the variance in this relationship.

Hypothesis 3.1: Consistent with the literature on mTBI recovery, we hypothesize participants with a mTBI will report fewer mood complaints and fewer subjective cognitive symptom complaints at the chronic time point compared to the sub-acute time point.

Hypothesis 3.2: We expect reductions in subjective cognitive symptom complaint reporting in mTBI participants between the sub-acute and chronic time points will be moderated by mood. We expect demographic variables, specifically ethnicity will account for some of the variance in the relationship with mood.

METHODS

Experimental Design

The current study is a secondary analysis of data collected at The University of New Mexico Health Sciences Center (UNMHSC). The UNMHSC Human Research Protections Office provided IRB approval of the study and all participants provided written informed consent. All participants were aged 18-55, were fluent in English, had no premorbid major medical or psychiatric conditions, no history of alcohol or substance abuse, and were not currently taking medications that interfere with cognitive functioning, with the exception of selective serotonin reuptake inhibitors.

Participants. mTBI patients were recruited from the Departments of Neurosurgery and Emergency Medicine from UNMHSC within two weeks following their injury. Participants from the mTBI group had a Glasgow Coma Scale of 13-15 if available (this information was sometimes missing from their records) and had experienced a brief loss of consciousness following injury. Control participants included sex- and age- matched individuals from the Albuquerque, New Mexico community. None of the mTBI or control participants had a previous head injury.

mTBI and control participants were invited to three assessment sessions. Session 1 was scheduled from 3-14 days post-injury. Session 2 was ~2 months and Session 3 was ~4 months (this data was collected but was not be used in the present studies analysis)

following Session 1. Participants were paid $20, $25, or $30/hour for participation in each respective session. One acute patient was removed from analysis of the third session for a head injury after their second session.

Questionnaires and Neuropsychological Assessments

All participants completed demographic and neuropsychological assessments in their first session. Only participants who scored above 45 on part one of the Test of Memory Malingering (TOMM) (Tombaugh, T. N, 1996) are included in these analyses. The TOMM is a widely used forced choice visual recognition test designed to distinguish between true memory impairments and malingering (Denning and Shura, 2017). TOMM trial 1 data has been reported to have higher sensitivity and accuracy compared to TOMM trial 2 in identifying invalid cognitive performance (Denning, 2012). Further, in mTBI samples a cut score of approximately ≤40 provides high levels of accuracy in predicting performance on the TOMM (Denning, 2012).

The Test of Premorbid Functioning. Participants completed the Test of Premorbid Functioning to assess preinjury functioning (TOPF, Pearson, 2009). The TOPF is an updated version of the Wechsler Test of Adult Reading (WTAR; Wechsler, 2001) and is standardized with the Wechsler Adult Intelligence Scale Fourth Edition (WAIS-IV; Wechsler, 2008). The TOPF is based on a reading paradigm wherein examinees are presented with a list of 70 words that have atypical grapheme-to-phoneme translations and are asked to read the words aloud. Individuals are not required to have knowledge or comprehension of word meaning (Pearson, 2009).

Symptom measures. Symptom, severity, mood, and executive functioning questionnaires were administered in each session. Symptom related measures included

the Neurobehavioral Symptom Inventory (NSI) (Cicerone & Kalmer, 1995) the Frontal Systems Behavior Scale (FrSBe) (Grace & Malloy, 2001) and the Patient-Reported Outcomes Measurement Information System (PROMIS, 2015).

Neurobehavioral Symptom Inventory (NSI). The NSI is a 22-item symptom checklist and each item is rated on a 5-point Likert scale from 0 (none) to 4 (very severe) wherein the patient rates how much the symptoms have impacted the patient in the past two weeks (Cicerone & Kalmar, 1995). Cronbach's alpha for the comprehensive scale was 0.95, which indicates a high degree of internal consistency. High alphas for each of the three subscales, Somatic, Affective, and Cognitive, are observed as well (0.88, 0.91, 0.92 respectively). There is a high degree of correspondence among most NSI items and the NSI total score, particularly anxiety, forgetfulness, difficulty concentrating, and poor frustration tolerance (r = 0.80 to 0.83) (King et al., 2012).

Frontal Systems Behavior Scale (FrSBe). The FrSBe is a behavior rating-scale comprised of 46 items, designed to measure dysfunctions regarding behaviors associated with the frontal regions of the brain (Grace & Malloy, 2001). Each item is rated on a 5-point Likert scale from 1 (almost never) to 5 (almost always) wherein the participants rate how they believe they were functioning before the injury and at present (after the injury). The FrSBe yields four scores for each time point (i.e., before the injury and after the injury): a total score, and three subscores (Apathy, Disinhibition, and Executive Dysfunction, respectively consisting of 14, 15, and 17 items). Higher scores are indicative of poorer self-reported functioning (Grace & Malloy, 2001). The total score and the three subscales have shown high internal consistency, satisfactory test-retest reliability in multiple studies Malloy & Grace, 2005). This suggests that the scales have

strong construct, discriminant, convergent, and ecological validity (Malloy & Grace, 2005). Specifically, high internal consistency has been shown in the FrSBe manual which reports α coefficients of 0.88, 0.72, 0.75, and 0.79 for the Total, Apathy, Disinhibition, and Executive Dysfunction scales for the self-report form in a normative sample respectively (Grace & Malloy, 2001). Factor-analyses of the FrSBe for clinical use in a population of patients with traumatic brain injury indicated that the total-score is an appropriate measure when considering its psychometric properties (Niemeier, Perrin, Holcomb, Nersessova, & Rolston, 2013). Reid-Arndt and colleagues (2007) found the FrSBe to be a significant predictor of community integration outcomes following traumatic brain injury. Specifically, their results showed more executive complaints (FrSBe Executive Dysfunction) predicted lower community integration Total Scores. Further, they found that increased apathy (FrSBe Apathy) was associated with reduced community integration productivity (Reid-Arndt, Nehl, & Hinkebein, 2007). Normative data for the FrSBe comes from the FrSBe Professional Manual and the normative sample is comprised of 436 men and women ranging in age from 18 to 95 years and in education from 10 years to doctoral level. The normative tables stratified for gender, age, and education provide *T* scores (Malloy & Grace, 2005).

Patient-Reported Outcomes Measurement Information System (PROMIS). The PROMIS is a National Institute of Health Roadmap initiative that is designed to develop self-report measures to assess well-being and functioning in the mental, physical, and social domains of health (Hays, Spritzer, Schalet, & Cella, 2018). The PROMIS Network develops and tests large banks of items related to health and researchers in turn select domains of functioning that related to their specific research question. The PROMIS is

designed to be used across all medical populations. The PROMIS-29 Profile v1.0 (PROMIS, 2015) assess seven health-related quality of life domains: physical functioning, anxiety, depression, fatigue, sleep disturbance, satisfaction with participation in social roles, pain interference, and pain intensity. Each domain provides separate specific instructions for score rankings. The physical functioning domain is ranked on a 5-point scale from 1 (without any difficulty) to 5 (unable to do) and does not provide a time frame. Participants are instructed to respond to the remainder of the questions in terms of their functioning over the past seven days. Questions related to anxiety and depression are ranked on a 5-point scale from 1 (never) to 5 (always). The domains of fatigue, sleep disturbance, satisfaction of social role, and pain interference are ranked 1 (not at all) to 5 (very much). Within the sleep disturbance domain, one question referring to sleep quality is ranked 1 (very poor) to 5 (very good). Pain intensity is ranked on a 10-point scale from 0 (no pain) to 10 (worst pain imaginable). In this study we examined the sleep disturbance, pain intensity, and pain interference scores as the captured additional information on the domains of sleep and pain that were not available in the NSI or FrSBe. Sleep disturbance, as previously discussed, is one of the most common complaints following a mTBI (Wickwire et al., 2016).

Rivermead Post-Concussion Symptoms Questionnaire (RPQ). mTBI severity was reported using the Post-Head Injury Symptoms Questionnaire that was adapted from the Rivermead Post-Concussion Symptoms Questionnaire (RPQ) (King, Crawford, Wenden, Moss, & Wade., 1995). In this measure, TBI post-concussive symptom severity is defined via self-report wherein individuals are asked to rate 16 post-concussive symptoms on a scale of 0 (not experienced at all) to 4 (severe problem) (Guise et al.,

2015). The 16 symptoms are divided into three subscales: cognitive (poor concentration, poor memory, taking longer to think), emotional (depression, frustration, irritability, restlessness), and somatic (dizziness, nausea, headache, noise sensitivity, fatigue, sleep disturbance, blurred vision, double vision, light sensitivity). Similarly, factor analysis of the RPQ finds a three-factor model of cognitive, emotional, and somatic factors, with a high degree of covariation overall. The two-factor model combined somatic and emotional factors into one factor and resulted in a similar goodness-of-fit to the data (Potter, Leigh, Wade, & Fleminger, 2006).

The test is divided into the RPQ-13 and the RPQ-3, with the RPQ-13 items assessing mood, cognition, sleep, and other physical symptoms that are later symptoms of post-concussion syndrome. The RPQ-3 items assess dizziness, headaches, nausea and/or vomiting and are early concussion symptoms. It is recommended that analyses are conducted separately for the RPQ-13 and the RPQ-3 because individually they demonstrate good construct validity and test-retest reliability, which is lost when they are combined (Eyres, Carey, Gilworth, Neumann, & Tennant, 2005). In the present study, per recommendations from the literature, the RPQ-13 will be used to assess mTBI severity.

Beck Depression Inventory-II (BDI-II). The Beck Depression Inventory (BDI-II) was administered as the mood measure in this study (Beck et al., 1996). The BDI-II is a 21-item self-report measure of the intensity of depressive symptoms for individuals ages 13 to 80. Individuals are asked to respond to statements about their mood over the past two weeks on a 4-point scale, with the overall score obtained by summing the ratings for all items. Scores ranging between 0 and 13 are indicative of minimal depression; scores that fall between 14 and 19 are considered to reflect a mild level of depression; scores of

20 to 28 are considered moderate; and a score ranging from 29 to 63 is labeled severe (Beck et al., 1996).

Executive Abilities: Measure and Instruments for Neurobehavioral Evaluation and Research (NIH-EXAMINER). EF was measured using the Executive Abilities: Measure and Instruments for Neurobehavioral Evaluation and Research (NIH-EXAMINER) (Kramer et al., 2014.) Analyses were conducted using the Executive Composite from the NIH-EXAMINER as well as the three factor measures provided by the test: the Fluency Factor, Cognitive Control Factor, and the Working Memory Factor. The EXAMINER aims to assess EF deficits in a variety of neurobehavioral conditions and is designed to be efficient, modifiable, and modular. The test is suitable for a broad range of ages and ability levels, available in Spanish and English, and notably includes individuals with TBI in its diagnostic cohort. The test is separated into domains of working memory, inhibition, set shifting, fluency, planning, insight, and social cognition and behavior. Regarding the psychometric properties of the NIH-EXAMINER, confirmatory factor analysis supports a one-factor and a three-factor model which are the basis for the executive composite score as well as subscale scores of Fluency, Working Memory, and Cognitive Control. Test-retest reliabilities for these scales range from .78 to .93 (Kramer et al., 2014).

Attrition. While the control group had relatively stable enrollment, the sub-acute mTBI group suffered from attrition, with about ¾ of participants returning for the second session. The consort diagram is shown in Figure 1.

Statistical Analyses

Data analysis was conducted using IBP SPSS version 23. SPSS Explore was used to detect extreme outliers and examine the distribution characteristics of all variables.

At the sub-acute time point, four variables relating to subjective symptom reporting (NSI emotion, NSI cognitive, NSI somatic, FrSBe total) were factor analyzed using principal component analysis with Direct Oblimin rotation. This analysis yielded one factor explaining a total of 78.56% of the variance for the entire set of variables. This factor was termed the Symptom Composite PC factor. This procedure was repeated for the chronic time point, again using the four subjective symptom reporting variables measured at the chronic time point. The second analysis yielded one factor explaining a total of 75.08% of the variance for the entire set of variables. This factor was reported as the Symptom Composite PC factor at the chronic time point. The Symptom Composite PC variables at the two time points were highly correlated ($r(63) = .69$, $p < .001$).

Symptom Composite PC z-scores were transformed into standard scores at both time points. Similarly, Executive Composite, Fluency Factor, Cognitive Control, and Working Memory scores were transformed into standard scores for ease of interpretation.

Specific Aim 1. A series of two-way ACNOVA's were conducted to evaluate the relationship between mood, subjective cognitive symptom complaints, and EF performance in mTBI and control participants at the sub-acute time point. Pearson correlations were computed to examine the associations between subjective cognitive symptom complaints and EF performance in both mTBI and control participants. Additional Pearson correlations examined the relationship between mTBI symptom severity and subjective cognitive symptom complaints and correlations with EF.

Specific Aim 2. To evaluate the role of mood in understanding group differences in EF and subjective cognitive symptom complaints we computed a two-way ANCOVA with the Executive Composite at the sub-acute time point as the dependent variable, gender as a fixed factor and covariates of TOPF, BDI-II at the sub-acute time point, Symptom Composite PC at the sub-acute time point.

Specific Aim 3. A series of Repeated Measures ANCOVA's were conducted to examine changes in mood and subjective cognitive symptom complaints in the mTBI group over time and determine if demographic factors, specifically ethnicity account for some of the variance in the relationship with mood. Six Repeated Measures ANCOVA's with a Greenhouse-Geisser correction were run with TOPF as a covariate and group and gender as between subjects factors for each of the relevant variables (BDI-II, Symptom Composite PC, and Executive Composite, Fluency Factor, Cognitive Control Factor, and Working Memory Factor) to establish if results changed between the sub-acute and chronic time points. The models examined main effects and interactions. Pearson correlations were run to assess the role of TOPF in the models. To address the ethnicity component of this aim we computed an additional Repeated Measures ANCOVA for the BDI-II at the sub-acute time point with a Greenhouse-Geisser correction. TOPF and ethnicity were selected as covariates and group and gender were between-subjects factors.

RESULTS

Demographic Characteristics

The demographic data for the mTBI and control groups are presented in Table 1 and Table 2. The 84 individuals who participated in the study included more men than

women, although the difference did not reach statistical significance. The majority identified themselves as Non-Hispanic White (NHW) with Hispanic being the second most prominent group. A majority of the individuals reported earning their high school diploma/GED or continuing onto higher education. In both groups, most of the individuals were currently employed with a larger proportion of individuals who identified as unemployed or disabled in the mTBI group than in the control group. The average age of individuals was 29.20 years (SD = 10.32). Similarly, the average age for individuals in the mTBI group was 28.96 years (SD = 10.24) and 29.59 years (SD=10.60) for individuals in the control group. With regard to time since the injury, the mTBI group was assessed within two weeks of sustaining their injury ($\bar{X}$= 9.92 days, SD = 3.22). The majority, 84.5%, of participants were assessed between eight- and fourteen-days post injury.

mTBI Injury Characteristics

For descriptive purposes, we next report only on individuals who had sustained a mTBI. Quantifying mTBI participants by mechanism of injury, the largest proportion of individuals sustained their mTBI in a motor vehicle accident, approximately 52% (Figure 2). Stratifying mechanism of injury by gender in Table 5, a chi square test indicated no significant association between gender and mechanism of injury ($\chi_2(3, N = 52) = 5.10$, $p = .16$). Both female and male participants were equally injured by assault, motor vehicle accident, fall and sports-related injuries. A one-way ANOVA was conducted to examine the role of the ethnicity variable in mTBI. In the mTBI sample, severity ratings as measured by the Rivermead Post Concussive Symptom Questionnaire (RPQ-13) were not significantly different between NHW and Hispanic participants, $F(1,49) = .81$, $p = .37$.

Sleep disturbance ratings and pain ratings from the PROMIS were explored in the mTBI group, as these symptoms are common after mTBI. The two pain measures selected from the PROMIS were pain intensity and pain interference. Pain intensity refers to the severity of pain rated on a 10-point scale from no pain to worst imaginable pain. Pain interference refers to the degree to which pain interferes or limits an individual's mental, physical and social activities. BDI-II, pain intensity, pain interference, and sleep disturbance scores were found to be very highly correlated at the sub-acute time point, Table 3.

In the mTBI group, subjective symptom reporting measures (NSI somatic, NSI emotion, NSI cognitive, FrSBe total after score) positively correlated with reports of sleep disturbance, pain intensity and pain interference, see Table 4. The Executive Composite score did not significantly correlate with any of the subjective symptom reporting measures or reports of sleep disturbance, pain interference, or pain intensity (Table 4). One-way ANOVAs found that mTBI participants experienced significantly greater levels of sleep disturbance compared to control participants, $F(1,82) = 18.88$, $p < .01$. Similarly, mTBI participants reported significantly greater levels of pain interference ($F(1,82) = 45.87$, $p < .01$) and pain intensity ($F(1,82) = 78.72$, $p < .01$) compared to control participants.

Measures

Outliers. SPSS Explore procedure was used to identify extreme outliers in the data sample for the mood, symptom, and executive functioning variables. No extreme outliers were found in the data set and therefore no additional procedures were implemented.

Relationships among subjective symptom reporting measures. The relationship among the subjective symptom reporting measures, specifically the NSI and the FrSBe, were examined to determine if data reduction was justified. As previously discussed, both of these measures, the NSI and FrSBe, were originally designed to capture TBI symptom sequelae and therefore appeared to be most relevant in addressing our hypotheses about subjective symptom reporting. Both the NSI and FrSBe assess participants perceived mTBI symptoms and measure complimentary functioning impairments.

In the entire sample, Pearson correlations among the symptom report measures were computed at the sub-acute time point. The NSI is comprised of three sub-scores, somatic, cognitive, and emotional and one total score. The somatic score is highly positively correlated with the cognitive ($r(83) = .85$, $p < .01$) and emotional scores ($r(83) = .82$, $p < .01$). The cognitive score is similarly highly correlated with the emotional score, $r(83) = .86$, $p < .01$. As expected, all three sub-scores are also highly correlated with the total score with the values ranging from r =. 93 to r = .96, see Table 7. High intercorrelations were also found between the NSI total score and FrSBe total after scores ($r(83) = .59$, $p < .01$).

The BDI-II was examined in relation to the subjective symptom reporting measures because the BDI-II, like the NSI and FrSBe, assesses symptoms that may present following a mTBI. Pearson correlations of the BDI-II, NSI total score, and FrSBe were computed and the BDI-II was found to correlate with the NSI total score ($r(38) = .75$, $p < .01$) and FrSBe ($r(84) = .59$, $p < .01$). While significant correlations were

found between symptom and BDI-II measures, we decided to characterize the BDI-II separately as a mood measure.

The BDI-II was next examined to determine the percentage of participants in the entire sample and in the respective groups who met the BDI-II criteria for experiencing minimal (0-13), mild (14-19), moderate (20-28), and severe depression symptoms (29-63). In the entire sample, 67.4% of participants reported experience minimal depression symptoms, 14% mild, 8.1% moderate, and 8.1% severe depression symptoms. In the mTBI group 59.6% reported minimal depression symptoms, 17.3% mild, 9.6% moderate, and 13.5% severe depression symptoms. In the control group the largest percentage, 84.4% reported minimal depression symptoms, 9.4% mild, and 6.3% moderate, and no participants in the control group reported experiencing severe depressions symptoms (see Figure 3). BDI-II scores at the sub-acute and chronic time points for mTBI and control participants is presented in Figure 5.

Relationships among neuropsychological assessment measures. Relationships between the neuropsychological measures used in this study were examined to provide support for inclusion or exclusion of variables in the analyses that follow. Neuropsychological measures included the NIH-EXAMINER and the Test of Premorbid Functioning (TOPF). Pearson correlations among the executive functioning measures were computed to examine relationships among these measures to provide justification for use of the executive composite score in the analyses. The NIH-EXAMINER provides four scores, a fluency factor, cognitive control factor, a working memory factor, and executive composite score. As expected, the fluency ($r(76) = .80$, $p < .01$), cognitive control ($r(76) = .62$, $p < .01$) and working memory factors ($r(76) = .66$, $p < .01$) are highly

correlated with the executive composite score, see Table 8. The cognitive control and working memory factors are correlated ($r(76) = .31$, $p = .006$) and the fluency factor is correlated with the working memory factor ($r(76) = .34$, $p = .002$). Notably, the fluency factor is not correlated with the cognitive control factor $r(76) = .192$, $p = .096$. Given that the NIH-EXAMINER provides the executive composite and we reported high intercorrelations it was deemed appropriate for use in the following analyses.

Following we examined the TOPF standard score as a possible covariate in our Repeated Measures analyses. One would expect the TOPF standard score would be strongly positively related to years of education and cognitive test scores. Pearson correlations found the TOPF total standard score correlated with years of education ($r(83) = .463$, $p < .01$). Independent t-tests (Table 6) revealed significant group differences in TOPF performance between the mTBI and control groups with control participants earning higher scores compared to those who sustained a mTBI. A histogram of TOPF scores by group is presented in Figure 7. Given these findings, the TOPF was selected as a covariate to address our hypotheses using Repeated Measures analyses.

Group differences. The assessment measures used in the analyses that follow are presented in Table 6. For descriptive purposes we also report independent samples t-tests between both groups at the sub-acute time point. In comparing mTBI and control participants, individuals with a mTBI had significantly higher scores on the BDI-II, all symptom variables, and the Symptom Composite PC. Similarly, control participants showed significantly higher scores on the executive composite score and the fluency factor. No significant difference was found between the cognitive control or working memory factors from the NIH-EXAMINER. Overall, participants with a mTBI had

higher rates of depressive and subjective symptom complaints compared to healthy controls, and healthy controls had slightly better overall executive functioning performance compared to mTBI participants.

Data Reduction. Given the high intercorrelations between the symptom variables (NSI emotion, NSI cognitive, NSI somatic, FrSBe total), these four variables were analyzed using principal components analysis with Direct Oblimin rotation. This analysis yielded one factor explaining a total of 78.56% of the variance for the entire set of variables. This factor was termed the Symptom Composite PC factor. This procedure was repeated for the chronic time point, again using the four subjective symptom reporting variables measured at the chronic time point. The second analysis yielded one factor explaining a total of 75.08% of the variance for the entire set of variables. This factor was reported as the Symptom Composite PC factor at the chronic time point. The Symptom Composite PC variables at the two time points were highly correlated ($r(63) = .69$, $p < .01$).

Follow-up Pearson correlations were computed between the Symptom Composite PC variable and the mood and symptom variables. As expected, the Symptom Composite PC score is significantly correlated with the symptom variables, NSI total score ($r(83) = .976$, $p < .01$) and the FrSBe total score ($r(83) = .742$, $p < .01$). The composite variable is also highly correlated with BDI-II, $r(84) = .798$, $p < .01$.

Cultural Considerations

Cultural expressions of emotional functioning, symptom reporting, and performance on executive functioning measures may influence the outcome of a mTBI, therefore ethnicity was examined in the entire sample at the sub-acute time point.

Assessment of the relevance of ethnicity is complicated by the variety of ethnic labels participants chose to describe themselves. For the current study we collapsed ethnic variation into two categories, NHW and Hispanic. Thus, two-way ANOVAs (group, ethnicity) were computed for the BDI-II, NSI somatic, NSI cognition, NSI emotion, FrSBe, Symptom Composite PC, Executive Composite, Fluency Factor, Cognitive Control Factor, and Working Memory Factor at the sub-acute time point for all participants.

Main group effects for ethnicity were found for the BDI-II ($F(1, 83) = 10.60$, $p = .002$, $\eta^2 = .12$), NSI somatic ($F(1,82) = 22.84$, $p < .001$, $\eta^2 = .22$), NSI cognition ($F(1,82) = 15.70$, $p < .01$, $\eta^2 = .17$), NSI emotion ($F(1,82) = 13.34$, $p < .01$, $\eta^2 = .14$), FrSBe ($F(1,83) = 6.37$, $p = .01$, $\eta^2 = .07$), Symptom Composite PC ($F(1,82) = 19.85$, $p < .01$, $\eta^2 = .20$), Executive Composite ($F(1,75) = 7.60$, $p = .007$, $\eta^2 = .10$), and the Fluency factor ($F(1,75) = 8.59$, $p = .005$, $\eta^2 = .11$). No significant main effects were found for the Cognitive Control factor ($F(1, 75) = 2.82$, $p = .10$, $\eta^2 = .04$) or Working Memory factor ($F(1,75) = . 06$, $p = .80$, $\eta^2 = .00$). Importantly, no group by ethnicity interactions were found for the BDI-II ($F(1,83) = .01$, $p = .97$, $\eta^2 = .00$), NSI somatic ($F(1,82) = .19$, $p = .66$, $\eta^2 = .00$), NSI cognition ($F(1,82) = .61$, $p = .44$, $\eta^2 = .01$), NSI emotion ($F(1,82) = .29$, $p = .59$, $\eta^2 = .00$), FrSBe ($F(1,83) = 1.09$, $p = .30$, $\eta^2 = .01$), Symptom Composite PC ($F(1,82) = .02$, $p = .90$, $\eta^2 = .00$), Executive Composite ($F(1,75) = .89$, $p = .35$, $\eta^2 = .01$), Fluency factor ($F(1,75) = .02$, $p = .89$, $\eta^2 = .00$), Cognitive Control factor ($F(1,75) = 2.82$, $p = .10$, $\eta^2 = .04$), and Working Memory factor ($F(1,75) = .06$, $p = .80$, $\eta^2 = .00$).

Additional two-way ANOVA's on sleep and pain measures from the PROMIS revealed no significant interactions between group and ethnicity for sleep disturbance

(F(1,82) = 2.21, p = .14, η^2 = .03), pain intensity (F(1,82) = 1.82, p = .18, η^2 = .02), or pain interference (F(1,82) = .57, p = .45, η^2 = .01).

Statistical Analyses

Hypothesis 1a. A series of two-way ANCOVA's were conducted to evaluate if mTBI participants reported more mood complaints, more subjective cognitive symptom complaints, and performed worse on EF measures compared with control participants at the sub-acute time point. As previously reported, univariate t-tests revealed significant differences for all relevant variables except for the Cognitive Control Factor and Working Memory Factor at the sub-acute time point, see Table 6. To determine if both variables could be included in the same model, a Pearson correlation was computed for the BDI-II and Symptom Composite PC at the sub-acute time. The Pearson correlation revealed a highly significant positive correlation (r(83) = .80, p < .01). Given this result it was determined the models should be run separately to avoid problems due to multicollinearity.

Model 1.1: Mood as measured by the BDI-II. Differences in scores on the BDI-II between the two participant groups at the sub-acute time point were assessed in model one. We hypothesized individuals who sustained a mTBI would report more negative mood complaints, as measured by the BDI-II, compared to control participants. A two-way ANCOVA with TOPF as a covariate and group and gender as between-subjects factors was conducted to address the mood component of Hypothesis 1a. Between-subjects effects revealed a significant main effect of group, F(1,82) = 4.69, p < .01, η^2 = .19, indicating the mTBI group reported experiencing significantly more distress than the control group at the sub-acute time point. This finding provides support for the mood

component of Hypothesis 1a. A significant positive main effect of TOPF was revealed $F(1,82) = 4.69$, $p = .03$, $\eta^2 = .06$. No other main effects or interactions were significant.

Model 1.2: Subjective Symptom Complaints as measured by the Symptom Composite PC. Model 1.2 explored whether mTBI and control participants differed in terms of cognitive symptom complaint reporting. A two-way ANCOVA with TOPF as a covariate and group and gender as between-subjects factors was conducted to address the subjective symptom reporting component of Hypothesis 1a. Analyses revealed a significant between-subjects main effect of group, $F(1,81) = 27.07$, $p < .01$, $\eta^2 = .26$. mTBI participants reported experiencing significantly more symptom distress compared to control participants providing support for the symptom component of Hypothesis 1a. No other between-subjects effects were significant.

Model 1.3: Executive Functioning as measured by NIH-EXAMINER Executive Functioning Composite. The next model aimed to examine the role of EF, as measured by the NIH-EXAMINER, between the mTBI and control participants. We proposed mTBI participants would earn lower EF scores compared to their control counterparts. The preceding two-way ANCOVA was repeated for the Executive Composite. No between group differences were found in EF performance between mTBI and control participants ($F(1,74) = 1.19$, $p = .28$, $\eta^2 = .02$). Notably, while the univariate t-test was significant for the Executive Composite, the current analysis was not significant and therefore does not provide support for Hypothesis 1a. Given mixed findings for the NIH-EXAMINER Executive Composite, it was deemed necessary to examine the respective components of the composite individually, in the following models. A significant main effect of TOPF, $F(1,74) = 44.42$, $p < .01$, $\eta^2 = .39$ was revealed in this analysis.

Individuals with higher premorbid intelligence had higher EF scores. No other group effects were found to be significant.

Model 1.4: Fluency Factor of the NIH-EXAMINER. Given the mixed findings in model 1.3, three additional two-way ACNOVA's were computed to examine the role of the three factors that comprise the Executive Functioning Composite. Model 1.4 explored the Fluency Factor. We hypothesized individuals with a mTBI would score lower on the Fluency Factor measure compared to controls. Analogous to the preceding model, no between group differences were found in performance on the Fluency Factor between mTBI and control participants ($F(1, 74) = 3.45$, $p = .07$, $\eta^2 = .05$). Similar to model 1.3 a significant main effect of TOPF, $F(1,74) = 24.37$, $p < .01$, $\eta^2 = .26$ was revealed in this analysis. Individuals with higher premorbid intelligence had higher EF scores. No other group effects were found to be significant. The findings of this analysis do not provide support for Hypothesis 1a.

Model 1.5: Cognitive Control Factor of the NIH-EXAMINER. Model 1.5 explored the Cognitive Control Factor. As before, we expected participants with a mTBI would score lower on the Cognitive Control Factor compared to control participants. Analyses again revealed no between group differences in performance on the Cognitive Control Factor ($F(1,74) = .21$, $p = .65$, $\eta^2 = .00$) but did reveal a significant main effect of TOPF, $F(1,74) = 6.30$, $p = .01$, $\eta^2 = .08$. No other group effects were found to be significant. The findings of this analysis do not provide support for Hypothesis 1a.

Model 1.6: Working Memory Factor of the NIH-EXAMINER. The final model of Hypothesis 1a examined the Working Memory Factor. Similar to the preceding two models, analyses revealed no between-subjects group differences in performance on the

Working Memory Factor ($F(1,74) = .003$, $p = .95$, $\eta^2 = .00$). A significant main effect of TOPF was again present, $F(1,74) = 10.13$, $p = .002$, $\eta^2 = .13$). No other group effects were revealed to be significant. Again, these findings do not provide support for Hypothesis 1a.

Hypothesis 1a overall findings. Analyses revealed significant between-subjects effects for the BDI-II and Symptom Composite PC. No significant between-subjects group effects were found for the Executive Composite, Fluency Factor, Cognitive Control Factor, or Working Memory Factor. Therefore, these results do not provide support for Hypothesis 1a. mTBI participants reported significantly more mood complaints and more subjective cognitive symptoms than control participants at the sub-acute time point. In contrast, no significant differences in EF performance was found between the two groups.

Hypothesis 1b. We hypothesized that the presence of subjective cognitive symptom complaints, as measured by the Symptom Composite PC, will negatively predict EF performance in both mTBI and control participants. It was expected that the presence of more symptoms would result in poorer performance on objective neuropsychological assessment measures. While these variables were found to be negatively correlated, they did not reach significance ($r(75) = -.07$, $p = .52$). Therefore, Hypothesis 1.b was not supported.

Hypothesis 1c. Pearson correlations were conducted to assess if severity of the mTBI, as measured by the RPQ-13, was positively correlated with subjective cognitive symptom complaints and negatively correlated with EF. As expected, Pearson correlations revealed the severity rating positively correlated with the Symptom

Composite PC at the sub-acute ($r(50) = .81$, $p < .01$) and chronic time point ($r(36) = .57$, $p < .01$), indicating that the severity of the mTBI was related to subjective symptom complaint reporting. In contrast, Pearson correlations of severity and the Executive Composite were not significantly positively correlated at the sub-acute ($r(46) = .19$, $p = .20$) or chronic time points ($r(32) = .30$, $p = .09$). Therefore, Hypothesis 1c was not supported.

Hypothesis 2. We proposed the group differences in EF and subjective cognitive symptom complaints are due to mood problems. A two-way ANCOVA was computed with the Executive Composite at the sub-acute time point as the dependent variable, gender and group as fixed factors, and with covariates of TOPF, BDI-II at the sub-acute time point, Symptom Composite PC at the sub-acute time point. Between-subjects effects were revealed to be nonsignificant for the BDI-II ($F(1,73) = .17$, $p = .68$, $\eta^2 = .002$) and Symptom Composite PC ($F(1,73) = .02$, $p = .88$, $\eta^2 = .00$) but were significant for the TOPF ($F(1,73) = 57.56$, $p < .01$, $\eta^2 = .45$). This process was repeated at the chronic time point and yielded similar results. A significant main effect of TOPF was found ($F(1,73) = 25.53$, $p < .01$, $\eta^2 = .35$), but no other between-subjects effects were significant. Overall, participants EF scores did not change relative to their mood or subjective symptom reporting. Hypothesis 2 was not supported.

Hypothesis 3a. Consistent with the literature on mTBI recovery, we hypothesized participants with a mTBI would report fewer mood complaints and fewer subjective cognitive symptom complaints at the chronic time point compared to the sub-acute time point. To assess change over time for our relevant variables, we began by determining if within group differences in performance were present for each group across the two time

points. Following we assessed for between-subjects effects. Consistent with the statistical procedure implemented in Hypothesis 1a the models were run separately to avoid inflation due to multicollinearity.

Model 3.1: Mood as measured by the BDI-II. We hypothesized individuals who sustained a mTBI would report fewer mood complaints at the chronic time point compared to the sub-acute time point as we expected their mood to improve over time. A Repeated Measures ANCOVA with a Greenhouse-Geisser correction was computed with TOPF as a covariate and group and gender as between-subjects factors to address the mood component of Hypothesis 3a.

a. Examining within-subjects effects, a significant main effect of time, $F(1,59) = 7.07$, $p = .01$, $\eta^2 = .12$, was found, indicating mTBI participants mood changed slightly over time (Figure 4). Analyses revealed no significant interactions. The time by group interaction ($F(1,59) = 1.93$, $p = .17$, $\eta^2 = .03$) and time by gender interaction ($F(1,59) = .00$, $p = .99$, $\eta^2 = .00$) was not significant. Further, the three-way interaction between time, group, and gender was not significant ($F(1,59) = .41$, $p = .52$, $\eta^2 = .01$). Analyses revealed a significant time by TOPF interaction ($F(1,59) = 6.36$, $p = .01$, $\eta^2 = .09$). Premorbid intellectual ability was differently related to mood at the two time points. To clarify the role of the TOPF in this analysis, a median split of TOPF was computed at the sub-acute time point. A one-way ANOVA with group and TOPF median split as fixed factors revealed a significant main effect of group, $F(1,82) = 8.58$, $p = .004$, $\eta^2 = .10$, see Figure 6.
b. Regarding between-subjects effects, there was a significant main effect of group, $F(1,59) = 12.11$, $p = .001$, $\eta^2 = .17$, indicating that the mTBI group had

significantly greater (worse) mood scores than the control group at both time points. No other between-subjects effects were found to be significant.

Model 3.2: Subjective Symptom Complaints as measured by the Symptom Composite PC. Model 3.2 examined whether subjective cognitive symptom reporting would change over time for mTBI participants. A Repeated Measures ANCOVA with a Greenhouse-Geisser correction was computed with TOPF as a covariate and group and gender as between-subjects factors.

a. Within-subjects effects revealed a significant main effect of time ($F(1,57) = 7.65$, $p = .008$, $\eta^2 = .12$), indicating overall participants reported less subjective symptom complaints at the chronic time point, Figure 8. Within the groups, a time by group interaction was found, $F(1,57) = 4.82$, $p = .032$, $\eta^2 = .08$. No time by gender interaction was found, $F(1,57) = .002$, $p = .99$, $\eta^2 = .00$ and a three-way interaction between time, group, and gender was found to not be significant, $F(1,57) = 1.11$, $p = .32$, $\eta^2 = .02$. A significant time by TOPF interaction was found, $F(1,57) = 7.25$, $p = .01$, $\eta^2 = .11$, indicating that premorbid intellectual ability was differently related to symptom reporting at the two time points. This finding resembles the effect found in Model 3.1 and is expected given the high correlation between the BDI-II and the Symptom Composite PC.
b. Regarding between-subjects effects, there was a significant main effect of group ($F(1,57) = 13.20$, $p = .001$, $\eta^2 = .19$). Participants reported experiencing more subjective cognitive symptom complaints than did control participants at both time points, see Figure 8. No other between-subjects effects were found to be significant.

Model 3.3: Executive Functioning as measured by NIH-EXAMINER Executive Functioning Composite. The third model examined change over time in EF performance, as measured by the NIH-EXAMINER Executive Composite for mTBI participants. We proposed that Executive Composite scores would improve over time. A Repeated Measures ANCOVA with a Greenhouse-Geisser correction was computed with TOPF as a covariate and group and gender as between-subjects factors.

a. With regard to within-subjects effects, a significant main effect of time, $F(1,49) = 4.50$, $p = .039$, $\eta^2 = .08$, was found, indicating that Executive Composite scores improved overall across the two time points, Figure 9. The time by group interaction was not significant, $F(1,49) = .10$, $p = .75$, $\eta^2 = .002$. Further there was no time by TOPF interaction within each group, $F(1,49) = 3.66$, $p = .06$, $\eta^2 = .17$, indicating that within each group the participants scores on the TOPF did not impact EF as measured by the Executive Composite score. No time by gender interaction was found, $F(1,49) = .02$, $p = .88$, $\eta^2 = .000$ and a three-way interaction between time, group, and gender was found to not be significant, $F(1,49) = .001$, $p = .98$, $\eta^2 = .00$.

b. Between-subjects effects were not significant for group $F(1,49) = .26$, $p = .61$, $\eta^2 = .005$. The partial eta squared value was converted into Cohen's d for comparison to the literature estimates of EF effect size, d = .14. There was a significant main effect of TOPF, $F(1,49) = 31.30$, $p < .01$, $\eta^2 = .39$, indicating individuals with higher premorbid intelligence received higher EF scores. No other group effects were found to be significant, Figure 9.

To further assess the findings in Model 3.3, three additional Repeated Measures ANCOVA's were computed for the three factors that make up the Executive Composite, the Fluency Factor, Cognitive Control Factor, and the Working Memory Factor.

Model 3.4: Fluency Factor of the NIH-EXAMINER. Model 3.4 explored change over time in performance on the Fluency Factor. We hypothesized individuals with a mTBI would earn higher scores on the Fluency factor at the chronic time point compared to the sub-acute time point. As before, a Repeated Measures ANCOVA with a Greenhouse-Geisser correction was computed with TOPF as a covariate and group and gender as between-subjects factors.

a. No main effect of time was found, $F(1,49) = 1.05$, $p = .31$, $\eta^2 = .02$, Figure 9. Within each group, the time by group interaction was found to be significant $F(1,49) = 4.43$, $p = .04$, $\eta^2 = .08$. Therefore, overall there was an improvement in fluency scores at the chronic time point. There was no time by TOPF interaction within each group, $F(1,49) = .61$ $p = .44$ $\eta^2 = .01$, indicating overall the participants scores on the TOPF did not impact fluency factor scores. No time by gender interaction was found, $F(1,49) = .07$, $p = .79$, $\eta^2 = .00$ and a three-way interaction between time, group, and gender was found to not be significant, $F(1,49) = 3.00$, $p = .09$, $\eta^2 = .06$.
b. Regarding effects between the mTBI and control participants, group was not significant, $F(1,49) = .61$, $p = .44$, $\eta^2 = .012$, $d = .22$. There was a significant main effect of TOPF, $F(1,49) = 14.185$, $p < .01$, $\eta^2 = .224$, indicating individuals with higher premorbid intelligence had higher fluency factor scores. No other between-subjects effects were found to be significant, Figure 9.

Model 3.5: Cognitive Control Factor of the NIH-EXAMINER. Model 3.5 examined change over time of the Cognitive Control Factor of the NIH-EXAMINER. We hypothesized that Cognitive Control Factor scores would improve at the chronic time point for mTBI participants. We again computed a Repeated Measures ANCOVA with a Greenhouse-Geisser correction with TOPF as a covariate and group and gender as between-subjects factors.

a. No within-subjects main effects or interactions were found for the Cognitive Control Factor.
b. Between-subjects effects revealed group was not significant, $F(1,49) = .47$, $p = .49$, $\eta^2 = .01$, $d = .20$. There was a significant main effect of TOPF, $F(1,49) = 8.33$, $p = .006$, $\eta^2 = .14$, indicating individuals with higher premorbid intelligence had higher Cognitive Control Factor scores. No other between-subjects effects were found to be significant, Figure 11.

Model 3.6: Working Memory Factor of the NIH-EXAMINER. Model 3.6 examined whether scores on the Working Memory Factor from the NIH-EXAMINER would change over time for mTBI participants. We again ran a Repeated Measures ANCOVA with a Greenhouse-Geisser correction with TOPF as a covariate and group and gender as between-subjects factors.

a. No within-subjects main effects or interactions were found for the cognitive control factor.
b. Regarding effects between the mTBI and control participants, group was not significant, $F(1,49) = .44$, $p = .51$, $\eta^2 = .009$, $d = .19$. There was a significant main effect of TOPF, $F(1,49) = 9.93$, $p < .01$, $\eta^2 = .17$, indicating that individuals with

higher premorbid intelligence had higher Working Memory Factor scores. No other between-subjects effects were found to be significant, Figure 1.

Hypothesis 3b. We expected reductions in subjective cognitive symptom complaint reporting, as measured by the Symptom Composite PC, in mTBI participants between the sub-acute and chronic time points will be moderated by mood. We expect demographic variables, specifically ethnicity, will account for some of the variance in this relationship. The Repeated Measures ANCOVA from model 3.2 revealed the time by group interaction was not significant for the Symptom Composite PC. There was no significant difference in subjective symptom complaint reporting between the mTBI and control group and over time.

We computed an additional Repeated Measures ANCOVA for the BDI-II at the sub-acute time point with a Greenhouse-Geisser correction. TOPF and ethnicity were selected as covariates and group and gender were between-subjects factors.

a. Examining within-subjects effects, a significant main effect of time, $F(1,58) = 4.91$, $p = .03$, $\eta^2 = .08$, was found, indicating mTBI participants mood became worse over time. A significant time by TOPF interaction was found, $F(1,58) = 4.93$, $p = .03$, $\eta^2 = .08$. All other within-subjects interactions were not significant.
b. Regarding between-subjects effects, there was a significant main effect of group, $F(1,58) = 12.47$, $p < .01$, $\eta^2 = .18$, indicating the mTBI group had significantly greater (worse) mood scores than the control group at both time points. No other between-subjects effects were found to be significant for ethnicity ($F(1,58) = .54$, $p = .47$, $\eta^2 = .009$).

Given that there was no change over time in symptom reporting and no main effect or interaction with ethnicity we are unable to further assess Hypothesis 3b.

DISCUSSION

This study included a secondary analysis of mTBI survivors and control participant's self-report of mood symptoms, subjective cognitive symptoms, and objective neuropsychological assessment of EF deficits. The purpose of the study was to explore and clarify the relationship among self-reported behavioral and cognitive complaints with objectively measured EF in individuals with mTBI in the sub-acute and chronic states of recovery. Specifically, this research attempted to explore if mood moderated the relationship between subjective cognitive complaints and objective EF as measured by the NIH-EXAMINER.

The data indicated at the semi-acute state of recovery, mTBI survivors reported experiencing higher levels of mood symptoms and higher rates of mTBI symptom sequelae. In contrast to Hypothesis 1a, mTBI participants performed as well as their non-injured counterparts on objective EF assessment. While this finding is not particularly novel, it does provide continued evidence for higher rates of depressive symptoms (Bombardier et al., 2010) and subjective symptom reporting (Ponsford, Cameron Fitzgerald, Grant, & Mikocka-Walus, 2011) in mTBI patients compared with health controls. With regard to EF scores as assessed via the NIH-EXAMINER, we did not find significant group effects for the Executive Composite or the three factors, Fluency, Cognitive Control, and Working Memory. Approximately two weeks post injury, mTBI participants performed approximately the same as healthy controls on our measure of EF.

We proposed in Hypothesis 1b that the presence of subjective cognitive symptom complaints would negatively predict EF performance for both mTBI and control participants. We expected that the presence of more symptoms would impact participants ability to perform successfully on objective neuropsychological assessment measures resulting in lower EF scores. While our Subjective Symptom PC and Executive Composite scores at the sub-acute time point were negatively correlated, this did not reach significance. Therefore, Hypothesis 1b was not supported. Similarly, we hypothesized (Hypothesis 1c) that mTBI patient's subjective reporting of symptom severity would positive correlated with our subjective symptom measures at both the sub-acute and chronic time points. As expected, our analyses revealed these variables were significantly correlated. Patients who reported experiencing more severe symptoms also reported experiencing many symptoms. As well, we believed mTBI patient's reporting of more severe symptoms would correlate with neuropsychological assessment performance. This was in fact not the case, and therefore Hypothesis 1c was also not supported.

Following, consistent with the literature, in Hypothesis 2 we proposed that the group differences in EF and subjective cognitive symptom complaints would be driven by mood problems. Our results revealed EF scores did not change relative to their mood or subjective symptom reporting. Hypothesis 2 was not supported.

Examining the impact of time on recovery from mTBI, we hypothesized mTBI participants would improve over time in all measured domains. The literature consistently reports improvements over time, and we proposed that participants tested at the chronic time point would show improvements in mood, decreases in subjective reporting of

cognitive and behavioral symptoms, and improvements in their EF scores. This hypothesis was not supported. Overall, participants in our mTBI group experienced no change in scores over time for any of our relevant measures. In Hypothesis 3b we expected that in mTBI participants, reductions in subjective cognitive symptom complaint reporting between the sub-acute and chronic time points would be moderated by mood. Further, we expected ethnicity would account for some of the variance in this relationship. Our analyses revealed no significant change over time for the Symptom Composite PC. Further we found no significant effects for ethnicity.

TOPF

Though not specified in our hypotheses the Test of Premorbid Functioning (TOPF) was employed as a covariate in our analyses and revealed interesting results. First, mTBI participants performed significantly worse on the TOPF compared to mTBI patients despite matching for education. Our results indicated for the mood and symptom variables a significant time by TOPF interaction. It appears that premorbid intellectual ability was differently related to mood and symptom reporting at the two time points. At the sub-acute time point mTBI participants with higher premorbid intelligence reported experiencing more mood and cognitive symptoms. In contrast, at the chronic time point, mTBI participants with lower premorbid intelligence scores reported more mood and cognitive symptoms. With regard to the measures of the NIH-EXAMINER, between-subjects analyses revealed that individuals with higher premorbid intelligence received higher EF scores and individuals with lower premorbid intelligence received lower EF scores. This was true for the Executive Composite as well as the Cognitive Control, Fluency, and Working Memory Factors.

Effect Sizes

Our study revealed small effect sizes for group on the Executive Composite (d = .14), Fluency Factor (d = .22), Cognitive Control Factor (d = .20), and Working Memory Factor (d = .19) of the NIH-EXAMINER. While small, these effect sizes are consistent with other reported effect sizes in the literature for EF deficits following mTBI. A meta-analysis by Schretlen and Shapiro (2003) examined effect sizes of studies reporting on patients with mTBIs and patients with moderate to severe TBIs. Based on their review of 15 studies, the overall neuropsychological effect size (Cohen's d) for mTBI studies of cognitive effects was d = .24, and the effect size for moderate to severe TBIs was d = .74. Further, examining time since injury intervals, their analyses revealed smaller effect sizes as the time since injury increased. Injuries assessed less than seven days after the mTBI revealed significantly higher effect sizes (d = .41), compared to injuries assessed 7-29 days post injury (d = .29), which were significantly different than assessment conducted 30-89 days post injury (d = .08). The literature supports the model that for mTBIs, patients do not experience significant EF deficits three months post injury. And further, EF deficits in more severely affected patients diminish as a function of time, with more remote injuries being less problematic compared to more acute injuries. Belanger and colleagues (2005) in their meta-analysis of mTBI patients report similar small effect sizes for the EF domain (d = .21) within the first three months and no significant effect sizes after 90 days.

For individuals who sustain a mTBI, meta-analyses typically report overall mild neuropsychological impairment across domains within the first 90 days post injury. The largest effects of cognitive deficits are usually found in the domains of fluency and

delayed memory in the acute period. At three months follow up, these effects often fall to essentially zero (Belanger, Curtiss, Demery, Leibowitz, & Vanderploeg, 2005; Schretlen and Shapiro, 2003). Another recent meta-analysis (Karr, Areshenkoff, & Garcia-Barrera, 2014) examined neuropsychological outcomes of mTBI and found staggering variability in effect sizes for cognitive domains, including EF. It is important to note that studies and meta-analyses are often inconsistent in operationally defining each neuropsychological domain. For example, the meta-analysis by Belanger and colleagues (2005) defined fluency to encompass both verbal and nonverbal fluency whereas we assessed only verbal fluency. Additionally, Belanger & Vanderploeg (2005) included fluency tasks as EF measures and yet Belanger, Curtiss, Demery, Lebowitz, and Vanderploeg (2005) defined fluency as a separate construct.

Consistent with the literature, we would expect to see larger deficits in EF acutely with the expectation patients would recover and EF deficits would dissipate over time. Our study not only found small effect sizes for EF deficits in the mTBI population, our analyses also revealed mTBI and control participants did not differ significantly in EF performance, at least with the power available in this study. mTBI patients performed just as well as control participants on our measure of EF, the NIH-EXAMINER. Furthermore, mTBI patients showed no significant improvement in EF functioning between two weeks post injury and two months post injury. On objective EF testing mTBI patients showed no significant deficits compared with healthy controls. And yet, mTBI patients subjectively report experiencing cognitive changes including EF deficits that impact their ability to function successfully in daily life. Executive functions comprise higher-order cognitive abilities such as cognitive flexibility, problem solving, concept formation, self-

regulation, working memory, and attention (Raymar, Roitsch, Redman, Michalek, & Johnson, 2018). These skills are essential for goal-directed behavior and for responding to novel stimuli in an individual's environment. Reported subjective deficits in EF are among the enduring cognitive problems reported following a mTBI. These difficulties, whether perceived or objectively assessed in testing, can interfere with an individual's ability to complete instrumental activities of daily living as well as actively participate in cognitive and physical rehabilitation (Raymar et al., 2018). mTBI is often characterized by subjective complaints of cognitive impairment in the absence of objective neuropsychological findings that persist past the first few weeks post injury (Karr et al., 2014). mTBI survivors frequently report impairments in concentration, memory, and attention, although objective neuropsychological assessment beyond the first couple of weeks indicates performance in the normal range. These cognitive symptoms are generally transient for most individuals but persist in a small subset (Karr et al. 2014). In contrast, patients with moderate and severe TBIs may lack insight and be unaware of their cognitive deficits. On objective assessment they usually show impaired performance in one or more cognitive testing domain (Eshel, Bowels & Ray, 2019).

The Construct of EF

In interpreting the preceding results, it is necessary to consider the question, do performance-based measures and self-report ratings of EF assess the same construct? In this study, one performance-based measure (NIH-EXAMINER) and several rating measures were used (FrSBe, NSI, PROMIS) in an attempt to assess EF performance both objectively and subjectively. While this was one way to evaluate study participant's EF skills, we must turn to the literature to review the best way to operationalize and measure

EF. Performance-based measures involve the use of highly standardized procedures that are administered by an examiner to an examinee and the measures typically assess the accuracy and/or response time. The stimulus presentation is precisely controlled so each examinee completes the task in the same way as all other examinees (Toplak, West, & Stanovich, 2013). In contrast, rating scales and measures of EF involve either the individual or an informant reporting on perceived difficulties. Importantly, a central supposition underlying EF rating measures is that they are measuring behaviors that are related to the processes that performance-based measures are assessing. Therefore, if performance-based measures and rating measures of EF are measuring the same construct, then these measures should be strongly positively correlated. A recent meta-analysis by Toplak, West, and Stanovich (2013) examined 20 studies in the child literature to address this question. Overall, they found 24% of the correlational comparisons between performance-based measures and rating measures of EF were statistically significant and of those, the magnitude of the correlations were typically low. Across all studies, the median correlation was .19. The authors note that if both types of measures were measuring the same construct, then convergent validity should be present. A basic principle of convergent validity is that the operational measures of the same construct should be highly correlated, this is not the case in this study (Toplak, West, & Stanovich, 2013). While both types of assessment are valuable, it cannot be assumed that they are measuring the same construct and should not be used interchangeably. Therefore, it appears these measures are more related to mood than objectively assessed EF cognition.

NIH-EXAMINER Limitations

As previously reported, our study revealed significant differences between mTBI and control participants at the sub-acute time point for mood and subjective symptom reporting but not for the Executive Composite or the Fluency, Cognitive Control, and Working Memory factors of the NIH-EXAMINER. Another possible explanation for the lack of difference between mTBI and healthy control participants scores on EF measures is the test itself.

In an effort to make the NIH-EXAMINER tasks shorter and easier to administer to larger populations, one could argue that the authors compromised the integrity of the individual measures. One such example of this is the continuous performance task (CPT) subtest used in the NIH-EXAMINER. This measure aims to assess a participants sustained and selective attention and is typically used in assisting in the diagnosis of ADHD. Since the first version of the CPT was developed (Rosvold et al., 1956), the test has been modified several times and two of the most commonly used versions of the CPT are the Integrated the Test of Variables of Attention (T.O.V.A.) (Greenberg & Waldmant, 1993) and the Conners' CPT-II (Conners, 2000). The T.O.V.A is approximately 21.6 minutes long for adults (Greenberg & Waldmant, 1993) and the CPT-II has a duration of 14 minutes and is made up of six blocks that are split into three sub blocks (Homack & Riccio, 2006). In contrast, the NIH-EXAMINER CPT consists of 100 trials that are divided into four blocks of 25 trials but does not provide the overall task duration for the CPT (Kramer, 2001). Furthermore, on the website, the authors assert the CPT used on the NIH-EXAMINER is not the Conners' CPT-II. Instead the authors state that CPT is a custom task they designed specifically for this battery and it is based on pilot testing, but

they do not provide information on the pilot study or norms for this new CPT version (EXAMINER FAQ, 2019). This pattern of shortening sub-test measures is seen in several additional parts of the NIH-EXAMINER. Specifically, the dot-counting, flanker task, and set-shifting task have been shortened. Per the manual, to reduce time of administration and patient burden the trials of the dot counting task were reduced by half, from twelve trials to six trials. The flanker task trials were reduced from 64 to 48 and the set shifting subtest trials were reduced from 120 to 104 (Kramer, 2001). This might affect reliability less than validity, if TBI deficits are somewhat episodic

One of the core features or strengths of the CPT is the long duration an examinee has to sustain attention. This results in increased fatigue, assessment of distress tolerance, less opportunities for loss of concentration if they have attentional difficulties. One could argue that shortening the CPT may result in the test not measuring the construct it is intending to measure.

In the NIH-EXAMINER, the Fluency Factor is made up of phonemic and category fluency. These measures appear to be, for the most part, unchanged from traditional neuropsychological measures of verbal fluency including the Controlled Oral Word Association Test (COWAT) (Benton, Hamsher, & Sivan, 1994) and the F-A-S Test (Spreen & Benton, 1997). In the standard versions of the tasks, the examinee is given one minute to produce as many unique words as possible starting with a given letter (letter fluency) or within a semantic category (category fluency). The participant's score in each task is the number of unique correct words. Verbal fluency tasks are often conceptualized and used in research and clinical practice as a measure of EF. However, research has begun to consider the language component in these tasks. Successful performance on the

verbal fluency task has been cited in some studies as a complex interplay of both EF and language (Unsworth, Spillers, & Brewer, 2011). Whiteside and colleagues (2015) examined the underlying cognitive structure of verbal fluency using EF and language measures via exploratory factor analyses. Their results indicated a two-factor structure of language and EF. Surprisingly, FAS and Animal fluency loaded exclusively on the language factor (Whiteside et al., 2015). In our study, the Fluency Factor was revealed to not significantly differ between healthy controls and mTBI patients two weeks post injury one explanation for this is that we only tested verbal fluency and did not include measures of non-verbal fluency. It may be that acutely following a mTBI non-verbal fluency is impacted while verbal fluency is not. The effect size estimate from our verbal fluency measure was d = .22. while Belanger and colleagues meta-analysis (2005) fluency measure (verbal and non-verbal) had an effect size of d = .89.

While the authors provide plausible justification for their decision to remove trials in the manual (Kramer, 2001), this raises the question of norms as each altered sub test requires new norms to validate their claims. If the shortened versions of the test were substantially validated with normative data, the test designers would have stronger support of the validity of the NIH-EXAMINER. Thus, it is possible that the NIH-EXAMINER slightly underestimated the true extent of any cognitive deficit.

Normative Data

In neuropsychological testing, the conventional method used to identify abnormal cognitive performance is by comparing an individual's cognitive performance with that of a reference or normative group studied at a single time point (Kendall, Marrs-Garcia, Nath, & Sheldrick, 1999). The normative data often takes into account variables such as

an individual's age, gender, ethnicity, and level of education (Merritt et al., 2016). Normative data helps clinicians and researchers place the individual into a sociocultural context. A patient may feel they performed poorly on a task but upon comparison to the normative data sample the patient may instead present in the average range. The reverse scenario is also possible. Normative data is vital is assessing cognitive performance declines by placing performance into a context for accurate interpretation. The website for the NIH-EXAMINER specifies they lack normative data for individual subtest or composite scores for their assessment battery. The test designers acknowledge this is problematic and hope to assemble age or age and education based normative data, but as of this time this has not been done. Of note, they do offer the option for providers to contact them via email to have access to their data same of 600 controls organized by age (EXAMINER FAQ, 2019). While this is a nice gesture it is nonetheless problematic for clinicians. Not having normative data is not an issue for research studies with a health control group but it does pose problems for clinical applications. It is preferable to have an assessment test that can be used in both in a clinical and research setting.

A second essential component of normative data in neuropsychological assessment is relevant norms based on patient population (Merritt et al., 2016). Normative data at a minimum usually includes age and gender but unfortunately it often lacks satisfactory race and ethnicity norms. These norms are useful for assessment in the general population but sub samples of patient groups that more closely resemble the characteristics and demographics of the patient population in question are significantly more valuable (Merritt et al., 2016). The NIH-EXAMINER manual specifies the battery was tested on sample of 19 people aged 18 to 50 years, who sustained a TBI ranging in

severity from moderate to severe. All participants in this group had sustained their injury at least 6-months prior to testing (Kramer, 2001). This patient population stands in stark contrast to the participants in this study who all sustained mTBIs and were tested with this battery within two weeks of their injury.

The NIH-EXAMINER not only lacks patient population specific norms it also lacks even the most basic age, gender, race, ethnicity, and years of education normative data. This is again problematic for the clinical use of this instrument. A review of the literature indicates that researchers have been utilizing this measure across several medical and mental health populations to test its reliability and validity (Krueger et al., 2009; You et al., 2013; Possin, LaMarre, Wood, Mungas, & Kramer, 2014). Our study utilized a sample of 52 mTBI patients and found small effect size estimates that are comparable to the small EF effect sizes reported in the literature for mTBI patients.

Taken together, it appears the NIH-EXAMINER may be an adequate measure of EF even though the effect sizes are small. A possible explanation of this result is that mTBI patients are actually not experiencing EF deficits. mTBI patients, in contrast to those who sustain moderate or severe TBIs, on objective testing do not appear to have EF deficits. It may be that there is something inherent in the mTBI population wherein they report subjective cognitive complaints despite not evidence of objective impairment. This finding has implications for treatment.

Treatment

Overall, results of our study revealed mTBI patients experienced large effects for depressed mood and only small effects for EF impairments. We propose one of the most important and often overlooked components in the treatment of patients with mTBI is in

treating the functional impact these impairments have on the individual. Our study found no change over time for negative mood complaints and subjective symptom reporting of cognitive complaints. Patients in our study were just as depressed at the chronic time and had the same cognitive complaints as they did two-week post injury. This finding, which falls contrary to expected mTBI recovery trajectories, is particularly troubling and has implications for treatment.

As stated previously, while most individuals who sustain a mTBI typically recover completely within the a few weeks or months of their injury, a significant minority continue to experience persistent negative symptoms months to years later (Vanderploeg, Curtiss, Luis, & Salazar, 2007). This begs the question of what treatment is best suited for this population and when is the best time to administer intervention for best outcomes. One avenue for intervention that has been investigated is the question of whether psychoeducation and supportive interventions in the acute phase can prevent the progression of symptoms to persistent PCS. These interventions are based on the theory that persistent PCS are related to the misattribution of symptoms to the mTBI and the patient's negative expectations about recovery (Belanger, Barwick, Kip, Kretzmer, & Vanderploeg, 2013). Given that negative perceptions of mTBI are one of the best predictors of PCS at six months post-injury, this is an ideal treatment target (Hou et al., 2012).

Psychoeducation and supportive interventions typically involve education on post-concussive symptoms, guidance on rest and gradual reintegration of typical activities, education on coping strategies, and reassurance on the expectation of a complete recovery (Prince and Bruhns, 2017). Of all post-mTBI interventions,

psychoeducational and supportive interventions have the strongest empirical evidence (Comper, Bisschop, Carnide, & Tricco, 2005). Even simple education and support about mTBI symptom sequelae and expected recovery provided shortly after injury has been shown to results in a reduction in somatic and psychological complaints (Comper et al., 2005). Furthermore, a single session early intervention can prevent PCS as effectively as traditional outpatient therapy (Mittenberg, Canyock, Condit, & Patton, 2001).

Treatment of cognitive dysfunction following mTBI should be functionally oriented and should relate directly to the real-life context the individual exists within as much as possible. The two types of treatment for cognitive dysfunction are normally characterized as compensatory or restorative. A compensatory approach tries to provide internal mental strategies such as mnemonics or external aides such as notebooks to help with patient's daily activities despite the presence of a cognitive impairment. On the other hand, restorative approaches attempt to improve the overall performance of a cognitive system with the ultimate goal of improving performance of actives that depend on the functioning of a particular system (Eshel, Bowels, & Ray, 2018). A particular strength of mTBI patients in comparison to patients with moderate or severe TBIs is they typically have greater insight which may result in a better understanding of the injury and treatment process.

Another avenue of treatment for persistent post-concussion symptoms following a mTBI is the treatment of depression or depressive symptoms using empirically supported therapies. Our study results revealed that mTBI patients experienced more symptoms of depression than healthy controls and more symptoms two weeks post injury and 2 months post injury. While rates of depression in the general population range from 8-11%

(Guillamondegui et al., 2011), rates of depression in mTBI patients range from 10-77% (Silver, McAllister, & Arciniegas, 2009). Depression appears to be a clear treatment target. Looking at the depression literature, perceived cognitive impairment is a hallmark symptom of depression (Channon & Green, 1999; Zakzains, Leach & Kaplan, 1998). Treatment of depression following mTBI may alleviate other post-concussive symptoms such as cognitive impairment in addition to improving mood (Silver, McAllister, & Arciniegas, 2009). In our study effect size estimates for depressed mood were many times larger (d = .90) than effect size estimates of EF impairments (d = .14), thus making depression a prime treatment target.

Limitations

There were several major limitations to this current study. First, we experienced practical difficulties in participant attendance and attrition. While mTBI patients were approached immediately post injury they varied in the time between when they sustained the injury and when they completed the first session. The majority of patients, 84.5% did not attend the first study session until between 11- and 14-days post injury. There is the possibility that patients assessed more closely to the injury date would have shown change over time in self-reported mood and symptoms and possibly significant differences in EF performance. Given the small sample size of participants who came in between one- and seven-days post injury, we were unable to assess if these participants experienced a change in scores over time compared to more remotely assessed patients. One could hypothesize that the population of mTBI patients who participated in the study at the first and second time points were a subset of the least impaired individuals who had sustained an mTBI. These individuals presumably had access to transportation and were

functioning (i.e., in terms of pain complaints) well enough post-injury to participate in our study.

Another possible limitation with this study is participant employment status. While more mTBI patients than control patients were unemployed at the time of the first assessment, a large proportion 18.7% of control participants were unemployed (looking for work, temporarily laid off, or disabled) at the time of the first assessment. Per the literature, for those who are employed preinjury, unemployment rates at one-year post-injury have been found to be 31% among persons with mTBI (Doctor et al., 2005). The cognitive and behavioral sequelae that occur following a mTBI can interfere with returning to or gaining employment even in patients who were employed prior to their injury. Employment is an important measure of social functioning that has been shown to positively influence a variety of areas in an individual's life including: less health service usage, findings of better health status in people who are employed, a better sense of wellbeing, greater social integration within the community, more social contacts, and a better QOL than people who are not employed (van Velzen, van Bennekom, Edelaar, Sluiter, & Frings-Dresen, 2009). The high rate of healthy control participants who were unemployed at the time of the first session may have been a factor in mood and symptom reporting, resulting in higher than expected control scores.

An additional limitation is our examination of socio-economic status (SES). Given the complexity in selecting variables for SES we choose to use the dichotomized variable of ethnicity, and the TOPF as a measure of SES as the TOPF correlated strongly with education. In this study the mTBI and control groups did not differ significantly in terms of sex, age, race, ethnicity, years of education, and marital status. We did not

examine additional SES variables in our analyses which is a limitation to this study. Due to small sample sizes for our race and other potential SES variables, we were unable to fully examine the role of race and other SES factors on mood, subjective symptom reporting, and EF. While the participants in this study were all from the Albuquerque, New Mexico area, this study could benefit from neighborhood analysis to assess if some of the variance in our results could be accounted for by what neighborhood an individual grew up in.

The self-report and performance-based measures utilized in our study also present several limitations. Measures of mood were limited to one measure, the BDI-II. The BDI-II, while well validated in the literature (Segal, Coolidge, Cahill, & O'Riley, 2008) was not expressly designed to assess depression symptomatology in a TBI or mTBI population. As well, our measure of mood was actually a more specific measure of depression. The term mood encompasses several symptom presentations not just depression. For example, this study could have benefited from employing multiple measures of mood including measures of anxiety. mTBI patients typically report experiencing significant anxiety symptoms following a mTBI compared to healthy controls (Wood, O'Hagan, Williams, McCabe, & Chadwick, 2014). Multiple mood measures would have allowed for more in-depth analyses of the post-concussion symptom profile.

Our measure of subjective cognitive symptom reporting was developed using a principal components analysis of the variables that were available in this secondary analysis. While the principal component accounted for over 75% of the variance of symptom measures it also correlated highly with the BDI-II. A review of the NSI and

FrSBe items indicated significant overlap of mood question with the BDI-II. Given the limitations of the measures used for symptom reporting we were unable to clearly separate a pure mood and pure symptom variable.

The performance-based EF measure had several limitations. The central neuropsychological assessment measure utilized in this study, the NIH-EXAMINER assessment battery, was the only means of assessing EF. As previously discussed, this battery, had several potential drawbacks. First, the NIH-EXAMINER is a relatively new assessment battery that has not be adequately normed for clinical use with TBI or mTBI populations. This is not an issue for our research study that contained healthy controls but does pose limitations for clinical implications. Further, several sub-tests of the measure were shortened by the test designers with the aim of reducing time and test taking burden. Unfortunately, these changes, namely removing several trials and reducing the test taking time of the CPT by over half, may have altered the sub-tests and as a result could explain why our effect size for the Fluency Factor differed from literature reports.

Summary, Clinical Implications, and Conclusions

Although the main aim of the study, which was to determine if mood moderated the relationship between subjective cognitive complaints and objective EF deficits, was not supported by the current data, other valuable, preliminary findings arose that have important clinical implications. Our results revealed abundant evidence of mood dysfunction following mTBI that did not dissipate over time as the literature would suggest. We argue that research into the evaluation and treatment of post-concussion symptoms has focused almost exclusively on cognitive impairments at the expense of investigating mood problems. Clinical psychologists are well versed in a plethora of

empirically supported therapies for mood dysfunction, including Acceptance and Commitment Therapy for Depression, Behavioral Activation for Depression, and Cognitive Therapy for Depression. Psychologists have at their disposal several well validated treatments that may be directly applicable in the treatment of patients with post-concussion symptoms. Further our study revealed a small effect size for EF deficits (d = .14) that would not preclude the necessary EF skills to participate effectively in psychotherapy intervention.

A person's perception of reality is in turn their own reality. It is essential that mental health practitioners take patient's subjective reporting of symptoms seriously. These complaints should be respected comparable to clinician's perceptions of objective neuropsychological assessment. Even though performance-based and self-report assessments may measure different constructs of EF, dismissing these complaints is not only harmful to the patient, it also dismisses vital information that can have implications for rehabilitation. For example, a mTBI participant may report poor mood, fatigue, and apathy. One could propose that these symptoms may directly impact their ability to effectively participate in rehabilitation and further may be a barrier to care. Treatment of mood symptoms may increase patient engagement in vital rehabilitation services. Psychologists have a vital role to play in the treatment of post-concussion symptoms.

In conclusion, this study improves our understanding of the relationship between mood symptoms, subjective cognitive symptom complaints, and objective assessment of EF deficits. Focusing exclusively on subjective and objective cognitive deficits at the expense of mood deficits does a disservice to our patients. Psychologist have a unique

opportunity to be at the forefront of mTBI mood treatment to improve quality of life in a significant population of patients who continue to suffer with post-concussion symptoms.

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Appendix A: Figures and Tables

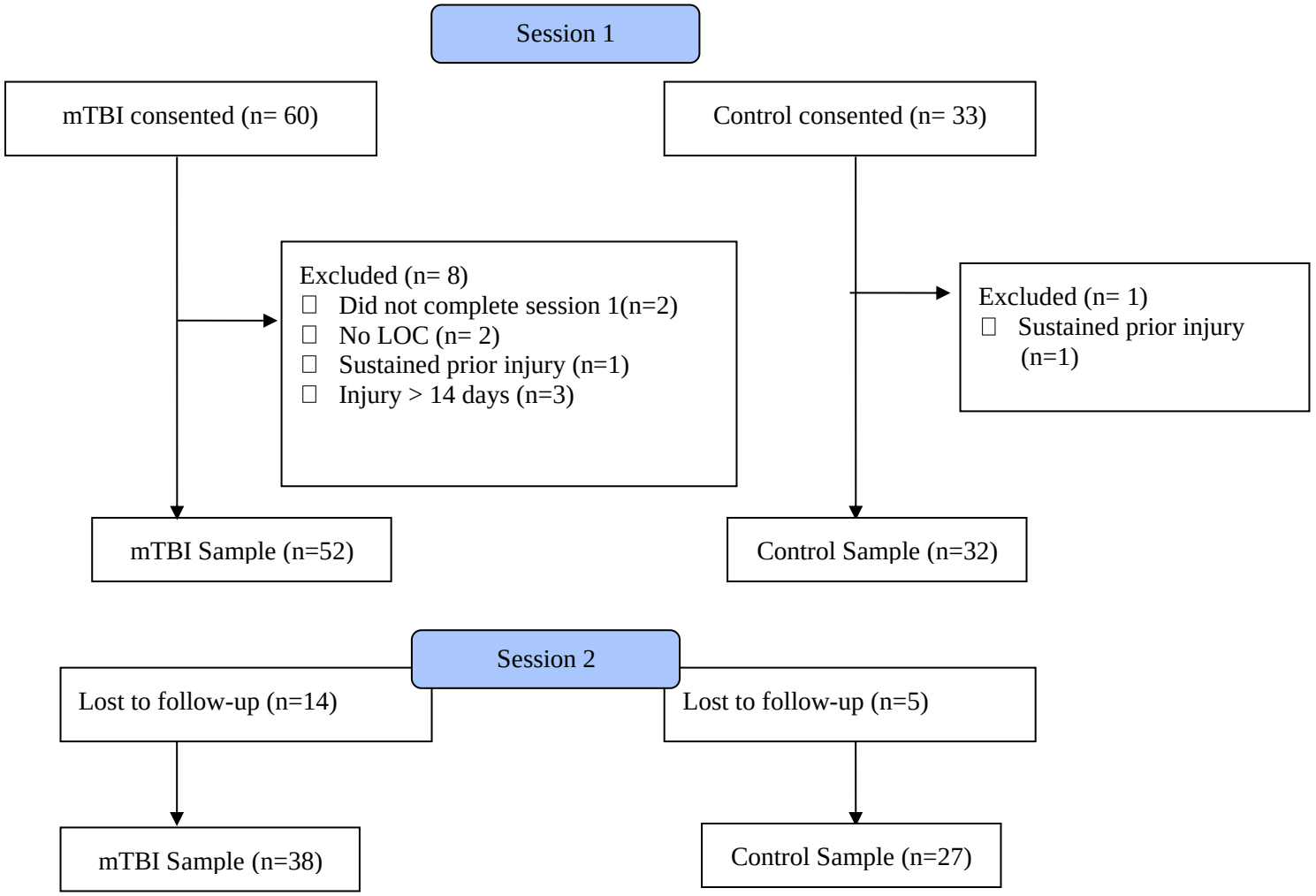

Figure 1. Consort diagram of study participants with attrition.

Table 1.

Demographics of individuals with a mTBI and Control participants

Variables		mTBI (n = 52) n (%)	Control (n=32) n (%)	Total n (%)	Significance Test
Sex					p = .224
	Male	33 (63.5)	16 (50.0)	49 (57.0)	
	Female	19 (36.5)	16 (15.0)	35 (40.7)	
Age (years)[a]		29.9(10.2)	29.6 (10.6)	29.2 (10.3)	p = .787
					p = .111
Race					
	Black	1 (1.9)	2 (6.3)	3 (3.5)	
	Asian	0 (0)	1 (3.1)	1 (1.2)	
	White	30 (57.7)	24 (75.0)	54 (62.8)	
	Native American	8 (15.4)	2 (6.3)	10 (11.6)	
	Other	13(25.0)	3 (9.4)	16 (18.6)	
					p = .161
Ethnicity					
	Non-Hispanic White	26 (50.0)	21 (65.6)	47 (54.7)	
	Hispanic or Latino	26 (50.0)	11 (34.4)	37 (43.0)	
Education					p =.204
	Less than High School	7 (13.5)	1 (13.1)	8 (9.5)	
	High School or GED	10 (19.2)	4 (12.5)	14 (16.3)	
	More than High School	35 (67.4)	27 (84.4)	62 (72.1)	
Marital Status					p = .218
	Never Married	39 (75.0)	18 (56.3)	57 (66.3)	
	Married	4 (7.7)	8 (25.0)	12 (14.0)	
	Domestic Partnership	4 (7.7)	4 (12.5)	8 (9.3)	
	Divorced	2 (3.8)	1 (3.1)	3 (3.5)	
	Separated	1 (1.9)	1 (3.1)	2 (2.3)	
	Widowed	2 (3.8)	0 (0)	2 (2.3)	

[a]Mean(SD)

Note: Significance testing χ^2 or t-test

Table 2.

Employment Variables

Variables		mTBI (n = 52) n (%)	Control (n = 32) n (%)	Total n (%)
Employment Status				
	Working now	31 (59.6)	20 (62.5)	51 (59.3)
	Only temporarily laid off, sick leave, maternity leave	3 (5.8)	1 (3.1)	4 (4.7)
	Looking for work, unemployed	6 (11.5)	4 (12.5)	10 (11.6)
	Retired	1 (1.9)	0 (0)	1 (1.2)
	Disabled, permanently or temporarily	3 (5.8)	1 (3.1)	4 (4.7)
	Keeping House	0 (0)	1 (3.1)	1 (1.2)
	Student	4 (7.7)	3 (9.4)	7 (8.1)
	Other	4 (7.7)	2 (6.2)	26 (7.0)
Job Classification				
	None	14 (26.9)	3 (9.4)	17 (19.8)
	Official/Manager	2 (3.8)	0 (0)	2 (2.3)
	Professional	7 (13.5)	8 (25.0)	15 (17.4)
	Technical	6 (11.5)	5 (15.6)	11 (12.8)
	Sales Worker	8 (15.4)	3 (9.4)	11 (12.8)
	Craft Worker	0 (0)	3 (9.4)	3 (3.5)
	Operative	2 (3.8)	0 (0)	2 (2.3)
	Administrative Support	1 (1.9)	0 (0)	1 (1.2)
	Laborer/Helper	6 (11.5)	6 (18.8)	12 (14.0)
	Service Worker	5 (9.6)	3 (9.4)	8 (9.3)
	Unknown	1 (1.9)	1 (3.1)	2 (2.3)

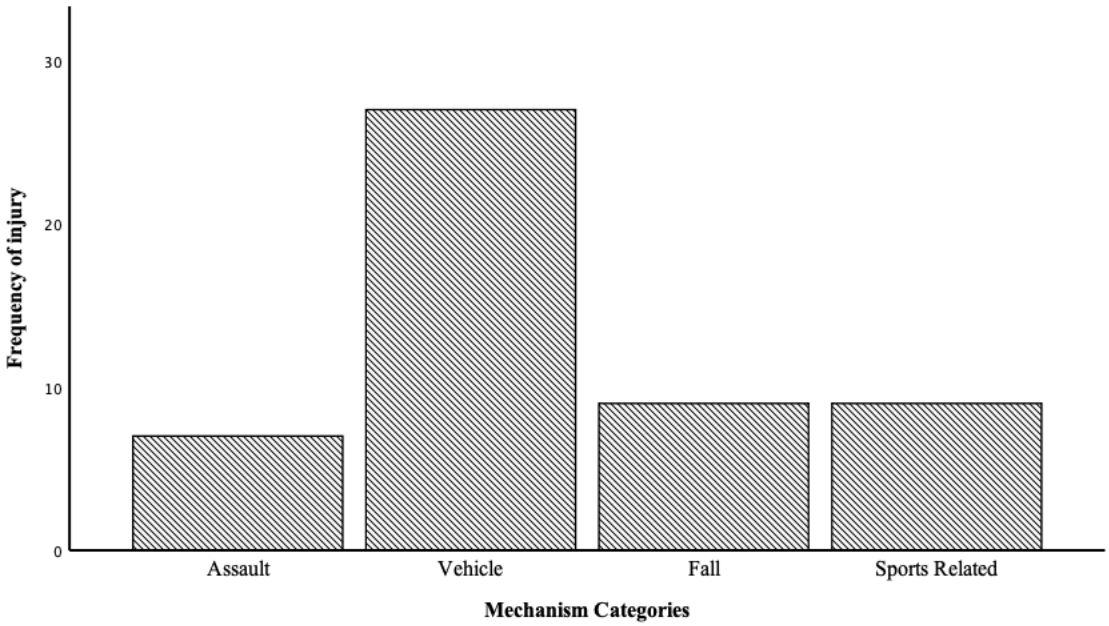

Figure 2. mTBI reported mechanism of injury

Table 3.

Pearson Correlations between BDI-II, pain, and sleep measures

	BDI-II[a]	Pain Interference[a]	Pain Intensity[a]
BDI-II			
Pain Interference	.589*		
Pain Intensity	.457*	.879*	
Sleep Disturbance[b]	.553*	.552*	.553*

[a]n = 83, [b]n = 84

*. Correlation is significant at the 0.01 level (2-tailed).

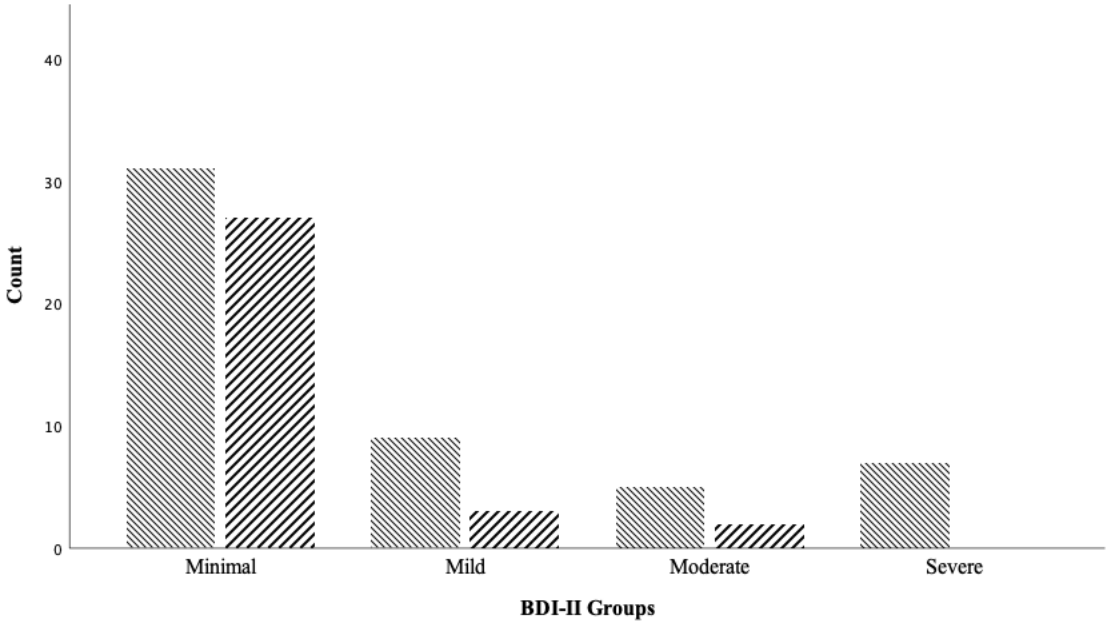

Figure 3. Number of individuals in each group who reported experiencing minimal, mild, moderate and severe depression symptoms on the BDI-II at the sub-acute time point. = mTBI, = Control.

Table 4.

Pearson Correlations between symptom, sleep, pain, and executive measures in the mTBI group at the sub-acute time point

	NSI Somatic[a]	NSI Cognitive[a]	NSI Emotion[a]	FrSBe[a]	Sleep Disturbance[b]	Pain Interference[b]	Pain Intensity[b]
NSI Somatic							
NSI Cognitive	.82**						
NSI Emotion	.80**	.85**					
FrSBe	.46**	.55**	.57**				
Sleep Disturbance	.34*	.37**	.54**	.32*			
Pain Interference	.57**	.54**	.68**	.57**	.44**		
Pain Intensity	.52**	.35*	.52**	.36**	.41**	.79**	
Executive Composite[c]	.09	.15	.07	.13	-.20	-.05	-.15

Note: NSI Somatic = Neurobehavioral Symptom Inventory Somatic sub-scale; NSI Cognitive = Neurobehavioral Symptom Inventory Cognitive sub-scale; NSI Emotion = Neurobehavioral Symptom Inventory Emotion sub-scale; FrSBe = Frontal Systems Behavior Scale.

[a]n = 52, [b]n = 51, [c]n=47

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Table 5.

Mechanism of Injury by Gender

	Female	Male	Total	Chi-Square
N	18	34	52	.164
Assault	2	5	7	
Vehicle	8	19	27	
Fall	6	3	9	
Sports Related	2	7	8	

Table 6.

Assessment variables reported mean, standard deviation and t-test significance between both groups at the sub-acute time point

		mTBI	Control		
	n	Mean (SD)	n	Mean (SD)	t-Test
TOPF Total Std. Score	51	94.5 (14.3)	32	105 (12.7)	p = .001
BDI-II	52	13.1 (11.0)	32	5.78 (6.9)	p = .001
Symptom Composite PC	52	105.27 (15.7)	31	91.16 (8.27)	p < .001
NSI Somatic	52	12.1 (10.8)	31	2.06 (2.8)	p < .001
NSI Cognitive	52	5.13 (4.85)	31	1.29 (2.1)	p < .001
NSI Emotion	52	8.48 (6.3)	31	3.35 (4.3)	p < .001
NSI Total	52	25.8 (20.6)	31	6.71 (8.7)	p < .001
FrSBe	52	60.8 (17.1)	31	52.1 (15.5)	p = .021
Executive Composite	47	109.18 (7.89)	29	114.33 (7.14)	p = .005
Fluency Factor	47	106.15 (9.55)	29	113.19 (8.79)	p = .002
Cognitive Control Factor	47	112.37(9.68)	29	115.02 (6.73)	p = .199
Working Memory Factor	47	105.03 (.9.42)	29	107.68 (.10.91)	p = .266

Note: TOPF Total Std. Score = Test of Premorbid Functioning Total Standard Score; BDI-II = Beck Depression Inventory II; NSI Somatic = Neurobehavioral Symptom Inventory Somatic sub-scale; NSI Cognitive = Neurobehavioral Symptom Inventory Cognitive sub-scale; NSI Emotion = Neurobehavioral Symptom Inventory Emotion sub-scale; NSI Total = Neurobehavioral Symptom Inventory Total Score; FrSBe = Frontal Systems Behavior Scale; Executive Composite = Executive Composite from the NIH-EXAMINER, Fluency Factor = Fluency Factor of the NIH-EXAMINER, Cognitive Control Factor = Cognitive Control Factor of the NIH-EXAMINER; Working Memory Factor = Working Memory Factor of the NIH-EXAMINER.

Table 7.

Symptom Measure Correlations

	NSI Somatic[a]	NSI Cognitive[a]	NSI Emotion[a]	NSI Total[a]
NSI Somatic				
NSI Cognitive	.851*			
NSI Emotion	.818*	.863*		
NSI Total	.964*	.935*	.931*	
FrsBe Total[a]	.514*	.584*	.610*	.589*

[a]n = 83.

*. Correlation is significant at the 0.01 level (2-tailed).

Table 8.

Pearson Correlations of Executive Functioning Variables

	Executive Composite[a]	Fluency Factor[a]	Cognitive Control Factor[a]
Executive Composite			
Fluency Factor	.799*		
Cognitive Control Factor	.622*	.192	
Working Memory Factor[a]	.657*	.343*	.314*

[a]n = 76.

*. Correlation is significant at the 0.01 level (2-tailed).

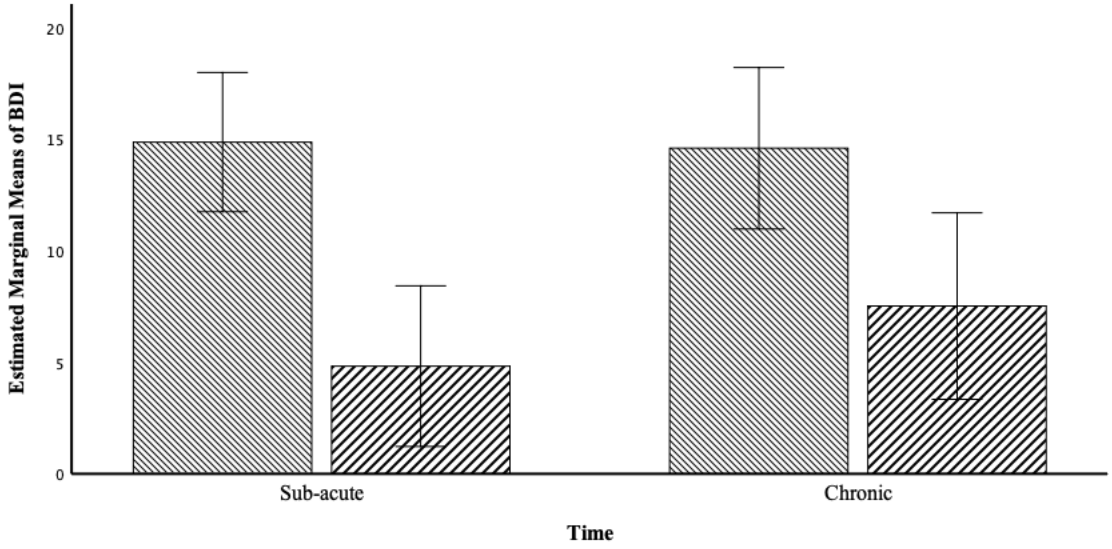

Figure 4. Mean BDI-II scores by group at the sub-acute and chronic time points. ▨ = mTBI, ▨ = Control. Error bars: +/- 2 SE.

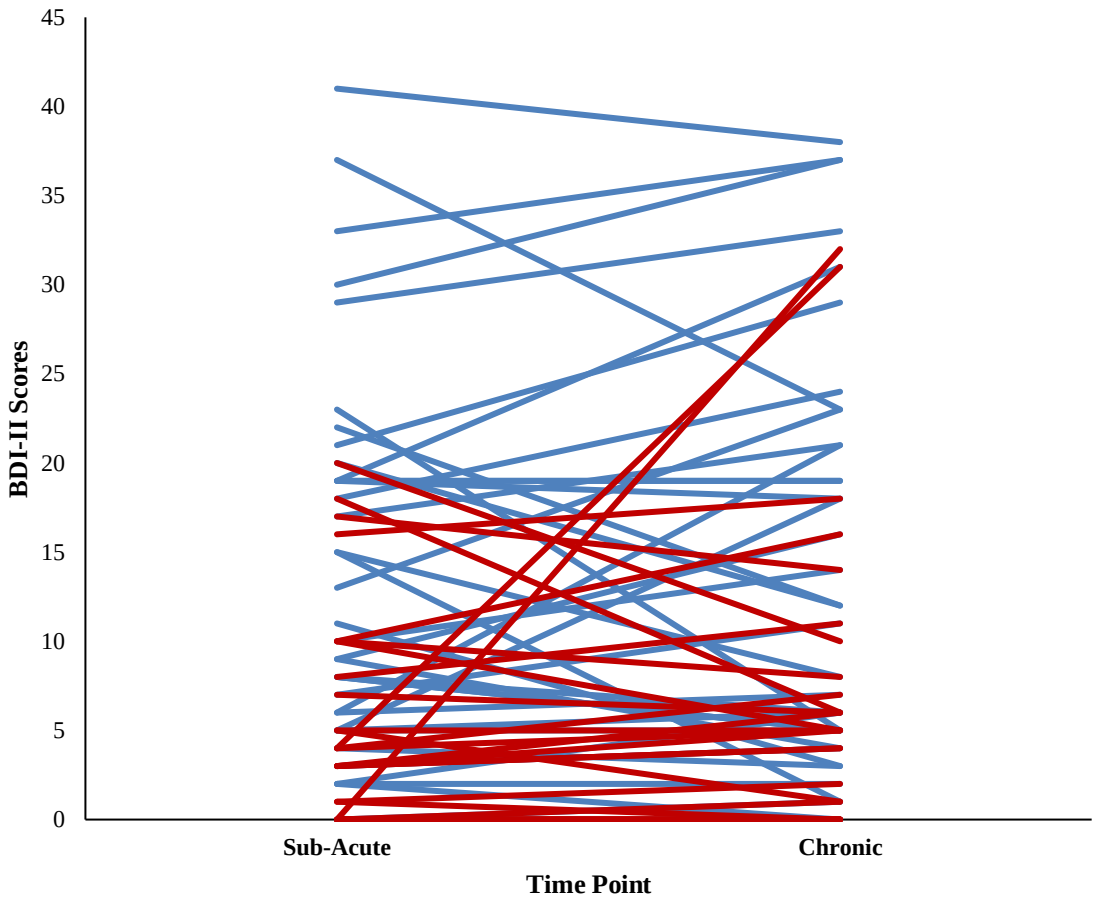

Figure 5. BDI-II scores at the sub-acute and chronic time points for mTBI and control participants = mTBI = healthy control.

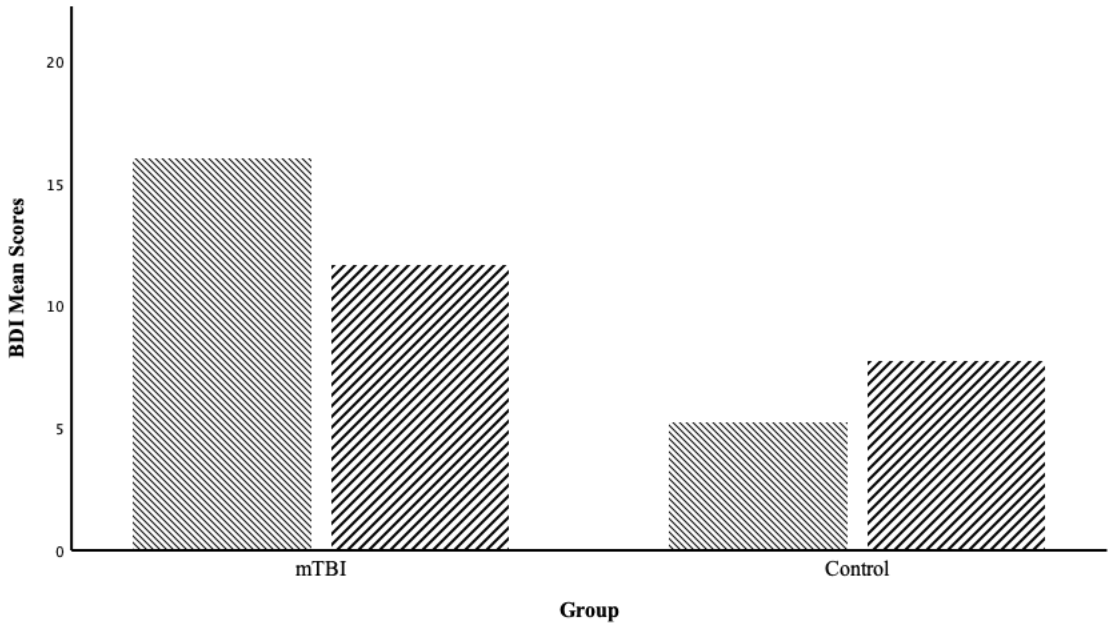

Figure 6. TOPF high and low scores by median split organized by group by BDI-II scores at the sub-acute time point = high TOPF score, = low TOPF score.

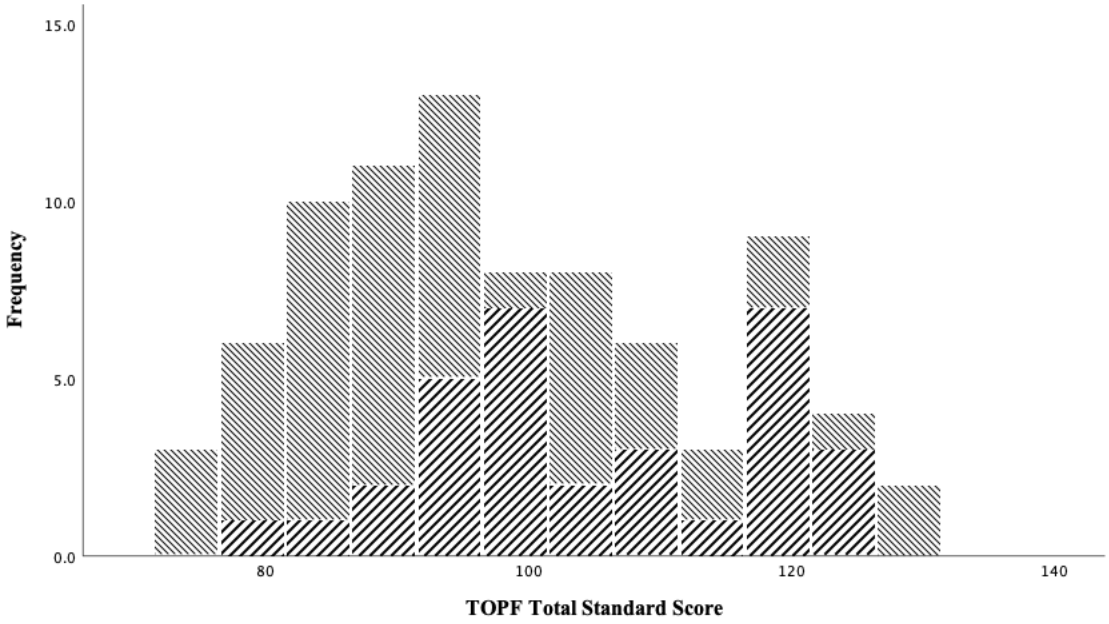

Figure 7. Histogram of TOPF Total Standard Score by frequency for each group at the chronic time point. = mTBI, = Control.

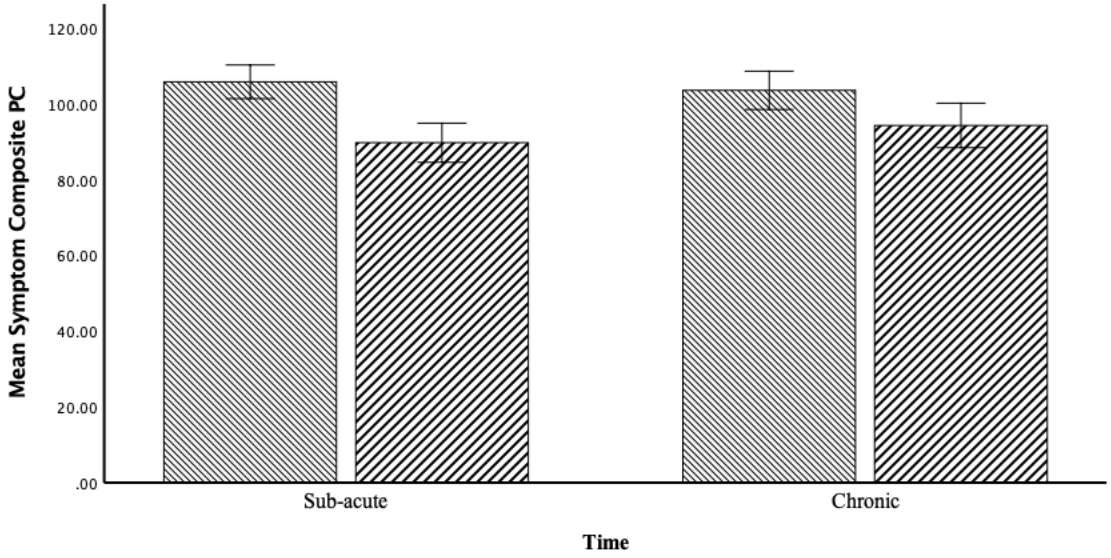

Figure 8. Mean Symptom Composite PC scores by group at the sub-acute and chronic time points. ▨ = mTBI, ▨ = Control. Error bars: +/- 2 SE.

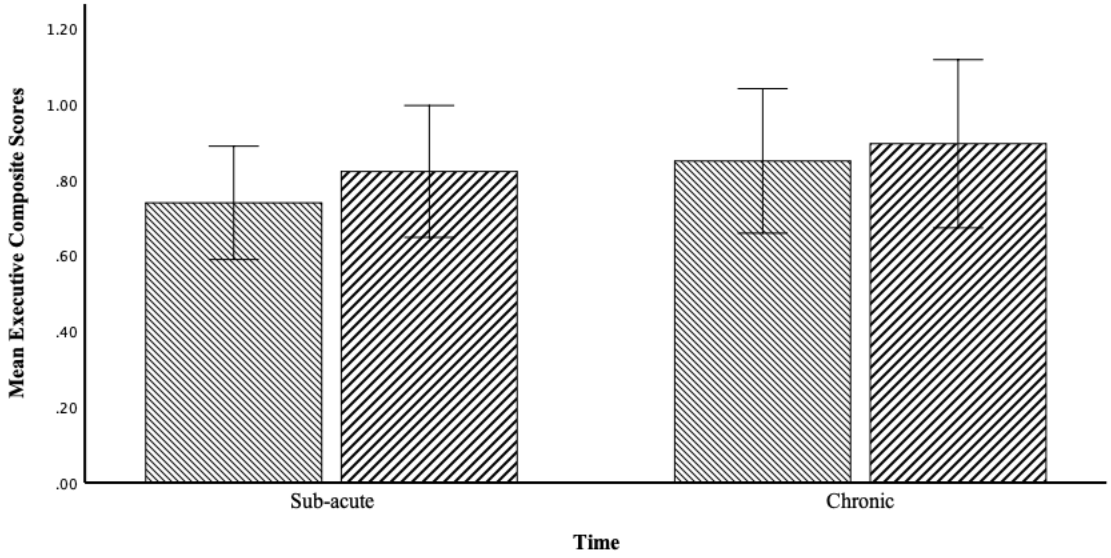

Figure 9. Mean Executive Composite scores by group at the sub-acute and chronic time points. ▨ = mTBI, ▨ = Control. Error bars: +/- 2 SE.

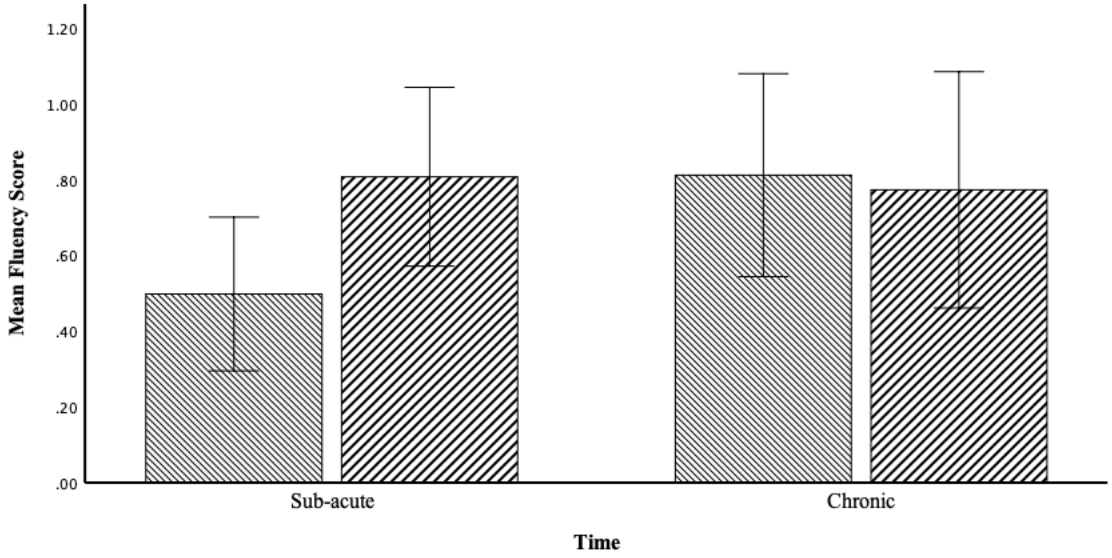

Figure 10. Mean Fluency Factor scores by group at the sub-acute and chronic time points = mTBI, = Control. Error bars: +/- 2 SE.

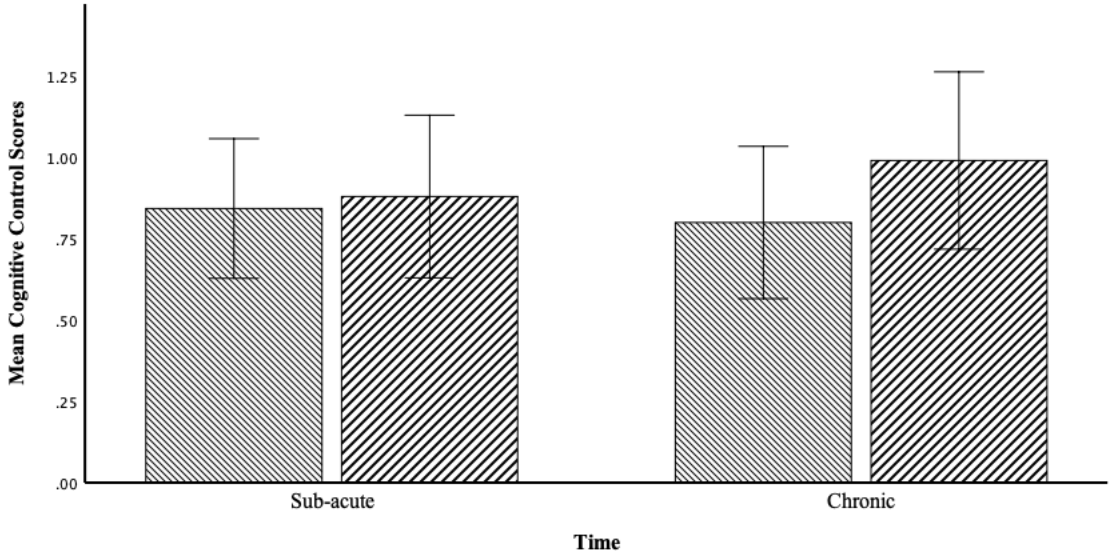

Figure 11. Mean Cognitive Control Factor scores by group at the sub-acute and chronic time points. ▨ = mTBI, ▨ = Control. Error bars: +/- 2 SE

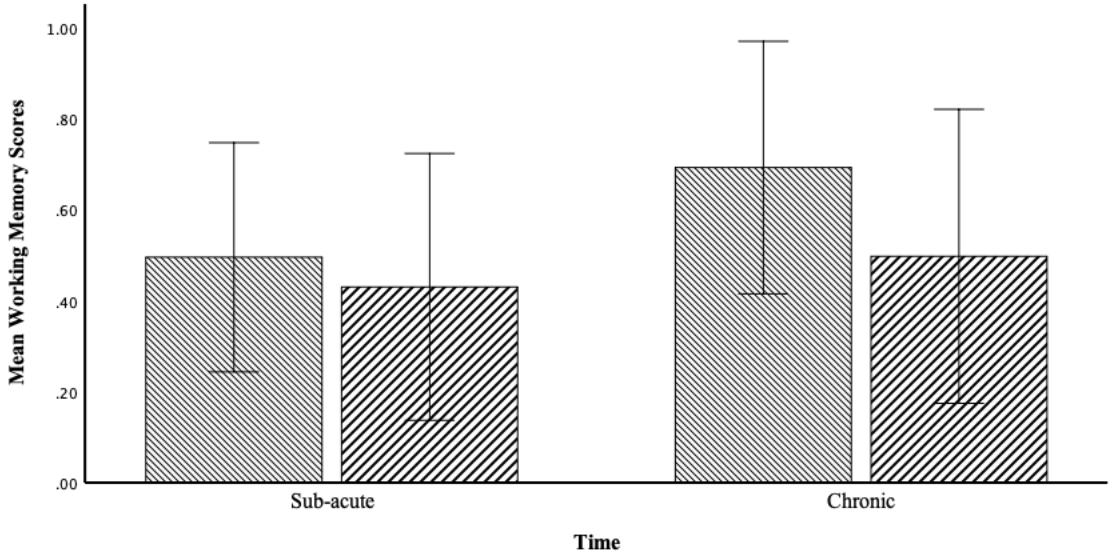

Figure 12. Mean Working Memory Factor scores by group at the sub-acute and chronic time points. ▨ = mTBI, ▨ = Control. Error bars: +/- 2 SE.