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**MILD TRAUMATIC BRAIN INJURY, EXECUTIVE FUNCTIONS,
AND POST-CONCUSSIVE SYMPTOMS**

By

Mark Lawrence Ettenhofer

A DISSERTATION

**Submitted to
Michigan State University
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ABSTRACT

MILD TRAUMATIC BRAIN INJURY, EXECUTIVE FUNCTIONS, AND POST-CONCUSSIVE SYMPTOMS

By

Mark Lawrence Ettenhofer

Relatively few studies have been conducted examining the relationships between mild traumatic brain injury (mild TBI), neurocognitive deficits, and self-reported post-concussive symptoms in the post-acute phase of injury. Additionally, interpretation of the existing literature has been complicated by the small, treatment-derived samples of participants, high rates of injury-related litigation, and inappropriate control groups typical of studies in this area. Consequently, the significance of mild TBI to cognitive functioning and post-concussive symptoms in the long term remains unclear.

In this study, theoretical models of long-term impairment following mild TBI were evaluated, with a particular focus on the role of executive functions. Several methodological limitations of previous studies were addressed through the use of a relatively large sample of participants recruited in a non-treatment university setting ($n = 126$), an orthopedic injury control group, and powerful latent and structural modeling techniques of data analysis. Time since injury ranged from three months to six years.

Contrary to hypotheses, mild head injury was not associated with impairments in any cognitive domains examined, which included executive functions, processing speed, and verbal memory. Further, no relationship was found between the incidence of mild TBI and severity of post-concussive symptoms (as measured by the PCSC; Gouvier et al.,

1992). Instead, post-concussive symptoms were strongly related ($r = .50, p < .05$) to severity of general psychiatric symptoms (as measured by the BSI; Derogatis & Spencer, 1982). These findings suggest that neurological injury may be of little clinical significance to long-term cognitive and symptom outcome following mild TBI, relative to other, non-neurological factors. Additional research is necessary to identify and characterize psychological and emotional factors that may be important to long-term recovery from injuries involving a mild TBI.

TABLE OF CONTENTS

LIST OF TABLES	vi
LIST OF FIGURES	vii
INTRODUCTION	1
Mild Traumatic Brain Injury: An Overview	2
Executive Functions.....	4
Neurocognitive Sequelae of Mild TBI.....	9
Post-Concussive and Psychiatric Symptoms	14
Overview and Predictions	17
METHODS	20
Participants.....	20
Procedure	22
Measures	23
Statistical Analyses	31
RESULTS	33
Sample Description.....	33
Descriptive Statistics.....	34
Confirmatory Factor Analysis of Cognitive Variables	35
Head Injury Status Predicting Cognitive Functioning	37
Post-Concussive Symptoms (PCS) and Cognitive Functioning	39
Relationships between Head Injury, Symptom Variables, and EF	40
Follow-up Data Analyses.....	40

TABLE OF CONTENTS (CONTINUED)

DISCUSSION.....	42
LIST OF REFERENCES.....	60

LIST OF TABLES

Table 1. Demographic Characteristics of Screening Sample.....	47
Table 2. Constructs and Indicators.....	48
Table 3. Executive Function Indicators and Theoretical Components	49
Table 4. Demographic Characteristics by Participant Group	50
Table 5. Injury Characteristics by Participant Group	51
Table 6. Descriptive Statistics and T-tests of Cognitive Variables	52
Table 7. Descriptive Statistics and T-tests of Self-reported Symptom Variables.....	53
Table 8. Confirmatory Factor Analysis of Cognitive Variables	56
Table 9. Models of Head Injury Status Predicting Cognitive Functioning.....	57
Table 10. Models of Post-Concussive Symptoms and Emotional/Psychiatric Functioning	58
Table 11. Correlations of Selected Symptom Variables	59

LIST OF FIGURES

Figure 1. Hypothesized Variable Relationships.....	46
Figure 2. Freely-estimated model of EF factor structure (Model EF ₂).....	54
Figure 3. Freely-estimated model of PS and VM factor structure (Model C ₁).....	55

INTRODUCTION

Although a significant amount of research has been conducted in the acute phase of mild traumatic brain injury (TBI), relatively few studies have been conducted that might illuminate the relationships between mild TBI, neurocognitive deficits, and post-concussive symptoms in the long term. Additionally, most studies in this area have had significant methodological limitations and have been primarily descriptive in nature. It has therefore been difficult to draw firm conclusions regarding possible neurocognitive mechanisms underlying the impairment that some individuals experience following mild TBI. This information is critical to the development and implementation of effective treatments.

The overarching aim of the current project was to evaluate theoretical models of long-term impairment following mild TBI, with a particular focus on the role of executive functions. This study addresses methodological limitations of previous studies by recruiting a relatively large and ecologically valid mild TBI sample as well as an orthopedic injury control group, including several theoretically and psychometrically strong measures of the executive construct, and testing primary hypotheses using powerful latent and structural modeling techniques of data analysis.

As used in the text, “post-concussive symptoms” or “PCS” refer to those self-reported symptoms composing the traditional “post-concussive syndrome”, rather than all possible symptoms that might result from mild TBI. Both “post-concussive symptoms” (PCS) and “psychiatric symptoms” are those which the individual may experience in the long-term, rather than those which may be experienced acutely following the injury.

Mild Traumatic Brain Injury: An Overview

Traumatic brain injury (TBI) has long been recognized as a condition with wide-reaching effects on an individual's personal, social, and occupational functioning. By current estimates, approximately 200 traumatic brain injuries occur annually for every 100,000 individuals (Ryan, O'Jile, Gouvier, Parks-Levy & Betz, 1996). Of these injuries, upwards of 75% could be classified as 'mild' by standard medical criteria (Hayden, 1997). However, because the majority of mild brain injuries are never reported to any public health agency, it is likely that the true incidence rate is much higher. Many individuals may not believe that their injuries warrant medical attention, or they may choose not to seek medical attention for financial reasons (Kraus & McArthur, 1996). Unfortunately, the vast majority of studies of mild TBI have recruited participants who have self-selected by obtaining services at a treatment facility, thereby excluding a very large segment of the mild TBI population and biasing the sample in significant ways (Lees-Haley, Green, Rohling, Fox & Allen, 2003; Bernstein & de Ruiter, 2000; Newcombe, Rabbitt, & Briggs, 1994). A survey of available North American epidemiological literature conducted by the World Health Organization (WHO) Collaborating Centre Task Force on Mild TBI found that males are at substantially greater risk for experiencing a mild TBI (with a relative risk of up to 2.5); teenagers and young adults were also found to be at increased risk for mild TBI (Cassidy, Carroll, Peloso, Borg, von Holst et al., 2004).

Some individuals who sustain a mild TBI experience chronic "post-concussive symptoms" (PCS) such as headaches, irritability, insomnia, distractibility, depression, and dizziness (Segalowitz & Lawson, 1995; Alves, Macciocchi, & Barth, 1993; Varney

& Menefee, 1993). Additionally, research indicates that individuals who experience a mild TBI may be at an increased risk for other negative health-related outcomes. For example, epidemiological studies suggest that head injuries of any severity increase the likelihood that an individual will subsequently develop Alzheimer's disease or another form of dementia (Plassman, Havlik, Steffens, Helms, Newman et al., 2000).

Evidence from experimental animal models and post-mortem studies in humans indicates that mild TBI can result in various forms of structural damage to the brain, including focal cortical lesions and, more commonly, diffuse axonal injury (De Kruijk, Twijnstra, & Leffers, 2001; Rizzo & Tranel, 1996; Zohar, Schrieber, Getslev, Schwartz, Mullins & Pick, 2003). Unfortunately, currently available structural imaging techniques are generally poor at detecting this damage in living humans, during both the acute and chronic phases of mild TBI (Bigler, 1999; Bigler & Snyder, 1995). The insensitivity of structural imaging may be due to the type of damage that is commonly associated with mild TBI, which is believed to be primarily microstructural or neurochemical in nature (Bigler, 1999; Rizzo & Tranel, 1996). Functional imaging has demonstrated greater sensitivity to the effects of mild TBI, but because differing patterns of activation do not necessarily imply damage or dysfunction, these techniques are not commonly used in clinical settings, and great caution must be used in the interpretation of these results (McAllister, Sparling, Flashman, & Saykin, 2001). As such, behavioral measures (particularly those that assess executive functions, described below) within the context of a neuropsychological assessment would appear to be the most sensitive indicators of impairment following mild TBI (Cicerone & Azulay, 2002).

Executive Functions

Executive functions (EFs) are a class of higher-order cognitive abilities that have traditionally been associated with the structural and functional integrity of the frontal lobes (see Lezak, 1995; Stuss & Benson, 1984). The term “executive” commonly connotes a supervisory or coordinating role over non-executive forms of cognition in the pursuit of one or more goals. Hart, Schwartz & Mayer (1999) define EFs as those cognitive functions which enable the purposeful organization of behavior in a manner that is flexible and responsive to changing demands over time. Although areas of general agreement are small regarding the cognitive functions that are most central to effective executive control and the neuroanatomical substrate(s) of these functions, strong associations between putative measures of EF and functional outcomes in a wide range of psychiatric and medical disorders (Royall, Lauterbach, Cummings, Reeve, Rummans, Kaufer et al., 2002) have spurred a great deal of research (and debate) in this area. While the validity of the EF construct and the reliability of the tests commonly used to measure it have been criticized somewhat extensively (see Lowe and Rabbitt, 1998; Phillips, 1997; Rabbitt, 1997), measures of EF continue to be used in a wide range of settings, and researchers continue to expand the literature on EF methodology, theory, and application.

Neuroanatomically, EFs are believed to rely upon a system of reciprocal cortico-subcortical circuits connecting the prefrontal cortex, anterior cingulate cortex, basal ganglia, and cerebellum (Heyder, Suchan, & Daum, 2004). Although the prefrontal cortex appears to be the locus for the greatest quantity and complexity of EF processing, it is important to note that EF deficits (such as disinhibition, poor planning, behavioral and cognitive inflexibility, and difficulty initiating, maintaining, and monitoring

behaviors) can also result from damage to frontal or non-frontal components of this network.

Concepts of EF are ever-evolving, and theories regarding the cognitive structure of information processing underlying EF abound. Regarding what appear to be the most significant theoretical advances within recent years, a number of leading researchers have posited that EFs are accomplished in large part through the activity of one or more multi-purpose forms of working memory. Engle and Kane (2004), for example, stress the importance of working memory processes in allowing task demands/goals to be maintained in a sufficiently active state for task-interfering stimuli to be inhibited. This conception describes working memory processes as being much less related to effective “storage” of any information (a function believed to be accomplished by separate, non-frontal neural networks), than to the inhibition of conflicting information (a function primarily dependent upon the prefrontal cortex and the anterior cingulate cortex) (Engle & Kane, 2004). As such, “working memory” can be reframed as an executive form of attentional control – the selective, continued activation of goal-relevant information to the exclusion of irrelevant information.

Consistent with the common conceptual attribution that EFs coordinate and supervise lower-order cognitive processes, a number of researchers (Duncan & Owen, 2000; Engle and Kane, 2004; Goldman-Rakic, 1998) have suggested that the areas of the prefrontal cortex and anterior cingulate cortex serving working memory serve to code, process, and integrate many different types of information across modalities. However, whereas Goldman-Rakic (1998) cites studies utilizing single-cell recording in animals and functional imaging in humans to support sub-regional specialization of prefrontal

cortex by form of informational content (i.e., spatial vs. linguistic), cognitive experiments by Engle and Kane (2004) have found that working memory capacity (measured within a dual-task paradigm) appears to be predictive of a wide range of cognitive performance, irrespective of the form or modality of the information being processed in the working memory task. This discrepancy has not yet been resolved.

Although these theories of EF centered on working memory have empirical support and include a large proportion of functions within the EF spectrum, they are somewhat less compelling in terms of their ability to capture categories of function such as abstract thinking, cognitive flexibility, and behavioral initiation. Aside from the fact that these functions have been conceptually grouped beneath the traditional umbrella of EF, studies of various tasks assessing these functions have found relationships to activity or structural integrity of the prefrontal cortex (particularly dorsolateral areas) (Lezak, 1995; Stuss & Benson, 1984).

In a significant attempt to provide a broader theoretical context for EF that appears more inclusive of those functions not obviously related to working memory, Barkley (2001) posited from an evolutionary perspective that executive functions have developed progressively as a way to allow what were previously overt behaviors to be directed inward and represented mentally in the service of one or more goals. In this manner, Barkley (2001) suggested that the most basic executive function, inhibition (the delay or interruption of an overt behavior) allows for more complex executive functions such as working memory (self-directed mental representations of experience), goal pursuit and motivation (self-directed affect), and flexibility, fluency, and generativity (self-directed, exploratory analysis and synthesis of prior experience).

A number of studies have been conducted examining the relationships between various putative measures of EF in order to evaluate existing theories of EF and elaborate on the composition and organization of the EF construct. However, when quantitative studies on the issue of EF structure (most commonly, factor analyses evaluating unity vs. diversity of functions) are examined as a whole (as in Royall et al., 2002), results appear largely inconclusive. Whereas some investigations have yielded support for the modularity of executive functions (Burgess, Alderman, Evans, Emslie & Wilson, 1998; Duncan, Johnson, Swales & Freer, 1997), other studies have produced ambiguous results, with separable but related factors (Boone, Ponton, Gorsuch, Gonzalez & Miller, 1998; Miyake, Friedman, Emerson, Witzki & Howerter, 2000), or results that support a unitary model of executive function (Della Sala, Gray, Spinnler & Trivelli, 1998; Ettenhofer, Hambrick & Abeles, in press).

It appears likely that this ambiguity stems, at least in part, from divergent methods used to measure EF, as the particular measures and indices used can have a strong impact on the apparent factor structure of EF. Potential sources of error could include the exclusion of measures tapping one or more important subcomponents of EF, as well as the inclusion of non-executive indices from putatively EF measures (e.g., the word and color trials from the Stroop task). Additionally, it is possible that the discrepancies between these various results are related to differences in the populations studied. For example, if current findings of functional imaging studies of EF are correct in suggesting anatomical modularity of executive functions to subdivisions of frontal and sub-cortical brain areas within reciprocally-connected networks (Heyder, Suchan & Daum, 2004), then populations with injuries/illnesses causing diffuse damage to the executive network

might be expected to exhibit greater communality of EF than populations with injuries/illnesses causing more restricted damage to fewer subcomponents of that network. Further, the structure of the EF construct might appear different still in participant groups whose EFs are operating in a more “normal” fashion (such as control groups or participants in non-clinical studies).

In consideration of the issues described above, the present study included a wide variety of EF measures representing the component EFs of working memory, inhibition, cognitive flexibility (i.e., set shifting), abstract reasoning, and behavioral initiation (e.g., verbal fluency/generativity). Interpretation of the obtained results and the evaluation of primary hypotheses (see *Overview and Predictions*) are guided by an examination of the structure of EF in the current sample and its coherence in terms of the relevant neurocognitive theories presented.

Rationale for Latent Modeling of EF. Latent or structural modeling techniques were used to test all theoretical models. Because executive functions serve to coordinate and monitor other “lower level” cognitive functions in the service of one or more goals, all measures of executive functioning necessarily contain one or more non-executive components (Lezak, 1995; Stuss & Levine, 2002). In this study, in order to permit a “purer” examination of executive function, multiple measures of EF were used to construct a latent EF factor. Whereas any single index of executive function would necessarily contain an indeterminable combination of “executive function” variance, “non-executive” variance, and error variance, a latent EF variable pools shared variance from several separate indices of executive function, maximizing construct-relevant variance and excluding variance that is unique to any single measure.

Because the category of functions described as “executive” is rather under-specified, tasks which assess executive function vary considerably. Although the relatively divergent nature of the EF measures utilized in this study would serve as an obstacle to more traditional methods of data analysis, latent modeling techniques are able to capitalize on this divergence by extracting elements that are common across measures. In this case, it would be expected that these common elements would be very highly related to the construct in question (executive function), since there appears to be little commonality between the non-executive components of these measures as a whole.

One powerful illustration of these principles comes from a prior study conducted by the present author and colleagues in which a single latent executive function variable (EF) was derived from four measures of executive function (Ettenhofer, Hambrick & Abeles, 2005). Although the average observed test-retest correlation of these individual variables was .67, the test-retest correlation of the latent EF variable constructed from these same variables was much higher, at .96. The present study employed similar latent modeling techniques, in which a latent EF factor model was derived from seven measures of executive functioning. This method of analysis is designed to increase the reliability and construct validity of EF, and allow conclusions to be drawn with greater confidence.

Neurocognitive Sequelae of Mild TBI

Although the clinical significance of mild TBI has been downplayed by a number of researchers (Dikmen, Machamer & Temkin, 2001; Lees-Haley et al., 2003; Satz, Alfano, Light, Morgenstern, Zaucha, Asarnow et al., 1999), a significant number of studies suggest that even mild head injuries may negatively impact an individual’s attention (Binder, Rohling, & Larrabee, 1997; Brewer, Metzger & Therrien, 2002)

memory (Fisher, Ledbetter, Cohen, Marmor, & Tulsky, 2000; Mangels, Craik, Levine, Schwartz & Stuss, 2002; Thornton, 2003), visuospatial abilities (Cremona-Meteyard & Geffen, 1994), processing speed (Hinton-Bayre, Geffen, & McFarland, 1997; Pinkston, Gouvier, & Santa Maria, 2000), and executive abilities (Brooks, Fos, Greve & Hammond., 1999; Raskin & Rearick, 1996; Stablum, Mogentale & Umilta, 1996; Wallesch, Curio, Kutz, Jost, Bartels et al., 2001). Nevertheless, it has long been assumed that these cognitive deficits resolve within a few weeks post-injury, and consequently, the majority of studies examining the cognitive deficits following mild TBI have focused on individuals in the acute stage of recovery. However, some studies that have been conducted with post-acute mild TBI populations have found that deficits may indeed persist beyond the acute phase, with approximately 14% experiencing long-term disability in terms of employment (Larrabee, 1999). Interestingly, impairments in executive processes such as working memory, behavioral inhibition, and abstract problem-solving appear to be the most commonly-reported types of cognitive deficits found in long-term follow-ups after mild TBI (McDonald, Flashman & Saykin, 2002; Varney & Menefee, 1993). It is possible that the relative vulnerability of the frontal lobes to damage in mild TBI (Capruso & Levin, 1996; McDonald, Flashman & Saykin, 2002) accounts this finding, as executive functions are believed to rely heavily upon frontal areas (Lezak, 1995; Stuss & Benson, 1984).

Two widely-cited studies have employed meta-analytic techniques in examining cognitive functioning in the long-term following mild TBI. From their analysis, Binder, Rohling, & Larrabee (1997) reported a small but significant mean effect size of mild TBI on overall long-term cognitive performance ($d = .12$). Only one cognitive sub-domain

(Attention/Concentration) reached statistical significance ($d = .20$) in this meta-analysis. It should be noted that this sub-domain was defined broadly to also include a number of tasks that could be considered measures of executive functions, such as Trail Making Test Part B, and the Stroop Color-Word Test (see Stuss et al., 2001; Arbuthnott & Frank, 2000). Using alternative methodology, Frencham, Fox, and Mayberry (2005) found no significant cognitive effects of mild TBI in studies three months or later post-injury. However, the strength of this finding is limited somewhat by the small number of original post-acute studies included in the analysis (four), each with relatively small sample sizes ($N = 21$ to $N = 48$).

Additional data, derived from methodologically strong studies, would greatly enhance our ability to draw firm conclusions about cognitive functioning in the post-acute phase of mild TBI. Additionally, if long-term impairment is found to be present, a fuller examination of the precise relationships between mild TBI and specific forms of cognitive impairment would be an important secondary area of investigation. For example, any of a number of theoretical patterns of neuronal damage could be responsible for cognitive impairment, if present. One possibility is that mild TBI could cause direct, but non-selective damage to a variety of areas throughout the brain, affecting general information processing as well as the neurocognitive networks underlying executive, memory, and visuospatial functions. While relatively straightforward, this possibility fails to account for the theoretical interrelationships between neurocognitive networks, and appears to conflict with evidence demonstrating relative localization of both focal lesions as well as more diffuse axonal injury to the

frontal (and to a lesser degree, temporal) lobes (Capruso & Levin, 1996; McDonald, Flashman & Saykin, 2002).

Another, conceptually-related possibility is that mild TBI could result in diffuse forms of damage that, rather than directly affecting a wide range of neurocognitive networks, cause generalized cognitive impairment indirectly by slowing the overall rate of information processing (Wallesch et al., 2001). Theoretically, a reduced rate of information processing could impair component cognitive abilities by slowing task performance and reducing the availability of general cognitive resources (Salthouse, 1991). Providing some support for this possibility, Hart, Schwartz and Mayer (1999) found that those who had sustained a mild TBI made more “errors of action” when cognitive resources were limited by task demands. These errors were significantly correlated with measures of processing speed.

Although a model focused on reduced processing speed appears to offer a more coherent explanation of impairment following mild TBI than a direct-effects model, it is unclear whether deficits in processing speed can account for the full range of cognitive deficits that may follow mild TBI. For example, in a sample of individuals who had experienced a mild TBI, Chan (2002) found significant impairment in EF even after controlling for the presence of processing speed deficits. Additionally, in the study conducted by Hart, Schwartz and Mayer (1999) “errors of action” were also significantly correlated with measures of EF, which leaves room for another distinct mechanism of impairment.

If mild TBI can result in forms of cognitive dysfunction that are not explained by reductions in processing speed, a third possibility is that cognitive impairments following

mild TBI may result from the disruption of neurocognitive networks underlying the executive control of component cognitive processes. Because EFs have been theorized to be responsible for planning, execution, and on-line monitoring of component cognitive functions, and the networks underlying EF are reciprocally connected with many areas of the brain (Duncan & Owen, 2000; Goldman-Rakic, 1998; Heyder, Suchan, & Daum, 2004), it appears reasonable that EF impairment could negatively affect performance on a wide range of cognitive tasks. In such a scenario, reductions in apparent processing speed might reflect inefficient task performance as a result of poor planning, coordination, or monitoring of behaviors, rather than slowed speed of information processing as such. Limited support for this executive-mediated theory of cognitive dysfunction comes from a study in which individuals with mild TBI exhibited memory deficits relative to controls, but only under task conditions in which there were also demands upon the executive system (Mangels et al., 2002). Additionally, the possibility that mild TBI might impair executive control by disrupting communication between frontal and non-frontal areas appears to receive some support from an EEG study which found altered frontal-parietal coherence during an executive task among individuals who sustained a mild TBI (Cudmore, Segalowitz, & Dywan, 2000).

To summarize, there are a number of possible mechanisms by which mild TBI could affect long-term neurocognitive function. Those that were considered for examination in the present study included a) the direct effects of injury upon a wide variety of neurocognitive systems, b) diffuse injury that affects general cognitive functioning by reducing processing speed, and c) injury-related disruption of

frontal/executive networks that affects general cognitive functioning by preventing effective executive control.

It is also important to note that factors other than brain injury can affect the results of neuropsychological testing among some individuals who have experienced a mild TBI. Perhaps most importantly, many individuals who are (or who expect to be) involved in litigation related to a mild TBI may be unmotivated to perform well, or even motivated to perform poorly, because of factors surrounding compensation (Binder & Rohling, 1996; Larrabee, 1999). Therefore, the results of studies of mild TBI that draw participants from clinical treatment populations may be biased, especially if they are selected on the basis of symptoms complaints (Larrabee, 1999). As such, alternate sampling techniques were employed in the present study in order to minimize these biases.

Post-Concussive and Psychiatric Symptoms

Although the current study focused primarily upon cognitive abilities as measured through neuropsychological testing, it is also important to consider the importance of subjective symptom reports to the overall picture of mild TBI and the varying trajectories of recovery experienced by different individuals. During the acute phase of recovery from mild TBI, it is common for patients to report symptoms such as dizziness, headache, fatigue, nausea, irritability, confusion, and difficulty concentrating. In the majority of cases, these symptoms appear to remit within the first few weeks or months of recovery; however, a substantial proportion of individuals who have sustained a mild TBI continue to report these or similar symptoms for a year or more following injury. A DSM-IV diagnosis of Post-Concussive Disorder applies when an individual experiences these symptoms to an impairing or distressing degree for more than 3 months following a mild

TBI. In terms of the typical trajectory of recovery from these symptoms, epidemiological studies of mild TBI have found the prevalence of clinically-significant post-concussive symptoms to be approximately 51% at six weeks post-injury (Rutherford, Merrett & McDonald, 1977), and 34% at three years post-injury (Fee & Rutherford, 1987).

Surprisingly, many studies have been unable to find solid relationships between post-concussive symptoms (PCS) and the actual occurrence of mild TBI. For example, despite a large sample size, Paniak, Reynolds, Phillips, Toller-Lobe, Melnyk et al. (2002) were only partially successful at discriminating mild TBI patients from controls using logistic regression based upon PCS. Likewise, neither Sawchyn, Brulot, & Strauss (2000) nor Gouvier, Cubic, Jones, Brantley & Cutlip (1992) were able to find a significant relationship between PCS and head injury status, but PCS were correlated significantly with levels of daily stress (Gouvier et al., 1992) and litigation activity (Bernstein & de Ruiter, 2000; Larrabee, 1999; Lees-Haley et al., 2003; Newcombe, Rabbitt, & Briggs, 1994). Thus, evidence strongly suggests that factors other than mild TBI impact the degree to which an individual reports these “post-concussive” symptoms.

Another such factor appears to be emotional functioning; individuals who experience high levels of psychiatric symptoms in addition to mild TBI appear to exhibit a greater number of PCS at long-term follow-up (Mooney & Speed, 2001; Sawchyn, Brulot, & Strauss, 2000). Further, individuals who have experienced a mild TBI appear to be more susceptible to the effects of experimentally-induced stress, which increases the frequency and severity of their post-concussive symptoms (Hanna-Pladdy, Berry, Bennett, Phillips & Gouvier, 2001).

Clearly, a theoretical model is needed to explain the somewhat enigmatic relationships between mild TBI, PCS, and psychiatric symptoms. Although previous research suggests that mild TBI might not directly increase levels of PCS, evidence appears to indicate that emotional/psychiatric factors play an important role. Psychiatric symptoms such as depression may be more common among individuals who have sustained a mild TBI (Mathias & Coats, 1999), and may also increase the severity of post-concussive complaints (Sawchyn, Brulot, & Strauss, 2000). Thus, models in which psychiatric symptoms mediate the relationship between mild TBI and PCS are worthy of examination.

In addition to the possible role of psychiatric symptoms in the expression of post-concussive symptoms following mild TBI, hypotheses have also been raised regarding the role of cognitive deficits in mediating the effects of mild TBI on levels of post-concussive symptoms. For example, in a relatively small sample of individuals with mild TBI, Pinkston, Gouvier, and Santa Maria (2000) found a significant relationship between processing speed and severity of post-concussive symptoms, suggesting that the relationship between mild TBI and PCS may be mediated by neurocognitive factors. Replication and extension of these preliminary results into a wider context might provide additional information about the relationship between mild TBI, neurocognitive function, and PCS. For example, because attentional abilities are conceptually related to EF (see Lezak, 1995), it could be argued that post-concussive complaints of inattention and distractibility may be accounted for, at least in part, by executive dysfunction. In addition, for both processing-speed- and EF-mediated models of PCS, impairments in cognitive functioning could be hypothesized to impact psychiatric symptoms by

decreasing stress tolerance levels and/or the effectiveness of psychological coping mechanisms.

To summarize, it appears likely that the presence of emotional/psychiatric symptoms increases the expression of self-reported post-concussive symptoms. However, a number of possibilities have been raised regarding mechanisms by which mild TBI may affect long-term PCS. These include a) the direct effects of injury in causing PCS, b) injury-related increases in psychiatric symptoms, which in turn result in post-concussive symptom complaints (PCS), c) diffuse injury that increases symptom complaints by reducing processing speed, and d) injury-related disruption of frontal/executive networks that increases symptom complaints by preventing effective executive control.

Overview and Predictions

Mild TBI is a highly prevalent public health concern, with inestimable costs to the individuals who experience long-term symptoms. However, directed studies of the effects of mild TBI are limited, and available evidence is insufficient to draw firm conclusions regarding the potential long-term effects. Additionally, it is unclear what relationship, if any, exists between mild TBI, psychiatric symptoms, and reported post-concussive symptoms (PCS). Clarification of the causal factors underlying the cognitive impairments that some individuals experience following mild TBI, and their relationship to psychiatric symptoms and PCS, is essential to the development and implementation of methods of remediation. In the present research, a number of theoretical models were evaluated in order to investigate the neurocognitive mechanisms that may result in cognitive impairments following mild TBI. These models also include an examination of

the theoretical relationships between mild TBI, psychiatric symptoms, and self-reported symptom complaints (PCS). Because it was believed that injury-related deficits in executive function (EF) were likely to be primarily responsible for the dysfunctions that may follow mild TBI (both cognitive and subjective/symptomatic), a central (mediational) role was hypothesized for EF. Primary hypotheses are depicted (in aggregate form) in Figure 1.

In addition to providing a theoretical framework for the examination of fundamental hypotheses of impairment following mild TBI, the present research addresses a number of limitations of prior studies. First, mild TBI is often associated with such factors as injury to other bodily systems and emotional distress; therefore it has been suggested that studies of mild TBI need to include an appropriate control group to rule out the possible effects of these non-neurological factors (Satz et al., 1999; Dikmen & Levin, 1993). This study included a control group composed of individuals with mild orthopedic injuries, thereby controlling for generic effects of injury. Second, the vast majority of previous studies have obtained participants from treatment centers, a practice which excludes a very large segment of the injured population and introduces potentially confounding psychogenic factors such as malingering (Lees-Haley et al., 2003; Bernstein & de Ruiter, 2000; Newcombe, Rabbitt, & Briggs, 1994). Ecological validity in this study was greatly enhanced by the inclusion of participants without regard to whether they sought medical attention for their injuries. Third, many studies of mild TBI have been statistically underpowered, limiting their ability to detect more subtle effects; sample size in this study ($N = 126$) provides sufficient statistical power (.80 or higher) to detect meaningful relationships for all analyses of interest. Finally, this study utilized a

battery of widely-used neuropsychological tests that were selected based upon their theoretical relevance to mild TBI and their strong psychometric properties. These tests provide a high level of sensitivity in detecting the potential effects of head injury, and facilitate the application of results to clinical assessment and treatment planning.

Specific Aim 1. To evaluate theoretical models predicting performance on measures of cognitive functioning based upon long-term mild TBI status. Three general classes of models were considered, and tested as appropriate: direct effects models, processing speed (PS)-mediated models, and executive functions (EF)-mediated models. It was hypothesized that head injury status would be a significant predictor of all general areas of cognition measured, but that an EF-mediated model would best represent the data collected.

Specific Aim 2. To evaluate theoretical models predicting patient-reported “post-concussive symptom” complaints (PCS) from long-term mild TBI status. Three general classes of models were considered, and tested as appropriate: direct effects models, psychiatrically-mediated models, processing speed (PS)-mediated models, and executive function (EF)-mediated models. It was hypothesized that mild TBI would be a significant predictor of PCS, but that an EF-mediated model would best represent the data collected.

METHODS

Participants

Part 1: Screening. All individuals enrolled in courses at Michigan State University that utilized the Human Participants Subject Pool (HPSP) were eligible for participation in the initial screening phase of the study. The HPSP required all students who wished to participate in research to register at a central web site maintained by the university. Through this web site, students were able to obtain course credit for the completion of web-based measures. The screening questionnaire described below was one such measure, identified as part of a “study of injury” for which all students qualified to participate in the subject pool were eligible. Informed consent for participation was obtained with the use of electronic signatures, an IRB approved practice that is self-contained within the HPSP web page. In addition to collecting information related to eligibility for Part 2 of the study, name, phone number, and e-mail address were obtained as contact information for participant recruitment. A total of 3,347 students participated in the HPSP screenings for this study. Demographic characteristics of the screening sample are shown in Table 1.

Part 2: Neuropsychological Testing. Of those individuals who participated in screening, 332 (9.92%) were invited to participate in Part 2 of the study (neuropsychological testing). Of these individuals, 141 (42.47%) volunteered to participate. Fifteen participants were excluded from analyses because of information obtained during interview which violated group eligibility criteria and was inconsistent with information obtained during screening. This included 4 individuals whose mild TBIs had occurred greater than six years previously, 3 individuals who had experienced

two or more orthopedic injuries within the previous five years, 2 individuals who had experienced a head injury less than three months prior to testing, 2 individuals who had experienced three or more lifetime head injuries, 2 individuals who had no injuries of sufficient severity to qualify as a lifetime mild TBI or an orthopedic injury within the last five years, 1 individual who had been hospitalized for greater than 24 hours as the result of an injury, and 1 individual who had experienced PTA for greater than 24 hours as the result of an injury.

This study follows the Kay, Harrington, Adams, Anderson, Berrol et al. (1993) definition of mild TBI: an episode in which an individual reports sustaining blunt head trauma that is accompanied by a loss of consciousness (LOC) that does not exceed 30 minutes in duration, and/or a period of post-traumatic amnesia (PTA) of less than 24 hours. Individuals who report ever experiencing LOC for greater than 30 minutes, PTA for greater than 24 hours, a penetrating head injury, or more than two lifetime head injuries were excluded from both the mild TBI (MTBI) and orthopedic injury (OI) groups. The OI group is composed of those individuals who do not report having ever sustained a head injury (of any severity) but do report having sustained a broken bone. Individuals who were hospitalized for greater than 24 hours for treatment were excluded from both MTBI and OI groups in order to control for injury severity. Other exclusions for both the MTBI and OI groups include the following: evidence of current substance dependence that may prevent sober testing, history of psychotic illness, history of autism, sensory-motor handicap, neurological illness, or native language not English. Individuals who were prescribed stimulant, anti-psychotic, anti-depressant, or anti-convulsant medications were also excluded.

One aspect of backup recruitment was also utilized, in which participants who had already completed the screening described above were offered and provided with monetary compensation for participation in the neuropsychological assessment portion of the study. This strategy enabled the research team to recruit individuals who had participated in the pre-screening in prior semesters, as well as individuals who participated in the pre-screening in the current semester but who may not need and/or want course credit for other reasons. Monetary compensation was offered at fair and appropriate levels (i.e., \$10 to \$15 per hour of participation) that minimized any possible coercive influence. Written informed consent was obtained from all individuals at the beginning of the testing session.

The final sample consisted of 63 participants who had sustained a mild TBI within the past 6 years (MTBI Group), and 63 participants who had sustained a mild orthopedic injury within the past 6 years (OI Group). A priori power analyses indicated that the power to detect medium effects sizes with this N would be .80 for independent-sample t-tests and ANOVAs, .86 for Chi-squares, .94 for correlations, and .93 to .99 for regressions analyses with 1 to 5 predictors. Power to test variable interrelationships in SEM models tends to be comparable to that in more traditional analytic techniques, but could not be estimated directly. However, the samples of the present size are considered “medium” by Kline (2004).

Procedure

Following the online screening, participants who met criteria for the MTBI or OI groups were contacted and asked if they would like to participate in a study of “individual differences in cognition” for additional course credit. Those who volunteered

met with an examiner, who was blind to the participants' personal history of injury, for a one-on-one neuropsychological testing session. The participant then completed a fixed battery of tests and questionnaires, as described below. Completion of this battery took two hours or less. Following testing, the examiner engaged the participant in a brief structured interview, described below. The participant was then debriefed in full, awarded course credit or paid as necessary, and released.

Measures

Constructs of interest and their indicators are summarized in Table 2. Indicators of executive function and their theoretical components are summarized in Table 3.

Screening Questionnaire. This online questionnaire was designed to obtain the following information: name, phone number, e-mail address, gender, age, racial/ethnic group membership, native language, level of education, handedness, current sensory/motor capabilities, frequency and type of alcohol and substance use, current medications prescribed, history of neurological illness, psychotic illness, and developmental disorder, and a detailed history of injuries. For head injuries, detailed information was obtained to allow classification of injury severity. Additionally, all items from the **Post-Concussion Syndrome Checklist (PCSC)** (Gouvier et al., 1992) were included in this questionnaire in order to obtain data regarding participants' current level of post-concussive symptomatology. Total PCSC score was used as a measure of post-concussive symptoms, as it has demonstrated validity and specificity for this purpose (Gouvier et al., 1992).

Wisconsin Card Sorting Test (WCST). The WCST is a measure of abstract reasoning, concept formation, and set-shifting. This test requires the participant to sort a

number of cards based upon shifting criteria of color, shape, and number (Heaton et al., 1993). Along with studies employing functional brain imaging which have shown that this task produces activation in prefrontal areas (Rezai, Andreasen, Alliger, Cohen, Swayze et al., 1993; Konishi, Kawazu, Uchida, Kikyo, Asakura et al., 1999; Berman, Ostrem, Randolph, Gold, Goldberg et al., 1995; Rogers, Andrews, Grasby, Brooks, & Robbins, 2000; Wang, Kakigi, & Hoshiyama, 2001), studies have also found that greater numbers of perseverative errors on this test are associated with prefrontal lesions (Anderson, Damasio, Jones, & Tranel, 1991) and frontal metabolic deficits (Lombardi, Anderson, Sirocco, Rio, Gross et al., 1999). This study employed a computerized 128-card version of the WCST that takes 10 to 15 minutes to administer. Total number of perseverative errors on the WCST was used as a measure of executive function.

Color-Word Interference Test (CWIT). The CWIT is a subtest of the Delis-Kaplan Executive Functioning System (D-KEFS; Delis, Kaplan, & Kramer, 2001a; Delis, Kaplan, & Kramer, 2001b) that has been adapted from the Stroop Color-Word Test (Stroop, 1935). This widely-used test contains four trials which are designed to measure the ability to focus attention, inhibit prepotent responses, and shift mental set. Participants are required to read as quickly as possible through a list of “xxxx” in blue, red, or green ink (Condition 1: Color Naming), a list of color words (“blue”, “red”, or “green”; Condition 2: Word Reading), a list of color words in which the color of the text is incongruent with the word itself (Condition 3: Inhibition), and a list which requires the participant to alternate between list demands of Conditions 1, 2, and 3 (Condition 4: Inhibition/Switching). Total time to administer this task is approximately 6 minutes. Studies have shown that individuals with frontal lobe lesions have greater difficulty with

the Inhibition trial than both matched controls (Vendrell, Junque, Pujol, Jurado, Molet et al., 1995) and individuals with non-frontal brain lesions (Stuss, Floden, Alexander, Levine, & Katz, 2001). Total time on Condition 3 (Inhibition) was used as an index of executive functioning for this study.

Trail Making Test (TMT). The TMT is a subtest of the D-KEFS (Delis, Kaplan, & Kramer, 2001a; Delis, Kaplan, & Kramer, 2001b) that has been adapted from Reitan's (1958) original test of the same name. This widely-used, timed paper-and-pencil test consists of five conditions. In Condition 1 (Visual Scanning), the participant is presented with a page of circles containing various letters and numbers, and they are required to identify and "cross out" all of the circles containing the number 3 only. In Condition 2 (Number Sequencing), the participant is presented with a similar page of circled letters and numbers and is required to draw a line connecting the numbered circles in sequential order. Similarly, in Condition 3 (Letter Sequencing) the participant is required to connect only letters in sequential order. Condition 4 (Number-Letter Switching) requires the participant to draw a line connecting numbered and "lettered" circles in alternating sequential-alphabetic order. Finally, Condition 5 (Motor Speed) requires participants to draw a line connecting empty circles in a pre-specified order (indicated by a dotted line on the page). Total time to administer this task is approximately 7 minutes. Score on each part of the TMT is determined by the time required to complete each trial. Whereas performance on Conditions 1, 2, 3, and 5 of the TMT depend largely upon the participant's psychomotor speed and visual search abilities, Condition 4, analogously to Trial B of Reitan's (1958) TMT, places additional demands upon the participant's working memory and cognitive flexibility (Crowe, 1998; Arbuthnott & Frank, 2000).

Total time on trial 4 (Letter-Number Switching) was used as a measure of executive function in this study.

Verbal Fluency Test (VFT). The VFT is a subtest of the D-KEFS (Delis, Kaplan, & Kramer, 2001a; Delis, Kaplan, & Kramer, 2001b) measuring verbal generativity/initiation (fluency) that has been adapted from a number of similar tests (Benton, Hamsher, & Sivan, 1983). This test consists of three conditions. In Condition 1 (Letter Fluency), the participant is asked to say as many words as possible within one minute that begin with a particular letter of the alphabet, excluding proper nouns as well as different forms of the same word. Condition 1 has three trials, one each for the letters “F”, “A”, and “S”. In Condition 2 (Category Fluency), the participant is asked to say as many words as possible that fall within a certain category (animals or boy’s names) within one minute each. Total scores for Conditions 1 and 2, respectively, are the total number of correct responses for all trials. Condition 3 (Category Switching) is similar to condition 2, but the participant is required to alternate between two categories (fruits and furniture) during a single one-minute trial. Total score (correct switches) for Condition 3 is the number of responses that fall within the specified categories and alternation pattern.

The total time to administer this task is approximately 7 minutes. Performance on measures of verbal fluency has been found to be sensitive to frontal lobe dysfunction (Raskin & Rearick, 1996), and is associated with prefrontal brain activation in studies of functional imaging (Frith, Friston, Liddle, & Frackowiak, 1991; Gourovitch, Kirkby, Goldberg, Weinberger, Gold et al., 2000). This study utilized total score for Condition 1 (Letter Fluency), total score for Condition 2 (Category Fluency), and total number of correct switches for Condition 3 (Category Switching), as indices of executive function.

Operation Span (OS). OS is a measure of working memory capacity adapted from Turner & Engle's (1989) study that is administered with the aid of a computer. The task consists of 12 trials total. During each trial, the participant is presented with a series of arithmetic problems followed by a word (e.g., "Is $(6 \times 2) - 5 = 7$? DOG"). The participant is instructed to respond "Yes" or "No" to the arithmetic problem, say the word aloud, and try to remember the word for later recall. Immediately after the participant says the word, the examiner presses a key that presents a blank screen for 500 ms. Subsequently, either another arithmetic problem and word, or a recall cue appears, depending upon the total number of items and the number of items already presented in the selected trial. When the recall cue appears, the participant is instructed to repeat all words from that trial. The number of items in each trial increases sequentially from two to five over each of the 12 trials. This study used total number of word errors as an index of executive function. Total time to administer this test was approximately 5 minutes.

Rotation Span (RS). RS is a measure of working memory capacity adapted from Shah and Miyake's (1996) study that is administered with the aid of a computer. The task consists of 12 trials total. During each trial, the participant is first presented (for 1000 ms) with a long or a short arrow in 1 of 8 rotational orientations. Subsequently, a normal or mirror-reversed letter (G, F, or R) is presented, in 1 of 8 rotational orientations. The participant is instructed to mentally rotate the letter so that they can verbally indicate whether it is normal ("Yes") or mirror-reversed ("No"). Immediately after the participant responds, the examiner presses a key that presents a blank screen for 500 ms. Subsequently, either another arrow or a recall cue appears, depending upon the total number of arrow-letter pairs and the number of arrow-letter pairs already presented in the

selected trial. When the recall cue appears, the participant is instructed to indicate all arrows (long or short) in their correct rotational orientations on an answer sheet. The number of arrow-letter pairs in each trial increases sequentially from two to five over each of the 12 trials. This study used total number of arrow errors as an index of executive function. Total time to administer this test was approximately 5 minutes.

California Verbal Learning Test – Second Edition (CVLT-II). The CVLT-II is a 16-item word-learning task that requires the participant to learn four words from each of four semantic categories over a series of four presentations, as well as after a short and a long delay (Delis, Kramer, Kaplan, & Ober, 2000). This study used the total number of correct Immediate Recall responses (trials 1-5), the total number of correct Short Delayed Recall responses, and the total number of correct Long Delay Recall responses as indices of memory functioning. Total time to administer this task was approximately 20 minutes, plus a 20-minute delay, during which other tasks were administered.

Letter Comparison (LC), Number Comparison (NC), and Pattern Comparison (PC). For these paper-and-pencil tests of processing speed, participants are required to examine a series of pairs of letter groups (LC), numbers groups (NC), or line segments (PC), and decide as rapidly as possible if each pair of stimuli is the same or different (Salthouse & Babcock, 1991). For each stimulus pair, the participant writes S on a line in between the pair if they are the same, or D if they are different. After being provided with 3 example pairs, the participant is given 30 seconds to complete as many items as possible for each of two total pages of the task. Total time to administer all three of these tasks is approximately 5 minutes. For each task, the participant received an overall score of total number correct minus total number incorrect; items that were not

attempted did not affect overall task score. This study used total score for LC, NC, and PC, respectively, as indices of processing speed (PS).

Intelligence. A two-subtest short form equivalent of the WAIS-III (Wechsler, 1997) was used to estimate Full-Scale IQ (FSIQ) for the purposes of demographic description of the sample and various between-group comparisons. Reports indicate that this short form, which utilizes only the Vocabulary and Block Design subtests of the WAIS-III, has good reliability (.94 to .95) and validity (.89 to .90) within an 18-34 year old sample (Jeyakumar, Warriner, Raval, & Ahmad, 2004). Data from Sattler (2001) were used to compute an estimate of FSIQ from the WAIS-III scaled scores from these tasks.

Vocabulary. Vocabulary is a subtest of the WAIS-III in which the participant is required to orally define a series of word that are presented both orally and in writing by the examiner (Wechsler, 1997). Each word is scored according to various criteria as inadequate (0 points), marginally adequate (1 point), or correct (2 points). Total score from this measure was used in combination with Block Design for the purpose of estimating FS IQ.

Block Design. Block Design is a subtest of the WAIS-III in which the participant is asked to use small blocks to reproduce a series of two-dimensional designs presented by the examiner (Wechsler, 1997). Each block has two white sides, two red sides, and two sides that are half red and half white. Up to 14 trials are administered (more commonly, 7 to 10) in order of increasing difficulty. Average time to administer this task is approximately 12 minutes. This study used total score as an index of

visuospatial functioning. Total score from this measure was used in combination with Vocabulary for the purpose of estimating Full-Scale IQ.

Brief Symptom Inventory (BSI). The BSI is a 53-item questionnaire measure that assesses nine psychiatric symptom dimensions, including somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism. Participants are asked to rate the amount of distress associated with each symptom on a 5-point rating scale (Derogatis & Spencer, 1982). In this study, total symptom severity score was used as an overall index of psychiatric symptoms for primary path analyses. Individual dimension scores were used for further exploratory analyses. Normative data for a university was used to determine clinical cutoffs levels of psychiatric symptoms (Cochran & Hale, 1985). Individuals with moderate to severe levels of overall symptoms were provided with an appropriate referral for assessment/treatment.

Structured Interview. The structured interview contained the same questions related to injury history that the participant provided in the screening questionnaire. Additionally, information was collected regarding the use of medications and other substances to ensure that screening criteria for medications, alcohol, or drugs were not violated. Because of the presumed greater validity of an in-person interview with a trained staff member than the initial on-line screening questionnaire, the structured interview was used as the primary source of information when verifying group inclusion criteria. However, some participants were re-contacted to voluntarily provide additional information for more information to resolve injury history discrepancies.

Statistical Analyses

Data Preparation. As recommended in recent methodological texts, extreme outliers ($z > 4.0$ and more than .5 SD from next score) were truncated to within .5 SD of the next nearest score to prevent the undue influence of single scores on linear models and reduce Type I and Type II error (see Wilcox, Keselman, & Kowalchuk, 1998). The expectation maximization (EM) method was used to impute missing data (0.53% of all data points).

Analysis. The AMOS 5.0 (2003) statistical package, using the Maximum Likelihood method, was used for all latent variable and structural analyses. The GPOWER (1996) statistical program was used for all power analyses. The SPSS 11.01 (2001) statistical package was used for all other analyses. For all SEM analyses, the following fit indices have been reported and interpreted as outlined by Kline (2004): (1) Pearson chi-square for absolute comparisons of fit between nested models; (2) Goodness of Fit Index (GFI; Joreskog & Sorbom, 1981) for which values .90 or greater are considered good fit; (3) Comparative Fit Index (CFI; Bentler, 1990) for which values .90 or greater are considered good fit; and (4) Root Mean Square Error of Approximation (RMSEA; Steiger, 1990) for which values .08 or less are considered acceptable and .05 or less are considered good fit. Although the use of structural equation modeling with manifest variable models is statistically equivalent to a regression-based path analytic approach (Kline, 2004), structural equation modeling also provides a number features and additional statistics that are useful in evaluating and comparing models. Hypotheses for specific variable relationships have been evaluated based upon the statistical significance of relevant path/covariance coefficients inherent to each model. Hypotheses regarding

the relative fit of a given model haven been examined through a comparison of relevant indices of overall model fit (e.g., χ^2 , GFI, CFI, RMSEA).

As a secondary check on the accuracy of structural SEM models, standardized composite variables were computed for all cognitive constructs of interest (executive functions, processing speed, and verbal memory) with the same indicators utilized in the SEM models, and all hypotheses were evaluated using traditional statistical techniques (e.g., t-tests & correlations). Results from these analyses were consistent in all cases with those reported below. Additionally, considering the possible effects of gender and the overrepresentation of females in the overall sample, all structural SEM models were re-evaluated with the inclusion of gender as a manifest variable in order to control for gender effects; all relationships in these models were consistent with those reported below.

RESULTS

Sample Description

Demographic information for the participant groups is provided in Table 4. Mean years of education (12.86 years for both MTBI and OI groups) reflects the recruitment methods, which obtained primarily Freshmen or Sophomores at the University. Mean Estimated FSIQ (113.32 and 111.90 for MTBI and OI groups, respectively) was elevated relative to the general population but also consistent with the university sample. Mean age, education, and FSIQ were not significantly different between the groups. The majority of participants were female (63.5% and 69.8% for the MTBI and OI groups, respectively), a proportion which was not significantly different between the groups. Mean alcoholic drinks per month (30.44 and 22.72 for the MTBI and OI groups, respectively) and mean days per month in which other drugs were used (1.11 and .62 for the MTBI and OI groups, respectively) were somewhat higher in the MTBI than the OI group (marginally significant at $p = .06$ for both comparisons). Consistent with the screening sample, both participant groups were primarily Caucasian (85.7% and 95.2% for the MTBI and OI groups, respectively). The overall chi-square for the between-groups comparison of ethnicity/race was non-significant ($\chi^2(4) = 7.52, p > .05$). Results were also non-significant when examining race/ethnicity as a dichotomous variable (Caucasian and Non-Caucasian; $\chi^2(1) = 3.32, p > .05$).

Regarding characteristics of the injuries sustained between the two participant groups, a significantly smaller proportion of participants received treatment for their injury in the MTBI group than in the OI group (73.0% and 92.1%, respectively, $p < .01$). However, neither group was significantly more likely to be hospitalized for their injury

(39.7% and 52.4% for the MTBI and OI groups, respectively). Participants were significantly more likely to be involved in litigation related to their injury in the MTBI group than in the OI group (4.8% and 0%, respectively, $p < .01$). However, it should be noted that the number of participants involved in litigation was very small overall relative to other available studies of injury. An examination of the cause of injury by participant group (Table 5) demonstrates that cause of injury in the MTBI group is somewhat more varied than that in the OI group; the chi-square statistic was significant at the $p < .05$ level for the overall comparison. However, participation in sports activities was the primary source of injury for individuals in both the MTBI and OI groups (50.8% and 74.6%, respectively), with accidental falls (22.2% and 11.1%, respectively) being the second most common cause. The cause of injury in this sample is somewhat discrepant from other studies of the U.S. population as a whole, which cite motor vehicle accidents and accidental falls as the primary causes of mild TBI (Bazarian, McClung, Shah, Cheng, Flesher et al., 2005; Cassidy et al., 2004). This contrast is likely due to differences between the university sample used in this study and the hospital/treatment samples typical of other studies.

Descriptive Statistics

Independent samples t-tests were conducted to examine potential differences between the MTBI and OI groups on all relevant cognitive and symptom variables (see Tables 6 and 7). As shown, the MTBI group performed significantly better than the OI group on two of the three processing speed tasks: LC [$t(124) = 2.34, p < .05$] and PC [$t(124) = 2.34, p < .05$]. A lack of significant differences between groups on TMT Trial 5 (Motor Speed, $t(124) = -.26, p > .05$), and significant correlations between head injury

and these variables when TMT Trial 5 was covaried, suggested that slowed motor performance could not account for these differences in processing speed. Significant differences were not found for any other cognitive variables, or for any of the symptom variables examined.

Confirmatory Factor Analysis of Cognitive Variables

In the first stage of model analysis, a series of CFAs was conducted to determine the appropriate factor structure of the EF construct. An initial CFA was conducted in which all EF variables (WCST perseverative errors, CWIT Trial 3 total time, TMT Trial 4 total time, VFT Letter, Category, and Switching scores, OS total correct trials, and RS total correct trials) loaded onto a single latent EF factor. In this model (denoted EF1), the unstandardized factor loading of VFT Letter score was set to 1 to scale the latent EF variable; all other factor loadings were freely estimated. Additionally, in order to properly account for method covariance, the error terms for VFT Letter, Category, and Switching were permitted to correlate with one another, as were error terms for OS and RS. Model EF1 demonstrated good overall fit, $\chi^2(16, N = 126) = 15.91, p > .05$, GFI = .97, CFI = 1.00, RMSEA = .00; however, the loading of VFT Switching on the EF factor was non-significant. Therefore, VFT Switching was excluded from subsequent models. Model EF2, which demonstrated good fit [$\chi^2(12, N = 126) = 14.59, p > .05$, GFI = .97, CFI = .98, RMSEA = .04], was constructed by removing VFT Switching from EF1. All factor loadings in this model were statistically significant at the $p < .05$ level, a result which was unchanged by testing the model with alternate indicators' factor weights set to 1 for scaling. Model EF2 is depicted in Figure 2.

In the next stage of analysis, a multiple-groups confirmatory factor analysis was conducted to evaluate the appropriateness of using the EF2 factor model with both the MTBI and OI groups, respectively. This is accomplished by evaluating the measurement model in both groups simultaneously with appropriate levels of cross-group equality constraints. If there is no significant difference in fit (as determined by a comparison of the chi-square statistic) of an unconstrained model to a model in which factor loadings are equality-constrained, then the construct's factor structure is judged to be comparable across the groups. However, a significant reduction in fit would suggest that the relations of the indicators to one another varied by group.

The baseline, unconstrained two-group model (EF3) demonstrated acceptable overall fit [$\chi^2(24, N = 126) = 34.54, p > .05$, GFI = .93, CFI = .91, RMSEA = .06]. The following model (EF4), which constrained the EF factor loadings to be equal across groups, demonstrated good overall fit [$\chi^2(30, N = 126) = 37.79, p > .05$, GFI = .93, CFI = .94, RMSEA = .05], which was not significantly changed from that of the baseline, unconstrained multi-group model EF3 [$\Delta\chi^2(6, N = 126) = 3.25, p > .05$]. It was therefore concluded that the factor structure of the EF construct was similar between the two groups, a finding which permitted interpretation of results from latent models of EF including all participants. Fit statistics for EF models are presented in Table 8.

Analytic procedures were modified slightly in order to evaluate latent factors for processing speed (PS) and verbal memory (VM), factors whose number of indicators (3 each) prevents the evaluation of each factor's fit as an isolated model. Instead, a measurement model (C1) was created with two latent factors: one factor for PS (whose indicators included LC, NC, and PC totals), and one factor for VM (whose indicators

included Immediate Memory total, Short Delay total, and Long Delay total).

Unstandardized factor loadings for LC and Immediate Memory were set to 1 for scaling of the latent PS and VM factors, respectively, and these latent factors were permitted to correlate. The initial fit of model C1 was good, $\chi^2(8, N = 126) = 7.41, p > .05$, GFI = .98, CFI = 1.00, RMSEA = .00. All factor loadings in this model were statistically significant at the $p < .05$ level, a result which was unchanged by testing the model with alternate indicators' factor weights set to 1 for scaling. Model C1 is depicted in Figure 3.

In the next stage of confirmatory factor analysis for the combined PS-VM model, a baseline, unconstrained two-group model was created. This model (C2) demonstrated good overall fit, $\chi^2(16, N = 126) = 11.60, p > .05$, GFI = .97, CFI = 1.00, RMSEA = .00. The following model (C3), which constrained all factor loadings to be equal across groups, demonstrated good overall fit [$\chi^2(20, N = 126) = 17.29, p > .05$, GFI = .96, CFI = 1.00, RMSEA = .00], which was not significantly changed from that of the baseline, unconstrained multi-group model C2 [$\Delta\chi^2(4, N = 126) = 5.70, p > .05$]. It was therefore concluded that the factor structure of the PS and VM constructs was similar between the two groups, which permitted interpretation of results from latent models of these constructs including all participants. Fit statistics for PS and VM models are presented in Table 8.

Head Injury Status Predicting Cognitive Functioning

Executive Functions (EF). Next, structural equation modeling was used to evaluate primary hypotheses about the relationship between head injury status and the cognitive constructs of interest. EF was examined first, as this construct was

hypothesized to play a mediational role between head injury status and other cognitive functions. The path model (denoted H1) was constructed such that the dichotomous variable representing head injury status directly predicted the latent EF variable (established in model EF2). Overall fit for model H1 was good, $\chi^2(18, N = 126) = 18.22$, $p > .05$, GFI = .97, CFI = 1.00, RMSEA = .01. Head injury status was not a significant predictor of EF ($B = .05$, $p > .05$), indicating that primary hypotheses about the relationship between head injury status and EF could not be supported. Further, this finding eliminated EF from consideration as a possible mediator between head injury status and other variables of interest.

Processing Speed (PS). The relationship between head injury status and PS was evaluated next. The path model (denoted H2) was constructed such that the dichotomous variable representing head injury status directly predicted the latent PS variable (established in model C1). Overall fit for model H2 was good, $\chi^2(2, N = 126) = 1.41$, $p > .05$, GFI = .99, CFI = 1.00, RMSEA = .00. Head injury status significantly predicted PS ($B = -.28$, $p < .05$), indicating that OI group membership was related to poorer PS performance relative to MTBI group membership, a relationship opposite of that hypothesized. This finding was not affected by the inclusion of gender as a control in the model.

Verbal Memory (VM). A third path model of this type (denoted H3) was constructed with the dichotomous variable representing head injury status directly predicted the latent VM variable (established in model C1). Overall fit for model H3 was good, $\chi^2(2, N = 126) = .10$, $p > .05$, GFI = 1.00, CFI = 1.00, RMSEA = .00. Head injury status was not significantly predictive of VM ($B = -.01$, $p > .05$), indicating that direct-

effects hypotheses about the relationship between head injury status and VM could not be supported. Fit statistics for models in which head injury status predicts cognitive functioning are presented in Table 9.

Post-Concussive Symptoms (PCS) and Cognitive Functioning

Executive Functions (EF). Next, structural equation modeling was used to examine the relationships between PCS and the cognitive constructs of interest. This model (denoted PCS1) was constructed such that the manifest variable PCSC was permitted to correlate with the latent EF variable (established in model EF2). Overall fit for model PCS1 was good, $\chi^2(18, N = 126) = 18.52, p > .05$, GFI = .97, CFI = 1.00, RMSEA = .02. PCSC was not significantly related to EF ($r = .17, p > .05$).

Processing Speed (PS). The relationship between head injury status and PS was evaluated next. This model (denoted PCS2) was constructed such that the manifest variable PCSC was permitted to correlate with the latent PS variable (established in model C1). Overall fit for model PCS2 was good, $\chi^2(2, N = 126) = 1.50, p > .05$, GFI = .99, CFI = 1.00, RMSEA = .02. PCSC was not significantly related to PS ($r = .02, p > .05$).

Verbal Memory (VM). The third model of this type (denoted PCS3) was constructed such that the manifest variable PCSC was permitted to correlate with the latent VM variable (established in model C1). Overall fit for model PCS3 was good, $\chi^2(2, N = 126) = .49, p > .05$, GFI = 1.00, CFI = 1.00, RMSEA = .00. PCSC was significantly correlated with VM, $r = .20, p < .05$, indicating better memory performance for individuals with more severe symptoms reported on the PCSC. This finding was not affected by the

inclusion of gender as a control in the model. Fit statistics for models examining PCS and cognitive functioning are presented in Table 10.

Relationships between Head Injury, Symptom Variables, and EF

Hypotheses regarding the relationships between head injury status, EF, psychiatric symptoms, and PCS were evaluated by constructing a model (denoted SYM1) which included the latent EF variable (established in model EF2) as well as manifest variables BSI (representing psychiatric symptoms), PCSC, and head injury status. All latent and manifest variables were permitted to correlate. Overall fit for model SYM1 was good, $\chi^2(30, N = 126) = 39.74, p > .05$, GFI = .94, CFI = .94, RMSEA = .05. No variables in this model were significantly related to EF or head injury status. BSI was significantly correlated with PCSC ($r = .50, p < .01$), indicating that increased severity of symptoms on the PCSC was related to increased severity of symptoms on the BSI. The bivariate correlation of PCSC and BSI was also $r = .50, p < .01$, when examined using traditional statistical techniques. Consistent with the results of partial correlations, this finding was not affected by the inclusion of gender as a control in the SEM model.

Follow-up Data Analyses

Pearson correlations between PCSC and BSI subscales (shown in Table 11) were examined in order to more fully explore the relationship between these two measures. Notable relationships included the following: BSI Somatization and PCSC Dizziness ($r = .33, p < .01$); BSI Somatization and PCSC Fatigue ($r = .32, p < .01$); BSI Hostility and PCSC Irritability ($r = .46, p < .01$); BSI Obsessive-Compulsive and PCSC Memory ($r = .26, p < .01$); BSI Obsessive-Compulsive and PCSC Difficulty Concentrating ($r = .37, p < .01$); and PCSC Anxiety and the BSI Total ($r = .37, p < .01$). Examination of BSI and

PCSC items revealed significant content overlap in each of these areas. As a result, a BSI adjusted total was calculated, excluding items #2 (“dizziness”), #5 (“trouble remembering”), #6 (“feeling...irritated”), #36 (“trouble concentrating”), and #37 (“feeling weak”). Additionally, a PCSC adjusted total was calculated excluding the Anxiety subscale. The relationship between the BSI and PCSC totals adjusted in this manner was similar, $r = .46$, $p < .01$, suggesting that the relationship between these two variables was not due solely to similarities in item content.

In order to explore further the relationship demonstrated between PCSC total score and the latent Verbal Memory variable in model PCS3, correlations were also examined between PCSC subscales and a Verbal Memory composite variable computed as the average of standardized Immediate Memory, Short Delay, and Long Delay variables. Significant correlations were found between the Verbal Memory composite variable and Total score of the PCSC ($r = .19$, $p < .05$), as well as the Memory ($r = .19$, $p < .05$) and Visual Disturbances ($r = .18$, $p < .05$) PCSC subscales.

DISCUSSION

This study was conducted to investigate the effects of mild TBI on cognitive functioning and post-concussive symptomatology in the post-acute phase of recovery, with particular focus on the possible role of executive functions. Results failed to provide support that mild TBI leads to impairment in any cognitive domains examined (including executive functions, processing speed, and verbal memory). Although this finding is not unique in and of itself (see Bernstein & de Ruiter, 2000; Frencham, Fox, & Mayberry, 2005), methodological strengths of the study permit a greater degree of interpretation of these results than has been possible in previous studies with similar findings, and offer insights into some non-neurological factors that may be more relevant to an individual's long-term functioning after a mild TBI than the head injury itself.

Paradoxically, the orthopedic injury control group in this study performed worse on measures of processing speed than the mild TBI group (although these differences were rather small in magnitude). Differences between the groups in motor performance could not account for this finding, which appears to rule out the possibility that certain forms of injury (e.g., previously broken arms, wrists, or fingers) could have slowed manual task performance. Similarly, gender did not appear to explain this relationship. Rather, differences in injury characteristics that were found between these two groups suggest that the orthopedic injuries sustained by individuals in this sample may have had more impact than the mild TBIs in some aspects. For example, individuals with orthopedic injuries received medical treatment at a significantly higher rate than individuals with mild TBIs, which could be related to more severe injuries in general, or also simply to the greater perceived importance of the need for treatment with a broken

bone. In any case, the relatively greater attention given to a broken bone than to a mild TBI might be expected to be related to increased psychological impacts (such as distress) related to the injury. Further, individuals with broken bones may experience physical disability that altogether prevents them from engaging in certain activities for a period of time, potentially increasing the psychological impact relative to the short-term cognitive impairment that may follow a mild TBI.

Additionally, as reported previously by others (Sawchyn, Brulot, & Strauss, 2000; Suhr & Gunstad, 2002), no relationship was found between head injury status and purportedly “post-concussive” symptomatology within the time frame of the current study (3 months to 5 years post-injury). Instead, these self-reported symptoms appeared better explained by factors related to general emotional/psychiatric distress. Though substantial item content overlap existed between the measures of post-concussive symptoms and emotional/psychiatric symptoms utilized in this study, elimination of overlapping items and subscales did little to mitigate the relationship between these two variables. Similar results have also been found previously for other measures of these symptom domains, such as the Post-Concussional Index (PCI), the Beck Depression Inventory-2 (BDI-II), and the Beck Anxiety Inventory (BAI) (Suhr & Gunstad, 2002; Trahan, Ross, & Trahan, 2001). As such, the validity of scales such as the PCSC as measures of “post-concussive” symptoms is suspect, and clinicians and researchers would be well-advised to collect comprehensive information on the emotional functioning of individuals who report high levels of these types of symptoms.

Post-concussive symptoms were not significantly related to executive function or processing speed, findings which failed to provide support for hypotheses linking these

aspects of neuropsychological functioning to self-reported problems with attention and concentration. PCSC was significantly correlated with VM, indicating slightly better memory performance for individuals with more severe symptoms reported on the PCSC. Analysis of PCSC subscales revealed that this finding could be attributed in part to items on the PCSC relating to reports of memory problems and visual disturbances. Complaints of memory problems on the PCSC were therefore somewhat more common in individuals with better memory abilities; these findings are difficult to fully explain.

Though the results of this study failed to support primary hypotheses, strengths of the study's design give these findings substantially greater weight than many previously conducted studies. For example, few studies of mild TBI have had samples of the size available to this study ($n = 126$), drawn outside of a treatment setting, with a very low rate of involvement in litigation (2.4% overall). Additionally, no known previous studies of mild TBI have examined executive functions as comprehensively, using a large number of individual measures combined with latent modeling techniques of analysis to aid in data interpretation. Further, the inclusion of an injury control group is believed to have substantially mitigated the effects of a number of confounding factors related to generic effects of injury that are important to neuropsychological performance (see Dikmen & Levin, 1993; Satz et al., 1999). Additional factors that may affect cognition or symptom presentation related to injury type and treatment have also been highlighted for further investigation.

Many of these same methodological issues are also important when considering the study's limitations. Even though young adults are at a greatly increased risk for mild TBI relative to older adults (Cassidy et al., 2004; Bazarian et al., 2005), the restricted age

range of the present sample may limit generalizability of the present results to populations of older adults. Females were also over-represented in the current sample; although gender was not found to moderate any relationships of interest, this could have biased the results in subtler ways. Additionally, the inclusion of a second, non-injury control group might have allowed for more comprehensive evaluation of injury-related factors affecting neuropsychological performance. Finally, although the neuropsychological tasks used in this study are believed to have adequately assessed the cognitive domains of executive functioning, verbal memory, and processing speed, a number of cognitive domains (e.g., visuospatial ability, language) were not examined, and it is possible that an alternate battery of neuropsychological tasks might provide somewhat differing results.

In conclusion, clinically significant deficits in executive functions, processing speed, and verbal memory do not appear to be associated with mild TBI in the post-acute phase of recovery. Additionally, long-term post-concussive symptoms appear to be related to general emotional distress, rather than to the actual occurrence of a mild TBI. Valuable aspects of future studies in this area could include the examination of alternate cognitive domains, additional non-injury control groups, and more comprehensive evaluations of non-neurological factors related to neuropsychological performance following injury.

Figure 1. Depiction of Hypothesized Variable Relationships

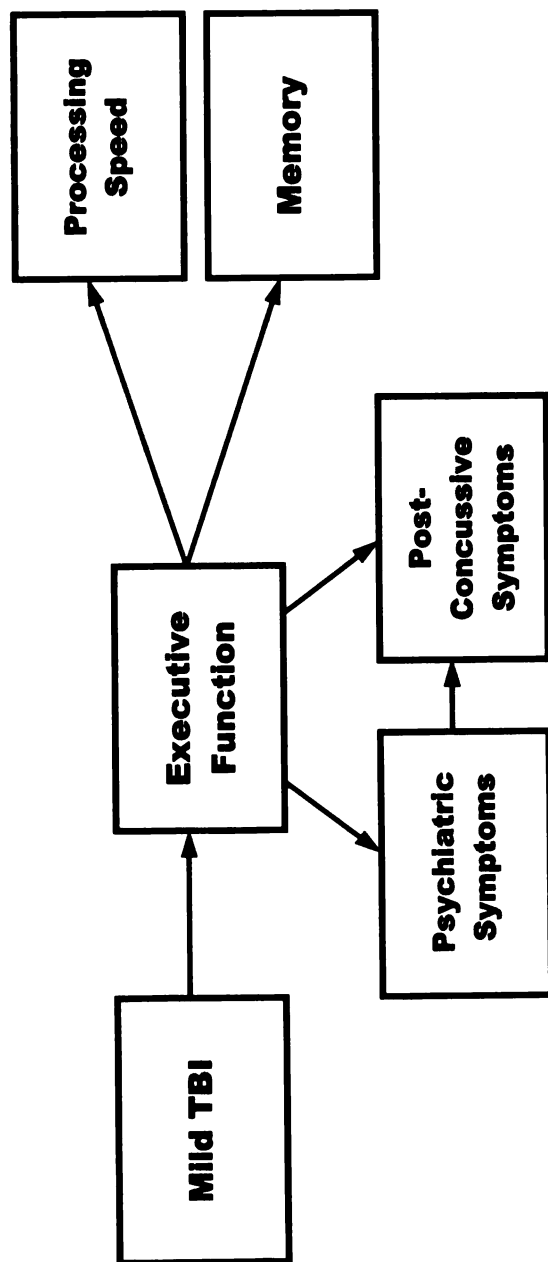


Table 1. Demographic characteristics of screening sample

(Total N = 3,347)	Valid N	Screening Sample
Mean Age in Years (SD)	3289	19.49 (1.78)
Mean Years Education (SD)	3325	13.02 (1.02)
% Female	3337	70.6%
% Hispanic or Latino/a	3337	1.9%
% Native American	3337	0.3%
% Asian	3337	4.9%
% African American / Black	3337	7.6%
% Native Hawaiian / Pacific Islander	3337	0.3%
% Caucasian / White	3337	81.4%
% “Other” Ethnicity / Race	3337	3.3%

Table 2. Constructs and indicators¹.

Construct	Measures (Indicators)
Executive Function	WCST (Perseverative Errors); CWIT (Trial 3 Total Time); TMT (Trial 4 Total Time); VFT (Letter, Category, and Switching scores); OS (Total word errors); RS (Total arrow errors)
Processing Speed	LC (Total Score); NC (Total Score); PC (Total Score)
Verbal Memory	CVLT-II (Immediate Recall total, Short Delayed Recall total, Long Delay Recall total)
Post-Concussive Symptoms	PCSC (Total score)
Psychiatric Symptoms	BSI (Total score)
Intelligence	Estimated FSIQ (WAIS-III Vocabulary and Block Design)

¹ WCST = Wisconsin Card Sorting Test, CWIT = D-KEFS Color-Word Interference Test, TMT = D-KEFS Trail Making Test, VFT = D-KEFS Verbal Fluency Test, OS = Operation Span, RS = Rotation Span; SDMT = Symbol Digit Modalities Test, LC = Letter Comparison, NC = Number Comparison; PC = Pattern Comparison, CVLT-II = California Verbal Learning Test 2, PCSC = Post-Concussion Syndrome Checklist, BSI = Brief Symptom Inventory.

Table 3. Executive Function Indicators and Theoretical Components ².

Measure	Indicator(s)	Theoretical EF Component(s)
WCST	Perseverative Errors	Abstract Reasoning, Cognitive Flexibility
CWIT	Trial 3 (Inhibition) Total Time	Inhibition, Cognitive Flexibility
TMT	Trial 4 (Number-Letter Switching) Total Time	Cognitive Flexibility
VFT	Letter, Category, and Switching scores	Behavioral Initiation / Fluency, Cognitive Flexibility
OS/RS	Total Word/Arrow Errors	Working Memory Capacity

² WCST = Wisconsin Card Sorting Test, CWIT = D-KEFS Color-Word Interference Test, TMT = D-KEFS Trail Making Test, VFT = D-KEFS Verbal Fluency Test, OS = Operation Span, RS = Rotation Span

Table 4. Demographic Characteristics by Participant Group

	MTBI Group	OI Group	<i>p</i> ¹
N	63	63	--
Mean Age in Years (SD)	19.19 (1.06)	19.10 (1.17)	<i>ns</i>
Mean Years Education (SD)	12.86 (.91)	12.86 (.91)	<i>ns</i>
Mean Estimated FSIQ (SD)	113.32 (12.49)	111.90 (9.21)	<i>ns</i>
Mean Alcoholic Drinks Per Month (SD)	30.44 (30.31)	22.72 (25.97)	.06
Mean Days Per Month Drugs Used (SD)	1.11 (3.47)	.63 (1.55)	.06
% Female	63.5%	69.8%	<i>ns</i>
<u>Ethnicity / Race</u> ²	MTBI Group	OI Group	
% Hispanic or Latino/a	3.2%	0.0%	
% Native American	0.0%	0.0%	
% Asian	4.8%	0.0%	
% African American / Black	3.2%	4.8%	
% Native Hawaiian / Pacific Islander	0.0%	0.0%	
% Caucasian / White	85.7%	95.2%	
% "Other" Ethnicity / Race	3.2%	0.0%	

¹ Results of independent-samples T-test.

² Chi-square statistic was non-significant for the overall comparison.

Table 5. Injury Characteristics by Participant Group

	MTBI Group	OI Group	<i>p</i>
% Received Treatment for Injury	73.0%	92.1%	<.01
% Hospitalized for Injury	39.7%	52.4%	<i>ns</i>
% Involved in Litigation	4.8%	0%	<.01

<u>Cause of Injury¹</u>	MTBI Group	OI Group
% Motor Vehicle Accidents	14.3%	3.2%
% Sports Participation	50.8%	74.6%
% Accidental Falls	22.2%	11.1%
% Interpersonal Violence	1.6%	0.00%
% Other Cause of Injury	11.1%	11.1%

¹ Chi-square statistic was significant at the $p < .05$ level for the overall comparison.

Table 6. Descriptive Statistics and T-tests of Cognitive Variables¹

Variable	df	MTBI Group Mean (SD)	OI Group Mean (SD)	Mean Difference ^a	<i>t</i> ^b	<i>d</i>
WCST Perseverative Errors	124	6.57 (3.41)	6.25 (3.03)	.32	.55	.10
TMT Trial 4 Time Total	124	54.00 (12.61)	54.31 (14.27)	-.31	-.13	-.02
TMT Trial 5 Time Total	124	21.81 (5.61)	22.06 (5.40)	-.26	-.26	-.05
VFT Trial 1 Correct	124	37.78 (8.86)	38.84 (10.91)	-1.06	-.60	-.11
VFT Trial 2 Correct	124	44.08 (7.08)	42.67 (6.81)	1.41	1.14	.20
VFT Trial 3 Switches Total	124	13.17 (2.84)	12.98 (2.70)	.18	.37	.07
OS Word Errors Total	124	20.03 (6.90)	19.16 (5.47)	.87	.79	.14
RS Arrow Errors Total	124	14.49 (6.72)	14.38 (7.16)	.11	.09	.02
CWIT Trial 3 Time Total	124	45.22 (8.54)	45.05 (8.42)	.17	.11	.02
CVLT Immediate Recall Total	124	54.31 (8.98)	54.05 (7.98)	.26	.17	.03
CVLT SD Free Total	124	11.71 (2.54)	11.66 (2.49)	.04	.10	.02
CVLT LD Free Total	124	11.70 (2.51)	11.71 (2.74)	-.02	-.04	-.01
LC Total	124	23.73 (5.16)	21.81 (3.99)	1.92	2.34*	.42
NC Total	124	20.32 (2.86)	19.82 (2.98)	.60	.87	.16
PC Total	124	42.38 (7.67)	39.55 (5.39)	2.83	2.40*	.43

¹ WCST = Wisconsin Card Sorting Test, CWIT = D-KEFS Color-Word Interference Test, TMT = D-KEFS Trail Making Test, VFT = D-KEFS Verbal Fluency Test, OS = Operation Span, RS = Rotation Span, LC = Letter Comparison, NC = Number Comparison, PC = Pattern Comparison, CVLT-II = California Verbal Learning Test-2

^a MTBI Group Mean – OI Group Mean

^b *t* value of independent-samples *t*-test

* Statistically significant at the ($p < .05$) level (2-tailed)

Table 7. Descriptive Statistics and T-tests of Self-Reported Symptom Variables

Variable	<i>df</i>	<u>MTBI</u> <u>Group</u> Mean (SD)	<u>OI</u> <u>Group</u> Mean (SD)	Mean Difference ^a	<i>t</i> ^b	<i>d</i>
BSI Somatization	124	.40 (.48)	.32 (.38)	.08	1.05	.19
BSI Obsessive-Compulsive	124	1.08 (.77)	1.16 (.66)	-.08	-.60	-.11
BSI Interpersonal Sensitivity	124	.54 (.61)	.61 (.59)	-.08	-.71	-.13
BSI Depression	124	.44 (.54)	.43 (.46)	.01	.12	.02
BSI Anxiety	124	.43 (.45)	.49 (.42)	-.06	-.75	-.13
BSI Hostility	124	.52 (.57)	.44 (.44)	.09	.94	.17
BSI Phobic Anxiety	124	.15 (.34)	.08 (.24)	.07	1.29	.23
BSI Paranoid Ideation	124	.48 (.54)	.52 (.52)	-.04	-.37	-.07
BSI Psychoticism	124	.32 (.51)	.32 (.38)	.01	.08	.01
BSI Total (GSI)	124	.49 (.41)	.49 (.32)	.01	.12	.02
PCSC Total	124	62.03 (12.77)	60.02 (12.75)	2.01	.89	.16

* Statistically significant at the ($p < .05$) level (2-tailed)

^a MTBI Group Mean – OI Group Mean

^b *t* value of independent-samples t-test

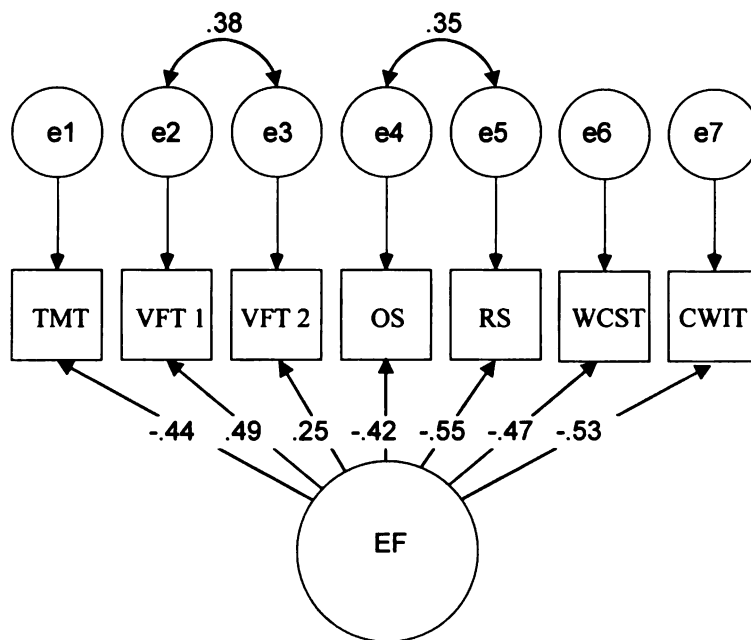


Figure 2. Freely-estimated model of EF factor structure (Model EF₂).

Standardized values shown. All factor loadings significant at the $p < .05$ level (2-tailed).

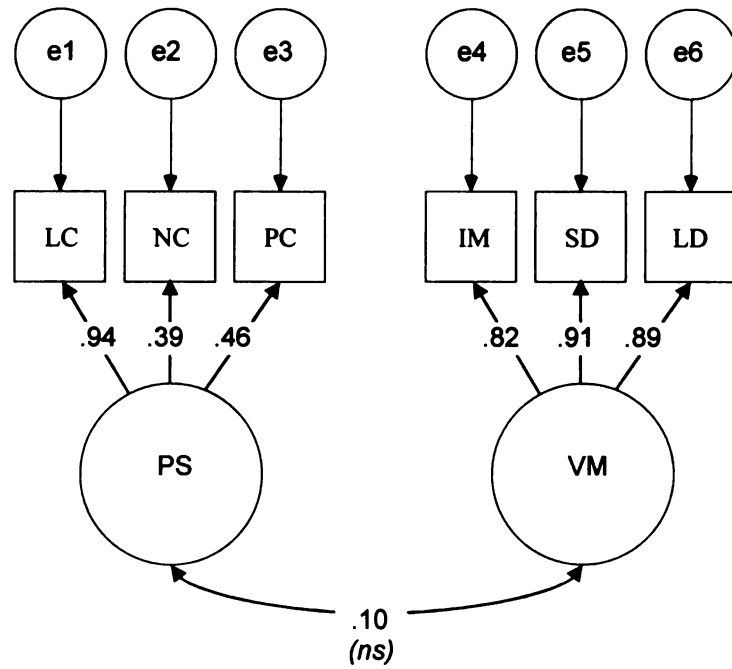


Figure 3. Freely-estimated model of PS and VM factor structure (Model C₁).
Standardized values shown. All factor loadings significant at the $p < .05$ level (2-tailed).

Table 8. Confirmatory Factor Analysis of Cognitive Variables

Model	χ^2	<i>df</i>	<i>p</i>	GFI	CFI	RMSEA
EF ₁	15.91	16	> .05	.97	1.00	.00
EF ₂	14.59	12	> .05	.97	.98	.04
EF ₃	34.54	24	> .05	.93	.91	.06
EF ₄	37.79	30	> .05	.93	.95	.05
C ₁	7.41	8	> .05	.98	1.00	.00
C ₂	11.60	16	> .05	.97	1.00	.00
C ₃	17.29	20	> .05	.96	1.00	.00

Note. $n = 126$ for all analyses; GFI = Goodness-of-fit index; CFI = Comparative fit index; RMSEA = Root Mean Square Error of Approximation.

Table 9. Models of Head Injury Status Predicting Cognitive Functioning

Model	χ^2	<i>df</i>	<i>p</i>	GFI	CFI	RMSEA
H ₁	18.22	18	> .05	.97	1.00	.01
H ₂	1.41	2	> .05	.99	1.00	.00
H ₃	.10	2	> .05	1.00	1.00	.00

Note. $n = 126$ for all analyses; GFI = Goodness-of-fit index; CFI = Comparative fit index; RMSEA = Root Mean Square Error of Approximation.

Table 10. Models of Post-Concussive Symptoms and Emotional/Psychiatric Functioning

Model	χ^2	<i>df</i>	<i>p</i>	GFI	CFI	RMSEA
PCS ₁	18.52	18	> .05	.97	1.00	.02
PCS ₂	1.50	2	> .05	.99	1.00	.02
PCS ₃	.49	2	> .05	1.00	1.00	.00
SYM ₁	39.74	30	> .05	.94	.94	.05

Note. n = 126 for all analyses; GFI = Goodness-of-fit index; CFI = Comparative fit index; RMSEA = Root Mean Square Error of Approximation.

Table 11. Correlations of Selected Symptom Variables^{1,2}

	PCSC HEAD	PCSC DIZ	PCSC IRR	PCSC MEM	PCSC CONC	PCSC FATG	PCSC VIS	PCSC NOI	PCSC JUDG	PCSC ANX	PCSC Total	BSI Total
BSI SOM	.21*	.33**	.21*	.21*	.25**	.32**	.19*	.10	.26**	.22*	.44**	.64**
BSI O-C	.14	.22*	.26**	.26**	.37**	.21*	.07	.16	.26**	.29**	.43**	.77**
BSI IS	.07	.22*	.22*	.20*	.19*	.24**	.15	.09	.26**	.34**	.38**	.84**
BSI DEP	.17	.27**	.18*	.22*	.26**	.23*	.10	.08	.22*	.29**	.39**	.82**
BSI ANX	.21*	.25**	.30**	.25**	.30**	.27**	.03	.23*	.26**	.33**	.48**	.81**
BSI HOS	.18	.13	.46**	.12	.19*	.23**	.18*	.23*	.16	.29**	.42**	.71**
BSI PHO	.17	.25**	.23**	.09	.29**	.22*	.12	.03	.31**	.36**	.40**	.64**
BSI PAR	.08	.01	.24**	.19*	.07	.08	.15	.16	.07	.15	.23**	.66**
BSI PSY	.15	.24**	.23**	.12	.23*	.19*	.10	-.01	.10	.29**	.32**	.76**
BSI Total	.18*	.28**	.32**	.26**	.32**	.28**	.16	.16	.29**	.37**	.50**	1
PCSC Total	.51**	.46**	.61**	.45**	.61**	.65**	.42**	.43**	.52**	.54**	1	.50**

¹ PCSC = Post-Concussion Syndrome Checklist; HEAD = Headache; DIZ = Dizziness; IRR = Irritability; MEM = Memory Problems; CONC = Difficulty Concentrating; FATG = Fatigue; VIS = Visual Disturbances; NOI = Aggravation by Noise; JUDG = Judgment Problems; ANX = Anxiety; BSI = Brief Symptom Inventory; SOM = Somatization; O-C = Obsessive-Compulsive; IS = Interpersonal Sensitivity; DEP = Depression; ANX = Anxiety; HOS = Hostility; PAR = Paranoid Ideation; PSY = Psychoticism

² $n = 126$ for all analyses shown

* Statistically significant at the ($p < .05$) level (2-tailed)

** Statistically significant at the ($p < .01$) level (2-tailed)

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