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A Multigroup Analysis of the Psychological Factors that Contribute to Persisting Working Attention Problems in Mild Traumatic Brain Injury and Chronic Pain

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A Multigroup Analysis of the Psychological Factors that Contribute to Persisting Working
Attention Problems in Mild Traumatic Brain Injury and Chronic Pain

A Dissertation

Submitted to the Graduate Faculty of the
University of New Orleans
in partial fulfillment of the
requirements for the degree of

Doctor of Philosophy
in
Applied Biopsychology

By

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ABSTRACT

A significant subset of mild traumatic brain injury (mild TBI) and chronic pain (CP) patients report, and sometimes show objective evidence of, persisting cognitive problems. Despite differences in injury mechanisms, there is considerable overlap in the types of persisting cognitive symptoms that are reported by the two populations. Psychogenic, rather than physiogenic, factors are thought to play an important role in the maintenance of these persisting symptoms. The current investigation examined the contributions somatization, depression, and anxiety had on an objective measure of “working attention.” In order to best elucidate the influences these psychological factors had on attentional performance, only individuals who passed well-validated and popular indicators of cognitive and self-report validity were included in the study. Two hundred and forty-nine individuals ($n = 116$ TBI; $n = 133$ CP) met the inclusionary criteria for the study. Psychological factors were assessed using Scales 1 (Hypochondriasis), 2 (Depression), 3 (Hysteria), and 7 (Psychasthenia) of the Minnesota Multiphasic Personality Inventory-II. “Working attention” was measured using the demographically-adjusted T-scores for the Working Memory and Processing Speed Indexes of the Wechsler Adult Intelligence Scale- 3. Results indicated that a high rate of psychological complications was observed in the mild TBI and CP groups but not the moderate-severe traumatic brain injury (M/S TBI) comparison group. Analysis indicated that psychological elevations were not significantly related to spontaneously-reported symptoms or working attention deficits for the mild TBI group but were for the CP and M/S TBI groups. The current results are important for understanding the psychological

complications that may occur in individuals exhibiting persisting cognitive problems in these clinical populations.

Keywords: traumatic brain injury, chronic pain, psychological factors, MMPI-2, working memory, processing speed

CHAPTER 1

In the United States, persisting cognitive, affective, and physical symptoms after head and spinal injuries can have significant effects on a person's functional outcome. These injuries lead to a substantial number of lost workdays and productivity, account for a significant proportion of worker's compensation claims, and cost the US healthcare system hundreds of billions of dollars in treatment (Guo et al., 1995; Guo et al., 1999; Meyers & Diep, 2000; Nicholson & Martelli, 2004). Because of this, a focus of research has been to identify the psychosocial factors that are thought to contribute to the "risk" a person may have for developing persisting symptoms.

Traumatic Brain Injury

Prevalence. Brain injuries are one of the leading causes of mortality and morbidity in the world and can have a serious impact on an individual's behavioral, psychological, and cognitive functioning. In the United States alone, around 1.5 million people sustain a brain injury each year; 250,000 to 290,000 are hospitalized; approximately 50,000 die; and 125,000 are still considered disabled after one year (Dikmen et al., 2009; Scherer & Madison, 2005). Of those brain injuries that present to the hospital, between 50% (Scherer & Madison, 2005) and 90% (Larrabee, 2005; Rose, 2005) are mild in nature. Based on incidence data from 1995, the Centers for Disease Control and Prevention estimated that the total lifetime cost (direct and indirect costs) for all TBI to be around 60 billion dollars, \$16.7 of which is allocated to the treatment of mild brain injuries alone (Thurman, 2001). Given these statistics, it is important to study outcome in these populations, particularly mild TBI.

Injury severity classification. According to the current systems used to classify brain injury severity, head injury severity is not defined in terms of outcome but rather the physiological symptoms that occur during and immediately following the injury (Alexander, 1995; Arciniegas, Anderson, Topkoff, & McAllister, 2005; Binder, 1997; Ruff, 2005). These acute injury characteristics include: duration of coma (if any), alterations of consciousness, length of post-traumatic amnesia period, objective findings on standard neuroimaging techniques, and whether focal neurological signs are present (Arciniegas et al., 2005; Bernstein, 1999; Dikmen, Machamer, Winn, & Temkin, 1995). These classification systems are used to identify whether someone has sustained a mild, moderate, or severe TBI and are beneficial in providing insight regarding the symptom and cognitive recovery a person will have. Appendix A provides a summary of the acute injury characteristics that are utilized in the most current severity classification systems.

In viewing these systems, it is apparent that there is a general consensus on how to classify moderate and severe TBIs, mainly because the injury characteristics are more easily identifiable and objective neuropathological findings are usually present. On the other hand, the lack of gross objective findings (Miller, 2001; Satz et al., 1999) and quick symptom resolution make it hard to establish whether an individual has sustained a mild brain injury or not (Ruff et al. 2009). Moreover, a number of individuals sustaining a concussion do not seek immediate medical attention, if any, so an over-reliance on self-report occurs when and if the patient presents with symptoms at a later point in time (McCrae, 2008).

As such, there has been some debate over what should be considered a mild TBI. In 1993, the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (ACRM; Mild Traumatic Brain Injury Committee, 1993) developed criteria of what mild TBI encompasses and has been widely used in subsequent empirical research on mild TBI (Ruff et al., 2009). In 2004, the World Health Organization (WHO) Collaborating Centre for Neurotrauma Task Force on Mild Traumatic Injury (Carroll, Cassidy, Holm, Kraus, & Coranado, 2004) provided a revision of the mild TBI criteria established by the American Congress of Rehabilitation Medicine that was more explicit in its description (see Appendix A). Regardless of the classification system used, each stress the importance of using a “multidimensional definition that incorporates information on the biomechanics, acute injury characteristics, and clinical course to assist clinicians in making the most accurate diagnosis of MILD TBI” (McCrea et al., 2009, p. 1369).

Recently, a “mild-complicated” severity level has been identified and studied in research. This designation is used for those individuals meeting the mild TBI criteria but who have positive neuroimaging findings. Research has shown that this group often performs similarly to moderate TBI patients on measures of neuropsychological functioning (Iverson, 2005; Williams, Levin, & Eisenberg, 1990).

Expected cognitive outcome following TBI. A number of reviews and meta-analyses have been conducted over the years that have provided detailed insight regarding the relationship between TBI severity and neuropsychological outcome. Dikmen and colleagues (1995) were some of the first to conduct a prospective study

examining neuropsychological performance as a function of injury severity. Their total sample consisted of 436 patients that ranged in head injury severity based on GCS scores in the emergency room, neuroimaging findings, functional findings, and time to follow commands. A sample of 121 non-head injury trauma controls was used for comparison. Each of the patients were administered a variety of neuropsychological tests, testing a variety of cognitive domains, approximately one year post-injury. The results showed that the head injury group performed significantly worse on all neuropsychological measures compared to the trauma control group and that the extent of the neuropsychological impairment was a function of injury severity. While these findings were seminal to the outcome literature, one limitation is worth mentioning. Although one-third (36%) of the Dikmen et al. (1995) study consisted of mild TBI patients, one of the minimum criteria for inclusion in the study was that the head injury be serious enough for hospitalization. This would put a majority of this subsample at the more “severe” end of the mild TBI category and thus, they may not have been representative of types of mild TBI patients that are typically evaluated. A follow-up study using less severe mild TBI criteria and patients at least six months post-injury; however, yielded essentially the same results as the earlier study (Dikmen et al., 2009) as did an independent review conducted by Schretlen and Shapiro (2003).

Meta-analyses have also been conducted using only mild TBI samples. One of the first meta-analyses conducted using only mild TBI patients was conducted by Binder, Rohling, and Larrabee (1997). Their meta-analysis consisted of 314 mild TBI patients seen at least three months after their injuries and 308 control subjects. The overall effect size that was calculated was not significantly different from zero ($g = .07$);

however, when individual cognitive domains were examined, a small but significant effect for attention was found ($g = .17$).

Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg (2005) conducted a meta-analysis on the effect of mild TBI across nine cognitive domains in a sample of 1463 mild TBI patients and 1191 control patients. While initial analyses showed significant effects of mild TBI across all domains ($d = .54$), especially attention ($d = .47$), follow-up moderating analyses showed that when time since injury (greater than 90 days) and litigation status were taken into account, no significant effects were observed. A subsequent large-scale review conducted by Carroll et al. (2004) and meta-analysis conducted by Frenchman, Fox, & Maybery (2005) also showed a significant moderating effect of time since injury on neuropsychological outcome.

Altogether, the above-reviewed studies consistently demonstrated that residual cognitive impairment can occur post-acutely but that the magnitude of the impairment corresponds to injury severity. Within mild TBI, larger effects have been observed in certain domains with attention being the largest. However, these effect sizes typically decrease with time since injury and are also dependent on the sample selection criteria for the studies (i.e. larger effects are observed for clinic-based or litigation samples as opposed to population-based samples) (Belanger & Vanderploeg, 2005).

Despite the consistency in these findings; however, it should be mentioned that meta-analyses represent an “aggregation of effect sizes from multiple comparison groups across multiple studies, but can obscure small subgroup or individual effects” (Iverson, 2010; Iverson, Brooks, Collins, & Lovell, 2006 as cited in McCrea et al., 2009, p. 1374). In support of this, there are a number of smaller-scale studies that suggest

persisting cognitive symptoms do occur in mild TBI patients, particularly in areas of attention and processing speed (refer to Attention section below). Persisting symptoms in mild TBI continue to be a large health care problem, and as such, there needs to be continued research to better elucidate the causal factors of them.

Post-Concussion Syndrome

The term Post-Concussion Syndrome (PCS) has been used to refer to a constellation of symptoms reported in a subset of individuals having sustained a mild TBI (Alexander, 1995; Belanger et al., 2005; Binder, 1986, 1997; Smith-Seemiller, Fow, Kant, & Franzen, 2003; Wood, 2005). Specifically, the syndrome consists of symptoms that represent three main functional domains: somatic complaints (e.g., headaches, nausea, vomiting, blurred vision, dizziness, fatigability); cognitive functioning (e.g., problems with working memory, poor attention and concentration, reduced processing speed); and emotional functioning (e.g., irritability, angry outbursts, depression, and anxiety) (Bernstein, 1999; Binder, 1997; McCrea, Iverson, McCallister, Hammeke, Powell, Barry & Kelly, 2009; McAllister & Arciniegas, 2002; Ryan & Warden, 2003; Satz et al., 1999; Smith-Seemiller et al., 2003; Williams, Potter, & Ryland, 2010)

When examining the literature to-date regarding the etiological mechanisms of post-concussive symptomatology, a dichotomy between physiogenesis and psychogenesis has emerged (Lishman, 1988). The general consensus in the literature is that Post Concussion Syndrome can be thought of in two stages - an early post-concussive period and a late post-concussive period (also known as Persistent Post-Concussion Syndrome [PPCS]); (Alexander, 1995; Jacobson, 1995). Early post-concussive symptoms are commonly experienced after a brain injury, are typically

somatic and cognitive in nature like the symptoms described above (Alexander, 1995; Binder, 1997; Jacobson, 1995; Macleod, 2010), and are thought to be the result of the neuropathologic and neurophysiologic changes that occur as a result of the injury to the brain. Nonetheless, these symptoms are short-lived and typically resolve within a few weeks to months after the injury (Binder, 1997; Dikmen et al., 1994; Dikmen et al., 1995; Macleod, 2010; McCrae, 2008; McCrae et al., 2009; Rose, 2005; Williams et al., 2010; Wood, 2004) once the pathophysiological changes associated with mild brain injuries resolve (Alexander, 1995; Binder, 1997; Gaetz, 2004; Iverson, 2005).

Despite this fact, a relatively small but clinically significant group of patients continue to have persisting symptoms beyond what is considered the “normal” recovery time (Alexander, 1995; Karzmark, Hall, & Englander, 1995; Ingebrigsten et al., 1998; Macleod, 2010; Ryan & Warden, 2003; Smith-Seemiller et al., 2003; Wood, 2004). Research over the past two decades has attempted to identify the non-pathophysiologic etiological mechanisms (i.e., psychogenic) that are contributing to symptom maintenance but this research has been complex for a number of reasons.

Non-specificity of Post-Concussion Syndrome. Studying the etiological mechanisms driving persisting post-concussive symptoms has been complicated by a number of factors. First, there is considerable variability in prevalence estimates of PCS. Current prevalence estimates range from about three percent (McCrae et al., 2009) to upwards of 40 percent of mild TBI patients (Alexander, 1995; Evered, Ruff, Baldo, & Isomura, 2003; Gunstad & Suhr, 2004). These variable estimates are in part due to biased sampling because a) approximately 25 percent of individuals who sustain a concussion do not seek treatment and are therefore not taken into account in

prevalence estimates and b) of those that sustain concussions, only a subset of those patients go on to have PCS symptoms (McCrae, 2008).

Estimating the prevalence of PCS is also made difficult because of varying operationalizations of what constitutes PCS (e.g., how many symptoms need to be endorsed and the timeframe required in order to be considered persistent). In an attempt to standardize diagnostic criteria for the syndrome, the Diagnostic and Statistical Manual – 4th edition, Text Revised (American Psychiatric Association, 2000) lists criteria for Post Concussion Disorder (see Appendix B for the criteria for PCD). An examination of the criteria; however, shows that only those individuals with more severe concussions who have experienced a loss of consciousness would potentially qualify for a diagnosis of PCD. Based on recent research showing that loss of consciousness does not take place in a majority of concussions, a significant number of individuals would not even qualify for the condition based on these criteria (McCrae, 2008).

In addition to the above-stated problems, there does not appear to be a uniform method of measurement for PCS symptoms (Gasquoin, 2000; Ruff, 2005). Research has shown a continued over-reliance on patient self-report, semi-structured interviews, and checklists in order to diagnosis PCS and these subjective findings are often not corroborated by objective evidence from neuropsychological measures (Greiffenstein, 2009; Larrabee, 2005; McCrae, 2008). Furthermore, thorough investigations into other factors (i.e. psychosocial) or a combination of factors that may be contributing to persisting problems are often not conducted (McCrae, 2008) and so it becomes difficult to determine the factors that may cause, mimic, or maintain these symptoms (Lange, Iverson, Brooks, & Rennison, 2010).

Finally, many of the affective, somatic, and cognitive symptoms that comprise the diagnostic criteria and that are self-reported by patients have been found to be commonly reported in non-head injury related populations. These include: general healthy populations (Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992; Iverson & Lange, 2003; Iverson, King, Scott, & Adams, 2001; Lees-Haley, Fox, & Courtney, 2001; Mittenberg, DiGiulio, Perrin, & Bass, 1992; Wang, Chan, & Deng, 2006), healthy individuals asked to simulate a variety of conditions (Gunstad & Suhr, 2002), individuals with psychological conditions (e.g., anxiety or depression; Fox, Lees-Haley, Earnest, & Dolezal-Wood, 1995; Iverson, 2006; Suhr & Gunstad, 2002; Trahan, Ross, & Trahan, 2001), and individuals with disorders characterized by medically unexplained symptoms (Binder, 2005). Individuals with chronic pain conditions, in particular, report many of the same cognitive symptoms as those observed in individuals with persistent post-concussive problems. These problems include (but are not limited to): attention, concentration, information processing, short-term memory/forgetfulness (Eccleston, 1994, 1995; Gasquione, 2000; Iverson & McCracken, 1997; Lees-Haley, Fox, & Courtney, 2001; McCracken & Iverson, 2001; Martelli, Grayson, & Zasler, 1999; Nicholson, 2000; Satz, Alfano, Light, Morgenstern, Zaucha, Asarnow, et al. 1999; Schnurr & MacDonald, 1995; Smith-Seemiller et al., 2003).

In order to illustrate the non-specific nature of PCS symptoms, Appendix C presents the self-reported PCS symptoms from a variety of the sample populations described above. This table is broken down by the DSM-IV TR diagnostic criteria for PCD and represents the symptoms that are most endorsed by patients in the studies mentioned above. Examination of the table shows that a number of non-TBI related

conditions endorse PCS-symptoms at similar, if not higher, rates than TBI patients. These individuals often meet diagnostic criteria for PCS (Garden & Sullivan, 2010).

Summary. It is currently well-accepted that the initial symptoms of PCS are directly influenced by the acute neurological effects of the injury whereas persisting symptoms are maintained by non-injury-related factors. As such, it is important to evaluate the psychosocial factors (e.g., psychological, social, and motivational) that contribute to persisting symptoms. Given PCS's symptom non-specificity, comparing a group of mild TBI patients with persisting symptoms with a non-head injury group also experiencing similar persisting cognitive symptoms will help elucidate the specific psychosocial factors that are likely contributing to the persistent symptomatology.

Chronic Pain

Prevalence. According to the International Association for the Study of Pain, pain is defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage” (Mersky & Bogduk, 1994 as cited in Turk, Robinson, Loeser, Covington, & Lippe, 2001, p. 556). Pain is typically classified according to the duration of the pain (acute versus chronic), cause (e.g., malignancy, trauma), anatomic region (e.g., cervical, thoracic, lumbar, peripheral areas), and mechanism (e.g., nociceptive, inflammatory, neuropathic) (Pappagallo & Werner, 2008). Chronic pain occurs when pain persists beyond the normal recovery time that it takes for tissue to heal from an injury, which is typically around three months (Pappagallo & Werner, 2008), although some researchers suggest six months if the pain transitions from acute to chronic (Hart, Martelli, & Zasler, 2000; Tunks, Crook, & Weir, 2008). Chronic pain is more likely to be characterized by

relatively ambiguous neuroanatomic pathways that can explain the somatic effects, a usual lack of tissue damage, a decrease or avoidance of activities, use of medication and services that prove to be minimally helpful in the reduction of pain, and significant behavioral or emotional changes (Hart et al., 2000).

Just as with TBI, chronic pain is a major cause of morbidity and significantly impacts society in both direct and indirect ways. According to the Joint Commission on the Accreditation of Healthcare Organizations, approximately 33 percent of Americans will experience chronic pain at some point in time in their lives and more than half of all Americans report experiencing current or chronic pain within the last year (Porter-Moffitt, Gatchel, Robinson, Deschner, Posamentier, Polatin, et al., 2006). As such, it is one of the most common causes of disability, partially or totally disabling around 50 million people each year (American Pain Society). Between 90 (Turk, 2002) and 125 billion dollars (Meyers & Diep, 2000; Nicholson & Martelli, 2004) is expended annually on healthcare in the US to diagnose and treat chronic pain. Other costs that cannot be directly measured are also accrued. Chronic pain accounts for a significant amount of lost work productivity, time off of work, and income replacement in the United States and accounts for approximately 25 percent of all workers compensation claims filed and 33 percent of total medical compensation costs (Guo et al., 1995; Guo, Tanaka, Halperin, & Cameron, 1999). It also has a significant impact on an individual's everyday functioning and can severely limit one's social interactions and ability to accomplish non-work related tasks (Gatchel et al., 2008). Given these statistics, research examining the factors, both pathophysiological and psychosocial, that contribute to chronic pain has become increasingly important.

Attention

As indicated above, both mild TBI and CP frequently report persisting problems that fall in the attentional domain. Persisting attention problems are especially problematic and impede on recovery of function as attention subserves many other cognitive processes such as learning and memory.

Attention problems in mild TBI. Within the mild TBI meta-analytic literature, inconsistencies have been observed regarding the extent of the attention problems that exist. Some report no differences between mild TBI patients and controls post-acutely while others have found small but significant effect sizes. One issue with the meta-analytic literature; however, is that data from a variety of different attention measures is combined to calculate these effect sizes. As discussed below (see “Problems with Existing Attention Studies”), differing operationalizations of attention and its subtypes and use of different measures may cause an underestimation of the types of problems that exist. Alternately, aggregating group statistics from multiple measures may diminish the extent of symptoms in individuals or subgroups (Iverson, 2010). Therefore, it is also important to examine smaller-scale studies examining attention problems in mild TBI in hopes of better understanding the specific problems that persist.

Smaller-scale studies investigating the cognitive problems in mild TBI patients have been conducted in two main ways. The first involves estimating the prevalence of persisting symptoms that are reported by the patient using self-report questionnaires such as the Rivermead Post-Concussive Symptom Questionnaire. For the sake of brevity, the reader is referred to Appendix C for a summary of the most frequently reported cognitive symptoms in mild TBI patients. The second method of examination

involves examining the performance of mild TBI patients on objective neuropsychological measures of attention.

Chan (2001) provides an excellent review of some of the studies that have been conducted examining performance on various types of attention in brain injury patients, particularly in individuals who are at least four weeks post-injury. Along with studies not reviewed by Chan, mild TBI patients have exhibited impairment in selective attention (Bohnen, Twijnstra, & Jolles, 1993; Chan, 2002; Kwok, Lee, Leung, & Poon, 2008; Mathias, Biall, & Bigler, 2004); sustained attention (Bohnen, Jolle, Twijnstra, Mellink, & Wijnen, 1995; Malojcic, Mubrin, Coric, Susnic, & Spilich, 2008; Mathias et al., 2004); divided attention (Chan, 2002; Kwok et al., 2008); attention control processing (Chan, 2002); speed of information processing (Cicerone, 1996; Mathias et al., 2004; Tiersky, Cicerone, Natelson, & DeLuca, 1998); and working memory (Malojcic et al., 2008). Other studies which did not specify the types of attention being analyzed also showed persisting symptoms in this population (Cicerone & Azulay, 2002; Raskin, Mateer, & Tweeten, 1998; Tiersky et al., 1998).

Attention problems in CP. There are no known meta-analyses that have been conducted summarizing the extent of attention problems in CP populations; however, a number of commentaries and reviews have been published that provide an overview of the most frequently experienced cognitive problems in CP (see Krietler & Niev, 2007; Hart, Martelli, & Zasler, 2000; Hart, Wade, & Martelli, 2003; Nicholson, 2000; Nicholson, Martelli, & Zasler, 2001) In general, chronic pain, independent from traumatic brain injury, appears to have an adverse effect on cognitive functioning. This effect appears to be most salient on aspects of attention, concentration, speed of processing, and

executive control, particularly on tasks that are complex and demanding (Krieter & Niev, 2007; Nicholson, 2000).

Problems with existing attention studies. While studies exist that have provided some evidence for attentional impairment in the context of these two samples, a number of methodological issues exist that may confound the interpretation of these studies' results. First, many studies do not statistically control for factors that may affect performance on neuropsychological measures such performance validity and psychological factors (these topics are reviewed in more detail below). For example, Cicerone (1996); Hess et al., (2003); Mathias et al., (2004); Raskin et al., (1998); and Tiersky et al., (1998) did not explicitly control and/or assess the effects of psychological factors and/or effort. The reviews conducted by Chan (2002) and Nicholson (2000) do not elaborate on which studies, if any, control for factors that could influence the interpretation of test results.

Second, a number of studies rely on self-report to determine the prevalence of attention problems in these populations. Many times, objective evidence from neuropsychological measures that could corroborate these problems are not conducted.

Third, various operationalizations of attention are employed. This can be problematic in both the self-report and objective test literature. As a result, the extent in which studies' findings can be compared with each other is limited. Relatedly, in studies that do employ objective measures of attention, the tests selected may not be valid and reliable measures of the attentional construct being examined, further complicating interpretations. Many studies view attention as a multidimensional construct and attempt to examine these different "types" of attention (e.g., sustained, divided,

selective) using tests that are thought to tap into one of these types when, in actuality, they are measuring more than one type (Chan, 2002). In addition, limited evidence has been found that attention is comprised of distinct components among attention measures and suggests that the majority of measures used are more likely related to a global dimension of attention (Cicerone, 1997; Eccleston & Crombez, 1999; Grisart et al., 2002; Schmidt, Trueblood, Merwin, & Durham, 1994). Therefore, it is important to identify the most current conceptualization of attention. In order to accomplish this, a brief review of the more prevalent attentional models in recent history will be discussed.

Conceptualization/Operationalization of attention. Attention is a fundamental yet multifaceted and complex process and involves the “focusing of mental processes on some aspect of the environment or on a concept” (Parente & Herrmann, 1996, pg. 83). It is well-agreed that the process of attention is vital for an individual’s everyday functioning. Over the decades; however, theorists have differed in their ideas of the overlying conceptualization of what attention is and the subcomponents that comprise it.

Regardless of the different theories of attention that exist, most view attention as being made up of three crucial elements. First, and possibly most importantly, attention is selective, which means that it allows for the exclusion of irrelevant stimuli in order for more necessary information to be processed (Behrmann & Haimson, 1999; Driver, 2001; Goldstein, 2002; Jones & Rizzo, 2004; Parente & Herrmann, 1996; Vecera & Rizzo, 2004). Because our sensory systems are inundated with sensory information at every moment, it is imperative for humans to have a system that will ensure that the appropriate and relevant stimuli necessary for vital functioning is being filtered and attended to while extraneous information is not (Chambers & Mattingly, 2005; Lavie

2005, 2006; Parente & Herrmann, 1996; Vecera & Rizzo, 2004). Second, attention is modulating because it allows for the increased allocation of cognitive resources as a situation becomes more cognitively demanding (Jones & Rizzo, 2004; Lavie, 2005; 2006). Finally, attention acts as a signal – it alerts individuals to important information in the environment that needs to be processed quickly (Parente & Herrmann, 1996).

In 1968, Atkinson and Shiffrin outlined the “Information Processing Model” of memory which stated that we have a brief duration of short-term memory that was limited in capacity for information storage (Atkinson & Shiffrin, 1968 as cited in Parente, Kolakowsky-Hayner, Krug, & Wilk, 1999). Kahneman (1973) and Shiffrin (1988) later expanded on this “limited capacity” idea by postulating that attention is a limited cognitive resource that can be divided amongst tasks – when multiple tasks exceed the attentional “allotment,” problems arise in one’s attentional functioning (Eccelston & Crombez, 1999).

One major criticism of the capacity models was that they viewed attention as a passive and uncontrolled process. Later models of attention, such as those developed by Baddeley and Hitch, proposed a more active process. They proposed that attention was a controlled and active process and postulated that individuals have what is called a working memory. Generally, working memory can be defined as one’s ability to hold information while also processing potentially interfering distractions (Baddeley & Hitch, 1974; Jarrold & Towse, 2006). According to the “Baddeley and Hitch Working Memory Model,” the working memory system is comprised of three main components. Of most importance is a central executive system which functions in prioritizing information processing, coordinating and scheduling mental operations, and allocating the cognitive

resources necessary for attentional ability (Baddeley, 1986; McCallister et al., 2004; Norman & Shallice, 1986; Parente et al., 1999; Willmott, Ponsford, Hocking, & Schonberger, 2009). Connected to this system are subsidiary storage mechanisms that are concerned with maintaining information for short periods of time (for simultaneous processing) including the phonological (articulatory) loop and the visuospatial sketchpad (McCallister, Flashman, Sparling, & Saykin, 2004).

Since working memory is considered limited in capacity, there is a tradeoff between storage and processing. If a goal is particularly complex or taxing, the task is given processing priority over other tasks (Eccleston & Crombez, 1999; Jones & Rizzo, 2004). Given this, working memory is thought to work closely with one's information processing speed. Individuals that are more efficient at completing tasks use less of their cognitive resources and thus have more working memory capability (Kennedy, Clement, & Curtiss, 2003; Jarrold & Towse, 2006).

Examination of the main neuropsychological problems reported to persist in mild TBI and CP lend evidence for the use of a unidimensional construct of attention as most, if not all, of the impairments that are reported can be due to an impairment in the central executive system of working memory (Cicerone, 2002; McCallister et al., 2004; Serino et al., 2006). It is this system that is thought to responsible for allocating attentional resources and modulating less complex attentional processes (Chan, 2001). Given this recent conceptualization, researchers have postulated that the term working memory should be referred to as working attention to reflect this relationship (Willmott et al., 2009).

Summary. Overall, studies utilizing mild TBI and CP populations have shown that some cognitive processes appear to be more affected than others. Specifically, areas of attention and information processing seem to be the most sensitive to these injuries. However, a number of methodological issues exist that complicate the interpretation of the conclusions drawn from these studies. In addition to differing conceptualizations of the construct of attention, some studies lack an explicit examination of the influential role of psychosocial variables (e.g., performance validity/exaggeration, psychological factors) on the relationship between injury and performance on measures of cognitive functioning.

Contributing Factors to Persisting Cognitive Symptoms

Most researchers agree that persisting symptoms in individuals who have sustained uncomplicated mild TBI's are not a function of physiogenic factors but rather psychogenic ones. A number of non-organic factors have been proposed that are thought to contribute to the persistence of symptoms and poor outcome observed in some individuals. These include (but are not limited to): situational factors (e.g., litigation and compensation-seeking influences), pre-injury and post-injury psychiatric and psychological factors (e.g., anxiety, depression, and somatization), expectations and attributions after injury, post-injury adjustment stressors, and social issues (e.g., post-injury stressors, lack of social support system), among others (Iverson, 2007; Macleod, 2010; McCrea et al., 2009).

Exaggeration/effort. One significant influence proven to contribute to persisting symptoms is the role that motivation/effort plays during a clinical evaluation. In fact, numerous studies have shown that effort usually accounts for the most variance in

neuropsychological test scores (Iverson, 2005; Rohling, Allen, & Green, 2002; Stevens, Friedel, Mehren, & Merten, 2008). Specifically, some individuals choose to appear more disabled and symptomatic than is the case by either exaggerating their cognitive problems during neuropsychological testing, exaggerating self-reported symptoms, or both (Lange, Iverson, Brooks, & Rennison, 2010). In turn, attempting to identify and understand residual cognitive deficits associated with mild TBI and CP can be complicated by this exaggeration.

This has been shown to be the case when the patient is involved in litigation and/or there is a known external incentive, such as financial compensation, time off of work, or paid medical coverage and benefits. The presence of financial incentive has been found to be one of the strongest predictors of poor outcome in both mild TBI ($d = .47$) and CP ($d = .48$) (Binder & Rohling, 1996; Binder et al., 1997; Paniak et al., 2002; Reynolds, Paniak, Toller-Lobe, & Nagy, 2003; Rohling, Binder, Langhinrichsen-Rohling, 1995).

The presence of incentive may motivate some individuals to exaggerate or feign their symptoms (i.e. malingering) in such a way as to “reap” as much incentive as possible. One strategy requires the individual to report significant complaints in a variety of functional domains (e.g., reporting severe cognitive, emotional, and physical problems) (Bianchini, Etherton, & Greve, 2004; Bianchini, Greve, and Glynn, 2005). In a study examining the relationship between the rate of failure on cognitive indicators of malingering and magnitude of potential compensation, Bianchini, Curtis, and Greve (2006) found that the rate and extent of exaggeration increased with the magnitude of

incentive. In other words, the amount of exaggeration was in proportion to the amount of incentive the individual had the potential to gain.

Assessing exaggeration/effort. In order to determine the abilities and/or deficits an individual may have after having sustained an injury, it is important for individuals to undergo as comprehensive an evaluation as possible. This includes administering a broad range of neuropsychological and psychological tests that reliably measure a variety of domains and interpreting these results taking into account the patient's pre-, peri-, and post-injury factors. It is particularly important to assess any motivational and/or effort factors that may influence test validity and/or interpretation as well (American Academy of Clinical Neuropsychology, 2007; Heilbronner, Sweet, Morgan, Larrabee, Millis, & Conference participants, 2009; Iverson, 2007). As indicated above, the presence of incentive has been shown to be a strong motivational influence in some individuals and can affect the level of effort that is exhibited by the patient during the evaluation. As such, it is recommended that neuropsychologists include multiple measures of effort and validity throughout the course of their evaluations (Boone, 2009; Bush et al., 2005; Iverson, 2003; Lynch, 2004).

Performance validity indicators. One way shown to be effective at assessing level of effort is the use of symptom validity tests (SVTs; Pankratz, 1979). The SVT is a forced-choice measure that requires an individual to correctly choose between a previously shown target and a foil item. Since only two answer choices are available, an individual should select the correct choice approximately 50 percent of the time by chance alone (Bianchini, Mathias, & Greve, 2001). Therefore, if an individual shows below-chance performance, this is an indication that they are aware of the correct

answer but knowingly selected the incorrect one (i.e. negative response bias; Pankratz, 1983; as cited in Bianchini et al., 2001). Negative response bias; however, is rarely observed – most patients who exaggerate do not perform worse than chance but rather poor effort is revealed by scores that fall below empirically-derived cutoffs established and validated in samples of people with unequivocal cerebral dysfunction (Binder, 1993; Tombaugh, 1996).

Although numerous SVTs exist, a select number of SVTs are commonly used in clinical practice as they have received the most validation and empirical support in a variety of clinical populations. These include: the Portland Digit Recognition Test (PDRT; Binder, 1993), the Test of Memory Malingering (TOMM; Tombaugh, 1996, 1997), and the Word Memory Test (WMT; Green, 2005; Green, Allen, & Astner, 1996).

One major advantage of SVTs in general is that they are tests of cognitive effort but not cognitive ability; therefore, failure on an SVT is an indication of poor effort and not a sign of cognitive deficit (Bianchini et al., 2001). However, over recent years, increased availability of information regarding a specific test's goals and administrative procedures has made coaching a potential complication during evaluations (Suhr & Gunstad, 2007). As such, the use of internal validity indicators (i.e., embedded indicators) derived from standard clinical instruments has become increasingly important (for reviews see Boone, 2007; Larrabee & Berry, 2007). Internal validity indicators have drawn considerable research and clinical interest because 1) they enhance the sensitivity of a neuropsychological battery in detecting response bias without increasing the time required for the assessment; 2) they provide information

about the validity of performance on specific tests; and 3) they may be less likely to be coached than stand-alone validity tests (Meyers & Diep, 2000).

Self-report validity indicators. As part of a comprehensive evaluation, neuropsychologists often have to heavily rely upon the self-report of the patients regarding the details of their injuries and subsequent symptoms and issues that they are experiencing. This is especially the case in individuals who are evaluated long after the injury, when objective medical information regarding the injury is either scarce or not available. For example, the patient may not have sought medical care right away (if at all), medical documents such as emergency room records may not be accessible that would provide clinicians with acute injury characteristics (e.g., GCS, LOC, PTA), and/or the appropriate neurodiagnostic tests may not have been conducted that would detail the extent of the person's injury (e.g., CT, MRI). Other factors can further complicate the authenticity of self-reported symptoms, either because other factors are contributing to a person's report of diminished ability or increased disability (e.g., litigation, secondary gain), or because an individual lacks the insight into the extent of their problems, as is often observed in individuals who have sustained severe brain injuries (Heilbronner et al., 2009). As such, the validity of an individual's self-report needs to be assessed.

Heilbronner et al., (2009) provide a brief summary of the two main types of self-report measures that are utilized in clinical evaluations. The first are disorder-specific inventories or checklists that measure the types and frequencies of symptoms associated with a particular disorder such as depression or post-traumatic stress disorder. However, Heilbronner et al., (2009) stress the importance of using measures

that provide some sort of indication of the validity of the person's self-report and/or the presence of response bias.

A second type of self-report measure that is frequently employed is a personality inventory such as the Minnesota Multiphasic Personality Inventory-II. These measures provide an overall picture of a person's psychological functioning. Along with clinical scale information, these measures often contain a number of validity scales and/or indicators. For instance, one of the most used inventories (Rabin, Barr, & Burton, 2005), the MMPI-2, contains indicators that assess response validity (F, Fb, Fp) as well as specific measures designed to detect response bias (Symptom Validity Scale [FBS; Lees-Haley, English, & Glenn, 1991], Response Bias Scale [RBS; Gervais, 2005]).

Cognitive exaggeration. An enormous amount of research has been conducted over the past two decades regarding the methodology used to detect response bias, particularly cognitive exaggeration (for a review of the methods see Larrabee, 2007 and Morgan & Sweet, 2009). Studying the influence of cognitive exaggeration on neuropsychological test performance has primarily been done using one of two research methods – the “purification” method and methods using malingering classification systems (for a review of the two most commonly used classification systems see Bianchini, Greve, & Glynn, 2005; Slick, Sherman, & Iverson, 1999). The methods are similar in that both involve identifying individuals exhibiting poor performance on measures of cognitive validity; however, they differ somewhat in the questions they are trying to answer. In “purification” models, individuals exhibiting poor effort (as indicated by performance on measures of response bias) are identified and either excluded from analyses, or analyzed separately, in order to ascertain the true

neuropsychological deficits associated with an injury from those deficits that are attributable to poor effort. In malingering research, the focus is to determine the ways in which the performance of a malingerer can be distinguished from that of a non-malingerer and to determine the efficacy of an indicator at detecting malingering.

Green and colleagues were some of the first to stress the importance of controlling for effort while examining the cognitive deficits associated with brain injury. Green and Iverson (2001) first examined the moderating role of effort on the relationship between injury severity and olfactory discrimination, as measured by the Alberta Smell Test (Green & Iverson, 1998). Their sample consisted of 322 mixed-head injury severity cases (as determined by objective measures of injury) and 126 people with orthopedic injuries. All patients included in the study were involved in some form of compensation claim at the time of their evaluation. Every patient was administered the Alberta Smell Test and two SVTs, the Computerized Assessment of Response Bias (CARB; Allen, Conder, Green, & Cox, 1997; Conder, Allen, & Cox, 1992) and the Word Memory Test (WMT; Green, Allen, & Astner, 1996). The authors postulated a dose-response effect between injury severity and olfactory ability in those patients exhibiting good effort whereas no such relationship between these variables would exist in poor effort patients.

As expected, a clear dose-response relationship between olfactory abilities and injury severity was observed – patients with mild head injuries exhibiting good effort did not differ significantly from controls (patients with orthopedic injuries who passed SVTs) and patients with severe head injuries had significantly worse olfactory deficits compared to patients with mild head injuries. In the poor effort group, patients with mild

head injuries were more likely to produce impaired olfactory test scores than mild TBI patients who passed the effort measures. A follow-up study conducted by Green, Rohling, Iverson, and Gervais (2003) supported the findings of this study of a dose-response relationship between injury severity and olfactory ability when effort is controlled.

Green, Rohling, Lees-Haley, and Allen (2001) extended their investigation when they examined the effects of effort on neuropsychological test performance in a sample of compensation-seeking patients. Their sample consisted of 904 patients, 470 of which had brain injuries ranging in severity from mild to severe. All patients were given up to 43 neuropsychological tests, representing six cognitive domains (executive functioning, memory and learning, verbal comprehension, attention and working memory, perceptual organization, and psychomotor skills) and two SVTs, the CARB and WMT. Neuropsychological test scores were transformed to z-scores and averaged to create an Overall Test Battery Mean (OTBM). The results showed that when all patients were analyzed together, no dose-response relationship was observed between injury severity and OTBM scores. However, when only good effort patients were analyzed, the dose-response relationship emerged. Similar to the findings of Green and Iverson (2001) and Green et al., (2003), there was a greater proportion of poor effort patients within the mild head injury group and these patients had significantly lower scores (*mean OTBM z-score* = -1.34) when compared to more severely injured good effort TBI patients (*mean OTBM z-score* = -0.37). Overall, results showed that effort accounted for 53 percent of the variance in OTBM scores whereas acute injury characteristics each accounted for approximately one percent of the variance in scores.

Most recently, similar findings were observed by Meyers, Volbrecht, Axelrod, & Reinsch-Boothby (2011) in their study examining the effect of effort and litigation on neuropsychological test performance in a sample of 314 referrals with mild TBI. Effort was assessed using nine embedded SVTs from the Meyers Neuropsychological Battery (MNB; Meyers & Rohling, 2004). Involvement in litigation was consistently related to more SVT failures than not being involved in litigation and the correlation between SVT failure and the OTBM was significantly negative ($r = -.77$). This is very similar to the correlation that Green et al., (2001) found between failure on the WMT and performance on the OTBM ($r = -.73$). Additionally, the amount of variance in neuropsychological performance accounted for by effort in this study (50%) was almost identical to that obtained by Green et al., (2001) (54%).

As can be seen above, many studies focusing on the topic of effort have examined the role of effort on overall neuropsychological functioning. Few studies exist that look at the influence of effort on specific cognitive domains such as attention. Curtis, Greve, and Bianchini (2005) conducted a study comparing attention scores of mild TBI and CP patients while controlling for effort. Attention was measured using two variables from the California Verbal Learning Test-II (CVLT-II) and patients were grouped into either good or poor effort groups based on their performance on the Word Memory Test. Similarly, in 2010, Guise, Greve, and Bianchini examined the role of effort on different types of attention (focused, selective, divided, and sustained) in a sample of mild and moderate-severe TBI patients and a demographically-matched control group. In both studies, effort accounted for more variance in attention scores than injury severity.

The findings of the Curtis et al., (2005) and the Guise et al., (2010) studies are consistent with the effort literature. They add to the existing literature in that supplemental analyses were conducted examining the influence of psychological factors on lowered attention scores in good effort patients. However, the studies are limited by the variables chosen to measure attention - Guise et al., (2010) studied subtypes of attention and the tests selected may not have been the most representative measures of these subtypes and Curtis et al., (2005) used only two variables to represent attention and a limited sample size was used.

Symptom exaggeration. Symptom exaggeration is also a frequently observed phenomenon in TBI and non-TBI populations involved in litigation or compensation contexts and can involve exaggeration of neuropsychological, physical, and/or psychological symptoms. Appendix C contains some of the studies that have examined the rates of exaggeration in these areas via various post-concussion questionnaires. Additional studies, not included in Appendix C have examined the relationship between litigation and symptom exaggeration on various psychological measures in a variety of populations including: TBI (Boone & Lu, 1999; Greiffenstein & Baker, 2001a, 2001b; Larrabee, 2003c; Miller & Donders, 2001) psychiatric patients (Lees-Haley & Brown, 1993); pain (Gervais, Rohling, Green, & Ford, 2004; Larrabee, 2003b; Schnurr & MacDonald, 2001); and mixed groups (e.g., pain versus brain injury; Dunn, Lees-Haley, Brown, Williams, & English, 1995; Iverson, King, Scott, & Adams, 2001; Lees-Haley, Fox, & Courtney, 2001; Smith-Seemiller et al., 2003). For the purposes of this paper, a select group of studies will be reviewed in more detail because of their examination of

poor effort on self-reported cognitive symptoms in either TBI patients, CP patients or both.

Tsanadis, Montoya, Hanks, Millis, Fichtenberg, and Axelrod (2008) compared post—concussion symptom endorsement between a group of moderate-severe TBI ($n = 133$) patients and poor effort mild TBI patients ($n = 25$). Inclusion in the poor effort group required failure on at least two of three SVTs: the Recognition Memory Test, Word Memory Test, or Test of Memory Malingering. All of the poor effort patients met Slick et al. (1999) criteria for probable or definite malingered neurocognitive dysfunction. Post-concussion symptom endorsement was measured using the Postconcussive Symptom Questionnaire which is a 45 item measure that yields four index scores – psychological, cognitive, somatic, and infrequently reported symptoms. Statistical analyses showed that the poor effort group reported significantly more symptoms than the moderate-severe TBI on all of the indices. Analyses were also conducted to examine the effect of litigation on symptom report. As expected, a comparison of litigating poor effort mild TBI and litigating moderate-severe TBI patients showed that the poor effort mild TBI group reported significantly more psychological, cognitive, and somatic symptoms than the moderate-severe group.

Recently, Lange, Iverson, Brooks, & Rennison (2010) conducted a study looking at the influence of poor effort on neurocognitive test performance and self-reported symptoms in mild TBI. Sixty-three mild TBI patients who completed the Post-Concussion Scale and the British Columbia Cognitive Complaints Inventory were divided into two groups based on their performance on the TOMM. As expected, patients who failed the TOMM reported significantly more PCS symptoms than those

that passed the TOMM as well as significantly more cognitive complaints on the BC-CCI. Even though a majority of patients in both groups reported cognitive problems, the effect sizes were largest for the cognitive complaints of forgetfulness, poor concentration, and problem solving.

Following this study, Iverson, Lange, Brooks, and Rennison (2010) examined differences in retrospective ratings of pre-injury neuropsychological status to post-injury rates in a sample of compensation-seeking mild TBI patients averaging two months post-injury. Specifically, the authors sought to assess a phenomenon referred to as the “good old days” bias in which individuals perceive themselves as being healthier before an injury than might actually be the case. In turn, this results in an overestimation of the amount of cognitive change that has taken place after the injury. Additionally, the role of effort test performance on symptom reporting was also examined. The participants included 90 patients who were classified as having sustained a mild TBI, 95% of which met ICD-10 Criterion C for post-concussion syndrome, and a control group of 177 healthy adults derived from the samples of two earlier studies conducted by the authors. All of the participants completed the British Columbia Post-Concussion Symptom Inventory (BC-PSI) with the mild TBI patients completing the inventory twice – once to assess post-injury perceptions of their symptoms and the second to provide retrospective symptom ratings based on their cognitive functioning the month before their injuries.

As expected, significantly more post-injury symptoms were endorsed by the mild TBI patients compared to their pre-injury endorsement rates and the rates endorsed by the healthy controls (mild TBI pre- vs. post-injury Cohen’s $d = 1.21-3.13$; mild TBI; mild

TBI post-injury vs. controls $d = 0.78-2.74$). Additionally, the mild TBI group endorsed significantly fewer pre-injury symptoms than the control group ($d = 0.65$). In terms of effort, patients in the mild TBI group who failed effort testing, as measured by the TOMM, reported more post-injury symptoms and retrospectively endorsed fewer pre-injury symptoms than those who passed the TOMM. Both of the effort subgroups endorsed fewer symptoms prior to their injury compared to the control group, however, which suggests that additional psychosocial processes beyond effort could be influencing symptom reporting.

Two studies have been conducted that examine the relationship between litigation status and PCS symptom report comparing pain and traumatic brain injury patients. Iverson, King, Scott, & Adams (2001) examined the rate of cognitive symptom and psychological symptom report in four clinical samples: 20 chronic pain patients involved in worker's compensation claims, 20 chronic pain patients undergoing evaluation for spinal stimulators, 20 mixed head injury-severity patients involved in litigation, and 20 non-litigating head injury patients. The entire sample completed a symptom checklist developed by the authors to assess symptoms in the areas of motor, sensory, cognitive, and emotional functioning, as well as the MMPI-2. The results showed that 90% of the litigating head injury patients and 95% of the worker's compensation pain patients reported at least one cognitive problem and that the cognitive symptoms reported by each of these groups was equivocal. On the MMPI-2, the litigating groups had higher scores on F, Fb (both validity scales), Hypochondriasis (Scale 1) and Hysteria (Scale 3) than the non-litigating groups.

The second study examined the symptom endorsement rates of 63 patients with chronic pain (and no history of head injury) and 32 patients with mild TBI (based on GCS, PTA < 24 hours, and negative neurological findings) (Smith-Seemiller et al., 2003). Fifty-six percent of the mild TBI group and 83% of the chronic pain group had financial incentives available to them. The two groups did not statistically differ on their mean total scores on the Rivermead Postconcussion Questionnaire although there was a trend towards the mild TBI group endorsing more cognitive symptoms and the CP group endorsing more emotional symptoms. Post-hoc analyses looking at the impact of financial incentive showed that patients involved in litigation reported significantly higher scores than non-litigating patients.

Summary. Wood and Rutterford (2006) nicely summarize the findings of studies examining exaggerated self-report by stating that individuals involved in litigation or a compensation context report more symptoms that last longer and are more debilitating than the symptoms reported by individuals who are not involved in litigation. However, evidence of either or both types of exaggeration is not solely reflective of malingering (Iverson, 2007). Given that the base rate of malingering in either sample averages around 30%, it is important to understand other mechanisms that may be contributing to persisting symptoms in both of the these populations. Various psychological mechanisms have also been shown to contribute to the persisting cognitive symptoms as well.

Persisting symptoms are not solely the result of being involved in the medico-legal process. Many studies have demonstrated persisting symptoms in individuals in whom compensation-seeking is infrequent (Jacobson, 1995). In an interesting study

conducted by Mickeviciene et al. (2004), rates of persisting symptoms in concussed patients in Lithuania were examined. In this country, possibilities for economic gain are minimal and expectations of persisting symptoms are considerably less than in Western societies. The study showed that the vast majority of post-concussion symptoms after head injury lasted for less than one year. In individuals that reported more subjective cognitive dysfunction, persisting symptoms were found to be related to psychosocial factors (e.g., unmarried individuals, lower education levels, psychological factors).

Research has shown that of those involved in litigation, symptoms are often not alleviated once a settlement has been reached (Fee & Rutherford, 1988; Miller, 2001). Approximately one-third of patients who are symptomatic at the time of settlement are still symptomatic one year later (Miller, 2001) and some studies have shown that some litigants remain symptomatic even after five years (Bernstein, 1999; Mendelson, 1984; 1995). Furthermore, in studies looking at treatment efficacy immediately after an injury, compensation and non-compensation seeking patients often do not differ in recovery time (Jacobson, 1995). Finally, research has shown that even after reparative surgery, symptom resolution for some patients often does not occur (Arpino, Iavarone, Pariato, & Moraci, 2004; Ostelo, Vlaeyen, van den Brandt, & de Vet, 2005). This implies that other psychosocial mechanisms are likely contributing to persisting symptoms.

Psychological factors. In some individuals, pre-existing stressors and psychological factors can put a person “at-risk” for developing persisting symptoms (Evered, Ruff, Baldo, & Isomura, 2003). Research looking at the role of psychosocial influences of persisting symptoms in mild TBI and chronic pain has shown that four interpersonal mechanisms are consistently reported in the literature as contributing to

symptom chronicity. First, increases in emotional distress post-injury can cause an over-focus on physical symptoms, particularly if the person is experiencing injury-related anxiety (Brown, 2000; McBeth, Macfarlane, & Silman, 2002; Turk, 2002). Related to this, cognitive distortions, such as catastrophizing, can increase the subjective severity of symptoms (Geisser et al., 2003; Geisser, Robinson, Keefe, & Weiner, 1994; Linton, 2000; Turk, 2002). Working with the first two mechanisms, inaccurate appraisal and attribution may cause maladaptive coping strategies (Jacobson, 1995; Kendall, 2003; Kendall & Terry, 1996; Turk, 2002). Finally, low self-efficacy may cause the person to take on a helpless attitude which further prolongs their perceived disability (Brox et al., 2005; Rudy, Lieber, Boston, Gourley, & Baysal, 2003; Turk, 2002).

Emotional distress. Collectively, anxiety and depression are the most prevalent psychological disorders among the general population with lifetime occurrence rates around 30% (Kessler, Berglund, Demler, Jin, & Walters, 2005; Moore, Terryberry-Spohr, & Hope, 2006). Within the context of mild TBI and CP, these disorders may be even more prevalent (Crisp, 2005; Dersh, Gatchel, Mayer, Polatin, & Temple, 2006; Linton, 2000; Mayer, Towns, Neblett, Theodore, & Gatchel, 2008; Mooney & Speed, 2001). Post-injury levels of anxiety and depression have been found to have direct impacts on outcome from injury (Alexander, 1992; Geisser et al., 2003; Hart et al., 2000; Hart et al., 2003; Linton, 2000; Mendelson 1984; Williams et al., 2010) and are often mistaken as chronic effects of mild TBI and CP (McCrae et al., 2009). Although levels of anxiety and depression can vary, even mild anxious or depressive symptoms can influence a person's report of injury-related symptoms since these symptoms themselves are often

associated with problems in cognition, emotional, and physical functioning (Crisp, 2005; Hart et al., 2003; Krieter & Niev, 2007).

Effects of emotional distress on self-report. The above point is exemplified in studies looking at the types of symptoms reported in various non-head injury-related populations (the reader is again referred to Appendix C for a list of studies employing these populations and the symptom endorsement rates). For example, Fox et al., (1995a) found that psychiatric patients with no history of head injury exhibit high endorsement rates for PCS symptoms. A follow-up study by the same authors (1995b) showed that psychiatric patients report more PCS symptoms than controls and some medical patients and are comparable to the levels reported by head injury and neurology patients. Iverson (2006) found that approximately nine out of ten patients with depression (diagnosed by means of the Structured Clinical Interview for DSM-IV) met liberal self-report criteria for postconcussion syndrome and more than five out of ten met conservative criteria for the diagnosis. Seventy-two percent of the sample endorsed three or more symptoms post-concussive symptoms with ratings of moderate or higher.

Significant relationships between affective states and self-report of concussive symptoms have been shown in a number of studies utilizing healthy participants as well. Iverson and Lange (2003) administered the British Columbia Postconcussion Symptom Inventory- Short Form (BC-PSI-Sf), which is patterned after the ICD-10 criteria for Postconcussion Syndrome, as well as the Beck Depression Inventory-II to 104 healthy volunteers. Symptom reporting on the BC-PSI-Sf showed moderately high correlations with BDI-II scores ($r = .76$). To further investigate this correlation, the authors divided the total sample using a BDI-II cutoff score of 14 or greater. In individuals showing

elevated depression scores ($n = 24$), the most frequently reported PCS symptoms included: feeling fatigued (95.8%), irritability (91.7%), sadness (91.7%), nervousness (91.7%), poor concentration (91.7%) and poor memory problems (83.3%).

Following Iverson and Lange's (2003) data analysis approach, Garden and Sullivan (2010) found that a subsample of healthy volunteers with elevated BDI-II scores ($n = 24$; BDI-II scores ≥ 14) had significantly higher PCS scores and double the mean PCS score on the British Columbia Post-Concussion Symptom Inventory (BC-PSI) than non-depressed participants. The most commonly reported symptoms in the depressed subsample were headaches (95.8%), fatigue (83.3%), irritability (91.7%), feeling sad (95.8%), anxiety (95.8%), poor concentration (83.3%), temper problems (87.5%), and poor sleep (83.3%) (Garden & Sullivan, 2010).

Also using the BDI-II as a measure of depression, Wang, Chen, & Deng (2006) examined the rates of PCS endorsement in a sample of 124 healthy university students. The most highly endorsed cognitive problems included poor concentration (58.7%) and "taking a longer time to think" (60.3%). A moderately high correlation was found between scores on the Rivermead Postconcussion Questionnaire and BDI-II scores ($r = 0.615$) using the entire sample. When the sample was broken down by BDI-II scores into "low-symptom" and "high-symptom" reporting groups, the average number of PCS symptoms endorsed was ten times higher for the depressed subgroup compared to the whole group.

Similar results have been observed within the context of mild TBI. Panayiotou, Jackson, and Crowe (2010) conducted a meta-analytic review looking at the types of emotional symptoms that are associated with mild TBI. Their meta-analysis included

eleven studies yielding sample sizes of 352 mild TBI patients and 765 control participants who were either healthy (6 studies) or non-head-injured patients (5 studies). The authors found that the most frequent category of emotional symptom tested was depression, which contributed to 52.9% of the overall effect sizes, followed by anxiety (29.4%). Specifically, the average effect size for depression and anxiety were .80 and .53, respectively. When effects sizes were weighted by sample size, the effect sizes dropped down to .09 which constitutes a nearly negligible influence of mild TBI on emotional symptoms. However, given a number of individuals report persisting problems, this study lends evidence to the fact that alternative mechanisms beyond organic ones are what contribute to symptom chronicity.

Trahan, Ross, and Trahan (2001) conducted an interesting study examining the relationships between postconcussion symptom report, depression, and anxiety and whether these relationships differed by diagnosis. Altogether, the frequency and severity of symptom endorsement of 496 young adults with no history of head injury or depression were compared to a group of non-head injured depressed individuals ($n = 56$) and individuals with a history of mild head injury ($n = 40$) on the Beaumont Postconcussional Index (BPCI), Beck Depression Inventory-II, and the Beck Anxiety Inventory. All three measures exhibited high correlations with each other (BPCI and BDI-II [$r = 0.68$]; BPCI and BAI [$r = 0.64$]). In terms of symptom reporting, the mild head injury group reported more postconcussion symptoms than the control group; however, the depressed individuals reported significantly more postconcussion symptoms than either group.

Suhr and Gundstad (2002) also nicely demonstrated that the presence of PCS symptoms may reflect the influence of psychological factors rather than injury-related factors in their comparison of individuals with a history of mild head injury and those exhibiting depressive symptoms but no history of head injury. Six hundred and seventy-seven healthy undergraduate students completed a variety of self-report measures, including a measure of depression (Beck Depression Inventory-II) and a post-concussion symptom checklist developed by the authors. From the original sample, the authors selected four subsamples of individuals: those who self-reported having experienced a head injury at some point their past (HI, $n = 31$); individuals self-reporting a past head injury and obtaining a BDI-II score greater than 12 (HI/Dep, $n = 32$); individuals obtaining a BDI-II score of greater than 12 but no reported history of head injury (Dep, $n = 25$); and a group of controls who did not have a history of head injury and had BDI-II scores less than 7 (controls, $n = 50$). A 2 (depression versus no depression) X 2 (history of head injury versus no history of head injury) ANOVA with total PCS symptoms as the dependent variable showed only a main effect for depression – the subjects with depressive symptoms (collapsing across head injury status) endorsed significantly more PCS symptoms. No significant main effect for head injury status or an interaction between head injury status and depression level was observed.

Iverson and McCracken (1997) examined the base rate of cognitive symptoms in non-litigating pain patients with no history of head injury ($n = 170$). The average time between injury and evaluation was 79.7 months, making their pain chronic in nature. All participants completed the BDI, Modified Somatic Pain Questionnaire (MSPQ), and

Sickness Impact Profile. Forty-two percent of the sample endorsed at least one cognitive complaint. Specifically, 29% reported experiencing forgetfulness, 18% reported difficulty with attention, and 16.5% reported problems with concentrating or thinking. In comparing symptom endorsement rates with DSM-IV TR PCD diagnostic criteria, the authors found that 80.6% of the sample endorsed three or more symptoms from Category C (non-cognitive symptom) for PCS. Overall, 39% of the total sample would have met self-report criteria for PCS in that they reported at least one cognitive problem and three or more symptoms from Category C of the diagnostic criteria for PCS. A follow-up study conducted by the same authors showed comparable endorsement rates of cognitive symptom and the number of individuals meeting diagnostic criteria for PCS. Additional analyses showed that pain-related anxiety and depression were moderately correlated with the total number of cognitive complaints endorsed with depression accounting for the largest proportion of unique variance in predicting cognitive complaints in multiple regression analyses (McCracken & Iverson, 2001).

Another study examined the rates of memory complaints of two groups of pain patients (whiplash and low back pain) in comparison to a group of medical patients and a group of psychotherapy patients (Schnurr & MacDonald, 1995). All participants completed the Memory Observation Questionnaire – 2 (MOQ2), the Chronic Pain Memory Complaint Questionnaire (CPMCQ), the BDI, and the State-Trait Anxiety Inventory. Both pain groups reported significantly more memory problems than the other two groups on the MOQ2 and CPMCQ; however, an analysis of covariance controlling for levels of anxiety and depression showed that group differences

disappeared on the MOQ2 with depression being the only significant contributor to MOQ2 scores.

Finally, the samples in the Iverson et al., (2001) and Smith-Seemiller et al.,(2003) studies (reviewed in detail in the “symptom exaggeration” section above) analyzed the rate of PCS symptom reports in both CP and TBI. Even taking into account litigation status, a majority of the non-litigating/ no incentive groups in each of the studies reported cognitive problems. For example, all of the non-litigating head injury patients and 50% of the non-litigating pain patients reported at least one cognitive symptom and both non-litigating groups showed high elevations on Scales 1, 2, and 3 of the MMPI-2 (Iverson et al., 2001). These findings stress the importance of looking at the effects of psychological factors on symptom report in addition to the effects of incentive and/or litigation.

Effects of emotional distress on objective measures of cognitive functioning. As can be seen, a number of studies have been conducted examining the correlation between emotional status and self-reported cognitive problems. Fewer studies have examined the relationship of emotional state on objective measures of neuropsychological functioning. The studies reviewed below are ones that explicitly examined the interaction between emotional distress and neuropsychological functioning in mild TBI and CP.

Ponsford et al., (2000) studied factors that contributed to persisting cognitive symptoms in a sample of mild TBI patients ($n = 84$) evaluated one week and again three months after sustaining their injuries. At both time points, patients were administered the SCL-90-R and the Holmes-Rahe Survey of Recent Experiences to measure their

pre-injury psychological states and concurrent life stressors, respectively, as well as the Post Concussion Symptom Checklist and neuropsychological measures of attention, speed of information processing, and memory. Overall, the mild TBI group scored significantly worse than a non-head injury control group ($n = 53$) on measures of processing speed at one week post-injury but this difference disappeared by three months post-injury. However, there was a subgroup of individuals (24%) who reported significant ongoing psychological and cognitive problems. When their SCL-90-R profiles were studied, the group significantly differed from their mild TBI counterparts on all of the subscales – scores the SCL-90-R for the symptomatic group had significantly increased at this time point compared to the non-symptomatic groups, whose scores had decreased. This suggests that psychological adjustment levels deteriorated following the injury for this subset of individuals.

Stulemeijer, Vos, Bleijenberg, & van der Werf (2007) conducted an interesting study comparing non-referred, emergency-department admitted mild TBI patients six months post-injury with and without self-reported cognitive problems on a number of factors including neuropsychological test performance and reported levels of emotional distress. Patients were grouped into either “cognitive complaint” or “no cognitive complaint” groups based on their scores on the RPCQ. All of the patients were administered a neuropsychological battery that was representative of the cognitive domains that appear to be most affected by mild TBI (e.g., processing speed, attention, working memory, verbal memory; Stulemeijer et al., 2007). Emotional distress was measured by the Beck Depression Inventory for Primary Care, the SCL-90 Anxiety Subscale, and the Impact of Events Scale. Results indicated that the groups did not

statistically differ in their neuropsychological performance, although the average effect size was 0.30 with the “cognitive complaints” group scoring more poorly than the group without complaints. Interestingly, 39% of the “cognitive complaint” group and 25% of the “no cognitive complaint” group had a score below the fifth percentile on at least one neuropsychological test. In terms of emotional distress, patients with cognitive complaints reported significantly higher levels of depressed mood, anxiety, and post-traumatic stress than the group with no complaints.

Within the context of chronic pain, inconsistencies in results have been observed in studies assessing the relationship between emotional status and neuropsychological test performance; although there is a trend showing that psychological distress is associated with cognitive deficits. However, it is important to note that many of the studies that have been conducted thus far include samples of individuals with mixed or multiple pain sites, individuals with whiplash injuries and co-occurring head trauma, or have included individuals with pain syndromes associated with medically unexplained symptoms (e.g., fibromyalgia; Hart et al., 2000). For the purposes of this paper, only those studies that utilized non-head injury-related pain patients or whiplash patients without co-occurring head trauma will be reviewed in further detail.

Radanov, Dvorak, & Valach (1992) found that poor performance on a test of processing speed was associated with lowered ratings of emotional well-being and higher levels of self-reported nervousness. In a follow-up study, DiStefano and Radanov (1995) found that those patients who remained symptomatic and evidenced subtle attentional impairments six months and two years post-injury continued to rate their emotional well-being as lower.

Eccleston (1994) compared the performance of a sample of chronic “benign” pain patients, 30% of which suffered low back pain, to a sample of normal controls on an attention demanding numerical interference task. All of the patient’s pain complaints were non-head related, therefore, the study was focusing solely on the impact of pain on attention processing. The pain sample was divided into two groups based on their pain intensity reports on a visual analog scale and numerical rating scale. It was found that patients that reported greater pain intensity performed worse on the attention task compared to controls and those reporting lower levels of pain. While this study showed a relationship between pain level reports and performance on an attention task, Eccleston did not explicitly assess emotional status in this study.

In a follow-up study, Eccleston (1995) replicated these findings using a different sample of pain patients with “benign” pain. Again, patients with any head-related pain were excluded from the study. Unlike the earlier study, he assessed levels of anxiety and depression via the Hospital Anxiety and Depression (HAD) Scale. Analysis of the relationship between emotional state and performance on the attention task showed no correlation between the variables. It is important to note, however, that patients who had “severe” emotional problems were excluded from the study so it is possible that any effect of mood disturbance may have had on attentional performance was attenuated.

Iezzi, Archibald, Barnett, Klinck, & Duckworth (1999) evaluated patients with chronic pain who were recruited consecutively from hospital based pain services. Pain was musculoskeletal in nature and included patients with multiple pain sites. Statistical clustering was used to identify groups reporting high, moderate, and low levels of emotional distress based on their SCL-90-R profiles. The results showed that

differences in neurocognitive performance varied depending on the level of emotional distress a person was exhibiting - those patients with the highest emotional distress exhibited the most deficits in attention and processing speed (e.g., Stroop test, PASAT) compared with those individuals reporting lower emotional distress.

Brown, Glass, and Park (2002) evaluated a large community-dwelling sample of patients with rheumatoid arthritis ($n = 121$) in their examination of whether pain and depression negatively affected cognition. A composite measure of depression was derived from the Depressive Affect subscale of the Center for Epidemiological Studies Depression Scale (CES-D) and the Depression subscale of the Multiple Affect Adjective Checklist-Revised (MAACL-R). Pain was represented as a composite score consisting of the Pain subscale of the Arthritis Impact Measurements Scale 2 (AIMS2-Pain) and the Pain subscale of the Arthritis Pain, Stiffness, and Fatigue Questionnaire (APQ). Results showed that high levels of pain and depression were associated with poor cognitive performance in all four areas of functioning measured (information processing speed, working memory capacity, reasoning ability, and verbal memory). Structural equation modeling showed that depression mediated the relationship between pain and cognitive functioning (e.g., chronic pain causes depression, which causes impairment in cognitive functioning). The effects of pain on cognition were no longer significant after controlling for depression. A model with “paths” from pain to depression and from depression to cognition, but not from pain to cognition, explained 55% of the variance in general cognition.

Wade, Dougherty, Archer, and Prices (1996) provide a conceptual model, the four-stage model of pain processing, that lends itself to the study of attentional

performance in chronic pain patients and which provides a feasible explanation for the lack of consistency across studies. Wade and Hart (2002) postulate that existing studies focus primarily on the effects of early stage pain processing on performance rather than later stage processing. According to the four-stage model, the first two stages of pain processing (pain intensity and unpleasantness) both involve limited cognitive processing. As such, using variables that represent the first two stages and how they affect cognition may not yield salient results. On the other hand, the last two stages, pain suffering and pain behavior, are related to higher cognitive processes. Therefore, they recommend using variables that represent these last two stages in order to explore the relationship between pain and its effect on cognition.

To demonstrate their assertion that cognition is affected by later stage pain processing, Wade and Hart (2002) conducted a study examining the relationship between attention span and each of the four stages of pain processing in a large sample of chronic pain sufferers without a history of head trauma. Separate step-wise regression analyses were conducted using variables that represented each of the four pain stages as predictors of performance on an attention measure. For the predictor variables, pain intensity and unpleasantness were measured using a Visual Analog Scale (VAS), pain-related suffering was measured using a negative emotion VAS, and pain behavior was measured using the four subscale scores from the Psychosocial Pain Inventory. Attention was measured using the age-corrected scaled score from the Digit Span subtest of the WAIS-R. Overall, attentional impairment was associated with suffering and illness behavior and not pain intensity itself. Of all of the variables studied, level of depression, an individual' perception of their lifestyle interference due to pain,

and the degree of solicitous responses from others were each unique predictors of attentional performance.

Somatization. Certain individuals may exhibit prolonged symptomatology as a result of unconscious psychological processes. One such mechanism that is thought to contribute to persisting symptoms is somatization which refers to one's "tendency to experience and communicate somatic distress and symptoms unaccounted for by pathological findings and to attribute them to physical findings" (Binder, 1997, pg. 445; Gatchel, 2004). Somatic symptoms, such as fatigue, headache, dizziness, and nausea are common complaints in both mild TBI and CP patients (Brown, 2004; Fishbain, Lewis, Gao, Cole, & Rosomoff, 2009). In some individuals, experiencing these physical symptoms provides validation to the person that there is a physical/organic basis causing their poor outcome rather than accepting that psychological factors are behind the prolonged disability (Gatchel, 2004; Lamberty, 2008). As seen with depression and anxiety, several studies have shown that patients with high levels of somatization have higher perceived disability and poorer functional outcome (Dersh et al., 2002; Keefe, Rumble, Scipio, Giordano, & Perri, 2004; Lamberty, 2008; Linton, 2000).

Measuring psychological factors. A number of measures have been employed in both mild TBI and chronic pain research to assess the types and extent of psychological problems in these populations. Many studies have utilized one or more assessment measures that are well-established in the literature such as the Beck Depression Inventory and the Symptom Checklist-90-Revised, among others (Celestin, Edwards, & Jamison, 2009). The most commonly used psychological screening tool, however, is the Minnesota Multiphasic Personality Inventory-2 (Butcher et al., 1989;

Slesinger, Archer, & Duane, 2002) which provides clinicians with a profile of an individual's psychological functioning while concurrently assessing the validity of the person's symptom report. Given the interrelatedness of depression, anxiety, and somatization, (Fishbain et al., 2009), the MMPI-2 is an ideal tool for examining these interrelationships.

Examination of these interrelationships has primarily been conducted in samples of individuals with various pain-related conditions. When examining the frequency of psychological symptoms in pain populations using the MMPI-2, factor and cluster analytic techniques have yielded a number of characteristic patterns of psychopathology. Depending on the study, between three and five profile patterns have been identified, with four being the most commonly found factor solution. These profile patterns are: the "conversion-V" profile, which is illustrated by elevations on scales 1 (Hypochondriasis) and 3 (Hysteria) and most indicative of somatization (Arbisi & Butcher, 2004; Larrabee, 1998; Lebovits, 2000; Porter-Moffitt et al., 2006); a "neurotic triad" profile, characterized by elevations on scales 1, 2, and 3; a "depressed-pathological" profile, demonstrated by elevations on four or more scales; and finally, a "normal" profile which consists of individuals who do not have any elevated MMPI-2 scores (Gatchel, Mayer, & Eddington, 2006; Riley, Robinson, Geisser, & Wittmer, 1993; Slesinger et al., 2002). Given the similarities in psychological symptom report in patients with chronic pain or mild TBI, it is reasonable to assume that many of the conclusions drawn from the pain literature regarding psychological profiles are applicable to mild TBI as well.

Summary. There is some debate regarding the causal relationship between psychological factors and outcome from injury. Specifically, do preexisting psychological issues contribute to a worse outcome in some individuals or does the injury that is sustained result in increased psychological distress (Gatchel & Dersh, 2002; Linton, 2000)? Regardless of the etiology, researchers are in agreement that psychological factors significantly contribute to symptom chronicity in both populations and are major contributors to poor outcome in both populations. Therefore, attempting to isolate how these factors on specifically affect outcome is warranted.

Purpose

Over the past two decades, assessing effort and performance validity on neuropsychological measures has been stressed has become increasingly important, in part so that clinicians can make accurate conclusions regarding the residual impairments that may occur post-acutely in various clinical populations, especially mild TBI and CP. Surprisingly, there are relatively few studies that exist investigating the psychosocial factors related to persisting cognitive problems that compare samples of mild head injury patients with patients who have sustained non-head-related injuries (Satz et al., 1999). Of the ones that do exist, non-head injury and mild brain injury groups often do not statistically differ on measures of cognitive functioning. However, this research is limited in the sense that the sample sizes of studies are small, the research is based mainly on self-report, the relationship between psychological factors and objective neuropsychological test scores is typically correlational in nature, and/or, most importantly, performance and self-report exaggeration have not been adequately controlled.

Therefore, the primary purpose of the current study was to examine the psychological factors that may contribute to persisting cognitive (working memory and processing speed) problems in a sample of mild TBI and CP patients while controlling for cognitive and self-report exaggeration. A number of group analyses were conducted to see if, and in what ways, clinical groups differed on important psychological and working attention variables. The inclusion of a moderate-severe TBI group as a comparison group allowed one to see if and how these relationships differed in the presence of objective neurological trauma.

In order to accomplish these goals, a number of steps were implemented. First, potential participants were extensively screened on well-validated cognitive and self-report validity indicators. Only individuals exhibiting valid cognitive and psychological performance were included in the study. Second, select variables from a standardized measure, the Wechsler Adult Intelligence Scale – 3 (WAIS-3) were used to assess working attention ability. Third, select variables from the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), one of the most ubiquitous standardized self-report measures of psychopathology, was used to assess the psychological status of individuals.

Hypotheses

- 1) Given the similarities in symptom reports of patients with mild TBI or CP, it was expected that these groups would not differ in the number of spontaneously-reported symptoms that they reported. Additionally, it was expected that they would report more symptoms than the M/S TBI group.

- 2) The means for the four MMPI-2 scales being examined (Hypochondriasis, Depression, Hysteria, and Psychasthenia) were expected to be non-significantly different between the mild TBI and CP groups. It was expected that these two groups would have statistically higher means on the psychological scales than the M/S TBI group.
- 3) A higher proportion of patients in the mild TBI and CP groups was expected to show elevations on the MMPI-2 psychological scales representing “somatization” (Hypochondriasis [Scale 1] and Hysteria [Scale 3]) and “emotional distress” (Depression [Scale 2] and Psychasthenia [Scale 7]) compared to the proportion of individuals in the M/S TBI group endorsing symptoms on these same constructs.
- 4) The mild TBI and CP groups were not expected to statistically differ on variables representing “working attention” (Working Memory Index T-score, Processing Speed Index T-score) but were expected to be significantly lower than normal (based on the T-score distribution; $M = 50$). It was also expected that the scores for the mild TBI and CP group would be at comparable or even lower levels than those obtained for the M/S TBI group.
- 5) It was expected that the psychological scales would significantly predict both working memory and processing speed performance for the mild TBI and CP groups but not for the M/S TBI group.

CHAPTER 2: Methods

Participants

Retrospective data were obtained from patients seen for either a pain psychological or neuropsychological evaluations at a clinical psychology practice located in southern Louisiana. Specifically, the records of 848 pain patients (evaluated between 1998 and 2008) and 767 traumatic brain injury (TBI) patients (evaluated between 1998 and 2011) were reviewed in order to obtain the objective medical diagnostic test results, injury characteristics, and performance on measures of cognitive and self-report validity that contributed to the characterizations of the groups.

The inclusion criteria to be considered for the study were: 1) referral for persisting symptoms/complaints associated with a brain injury or spine-related injury (see Injury Group classification below for a detailed description of injury characteristics that were examined); 2) age between 18 and 60 years; 3) between eight and 15 years of formal education; and 4) time between injury and evaluation of at least six months but less than 15 years (see the exception for the M/S TBI group); 5) completion of the Wechsler Adult Intelligence Scale-3rd edition (Wechsler, 1997); 6) completion of at least two of the three cognitive performance validity measures at a level reflecting evidence of acceptable validity (see detailed criteria below); and, 7) completion of the Minnesota Multiphasic Personality Inventory-2nd edition (MMPI-2; Butcher et al., 1989).

The final sample was comprised of 249 cases ($n = 116$ TBI; $n = 133$ CP). The mean age for the full sample was 41.2 years ($s.d. = 10.8$). The sample had completed an average of 12.1 years of education ($s.d. = 1.4$) and were, on average, 31.6 months post-injury ($s.d. = 28.1$). The sample was 63.5% male and 77.1% Caucasian (African-

American = 18.9%; other or “not indicated” = 4.0%). In terms of referral source, a majority of the patients in the sample were referred by workers compensation companies ($n= 96$; 38.6%), attorneys ($n= 74$; 29.7%), or physicians ($n = 72$; 28.9%). Most of the patients (94.8%) in this study had known external incentive. Patients were primarily seen in the context of a worker’s compensation claim ($n= 173$; 69.5%) or were involved in a personal injury suit ($n = 48$; 19.3%).

Injury group classification.

Mild traumatic brain injury (mild TBI). Mild brain injury severity was classified based on criteria summarized by Ruff et al., (2009). Specifically, patients were classified as having sustained a mild TBI if they meet the following criteria: 1) at least one of the following: a) loss of consciousness (LOC) of approximately 30 minutes or less; b) posttraumatic amnesia (PTA) not greater than 24 hours; c) documented alteration in mental state at the time of the accident (e.g., feeling “dazed,” confusion, disorientation); and/or d) if present, focal neurological signs that are transient in nature; and 2) an initial Glasgow Coma Scale (GCS) of 13 to 15 after 30 minutes from the time of the injury. These factors cannot be attributable to any non-injury factors such as intoxication, sedation, intubation, or psychological trauma (Ruff et al., 2009).

When neuroimaging data was available, individuals with no neuroradiologic findings or individuals with minor findings were included in the mild TBI group. Examples of minor findings were the presence of a basilar, linear, and/or depressed skull fracture as long as the dura was intact and there were no intracranial abnormalities (e.g., hematoma, cerebral contusion, hemorrhage; Malec et al., 2007). Additionally, patients with significant peripheral damage and/or pain (e.g., broken bones, organ

contusions, etc.) and/or preexisting neurological conditions were not included in the mild TBI group.

Based on validity criteria (see Procedure) and injury characteristics, 71 individuals were included in the mild TBI group. Every individual had evidence of trauma to the head. Of those patients with GCS scores ($n = 57$), one patient had a GCS score of 13, five patients had GCS scores of 14, and 51 had GCS scores of 15 ($M = 14.9$, $s.d. = 0.4$). In terms of loss of consciousness, 39.7% ($n = 27/68$) reported no LOC, 27.9% ($n = 19/68$) reported “questionable” or “brief” (< 5 minutes) LOC, and the remaining patients (32.3%; $n = 22/68$) reported an LOC of less than 30 minutes. Regarding post-traumatic amnesia, data was not available for 32 patients, 26 reported no PTA, 3 reported “brief” (less than one minute) PTA, and 10 reported experiencing PTA for less than one day. Finally, neuroimaging results indicated that two individuals had positive findings on CT scans. Further review of their medical records indicated that both patients had evidence of basilar skull fractures with one patient also having facial fractures. None of the individuals had evidence of brain trauma related to the fractures.

Moderate-severe traumatic brain injury (M/S TBI). Any TBI patient who did not meet the mild TBI criteria stated above were classified as having sustained a moderate-severe TBI. In order to ensure an adequate sample size for statistical analyses, M/S TBI patients evaluated less than six months post-injury were included. Because this group was serving as a comparison group of the effects of neurological insult on objective cognitive tests, including individuals seen before six months post-injury was not expected to significantly influence results.

Forty-five individuals met the injury criteria for inclusion in the M/S TBI group. Of those patients with GCS scores ($n = 36$), patients averaged a score of 8.4 ($s.d. = 3.4$) on GCS. In terms of loss of consciousness, 25.0% ($n = 8/32$) reported LOC lasting for less than one day, 34.4% ($n = 11/32$) for greater than one day but one week or less, and 28.1% for greater than one week. Information was not indicated for 13 patients and four reported being “unsure” as to whether they lost consciousness. Regarding post-traumatic amnesia, data was not available for 25 patients, one patient reported no PTA, three were “unsure,” one experienced PTA for less than one day, and five experienced PTA for more than one day but not greater than one week. Approximately 50% of individuals in this group with documented PTA experienced it for greater than one week. Finally, neuroimaging results indicated that 37 patients had positive findings on CT scans (data was not available for eight patients and one had negative findings on neuroimaging).

Chronic pain (CP). The CP group consisted of patients referred for a pain psychological evaluation related to a reported back injury and who were experiencing chronic pain attributed to their back injury. Any individual who did not have a pain condition directly related to an accident or trauma was excluded from the study. Additionally, individuals who self-reported a co-occurring head injury, exhibited objective evidence of head trauma, or had a history of neurological trauma was excluded. Each patient’s medical records were examined for demonstrable objective abnormalities of the back as indicated by radiological testing (e.g., x-ray, computerized tomography [CT] magnetic resonance imaging [MRI], myelograms, electromyography [EMG], nerve conduction studies [NCS]), and/or surgery.

One hundred and thirty-three individuals met the injury criterion for inclusion in the Chronic Pain group. Eighty-nine percent of the CP sample reported having experienced or were currently experiencing spine pain. Specifically, 32.3%, 15%, 72.9%, and 37.6% reported having cervical, thoracic, lumbar, or sacral pain complaints, respectively. For those individuals that had pain rating scores, “current” pain ratings averaged 6.3 (*s.d.* = 2.0; *n* = 119), “best” pain was rated at 4.4 (*s.d.* = 2.2; *n* = 96), and “worst” pain was rated as a 9.2 (*s.d.* = 1.2; *n* = 102). Despite these relatively high self-reports of pain, only 33.8% of the sample had objective evidence of spinal pathology on imaging studies. Objective findings mainly included: degenerative disc disease (19.5%), herniated nucleus pulposus (3.8%), disc bulge/protrusion (22.6%), or neural impingement (1.5%). Roughly one-third of the sample had undergone at least one surgery – 30.8% received a discectomy/fusion and 15.0% underwent a decompression/laminectomy.

Procedure

The neuropsychological tests and validity measures that were utilized in this study were administered as part of a psychological or neuropsychological assessment battery. Every patient in the sample completed the Wechsler Adult Intelligence Scale-3 (WAIS-3; Wechsler, 1997) and the Minnesota Multiphasic Personality Inventory-2 (MMPI-2; Butcher et al., 1989, 2001). In addition to the WAIS-3 and MMPI-2, patients had to have completed at least two cognitive validity indicators. Because every patient completed the WAIS-3, all had data for the Reliable Digit Span, an embedded cognitive validity indicator. The remaining sample was administered one, or both, forced-choice cognitive validity tests, the Test of Memory Malingering (TOMM; Tombaugh, 1996,

1997) and/or Portland Digit Recognition Test (PDRT; Binder, 1993). Examination of the final sample data showed that 9.2% of the sample completed two cognitive validity measures (7.2% PDRT and RDS; 2.0% TOMM and RDS), while 90.8% of the sample had scores for all three cognitive validity indicators.

Validity Filtering Method. In order to be included in the study, patients had to exhibit valid performance on a range of cognitive and self-report validity indicators. Individuals were initially screened based on their performance on the variable response inconsistency (VRIN) and true response inconsistency (TRIN) scales of the MMPI-2. Any individual who obtained a score of ≥ 80 on either variable was removed from the dataset. Next, each patient was coded in the dataset as being “negative” (0), “indeterminate” (1), or “positive” (2) on each remaining validity indicator (cognitive or self-report) they were administered with “negative” indicating that the individual had passed the validity test. With the exception of RDS, cutoffs used to determine validity classification were based on published cutoffs in the test manuals. Table 1 provides the cutoffs associated with each cognitive and self-report validity indicator.

Table 1.
Cognitive and self-report validity cutoffs used to determine study eligibility

Cognitive Validity Indicators	Positive	Indeterminate	Negative
Portland Digit Recognition Test - Easy	≤ 23	24	≥ 25
Portland Digit Recognition Test - Hard	≤ 20	21	≥ 22
Portland Digit Recognition Test - Total	≤ 45	46	≥ 47
Test of Memory Malingered - Trial 2	≤ 45	46 - 47	≥ 48
Test of Memory Malingered - Retention	≤ 45	46 - 47	≥ 48
Reliable Digit Span	≤ 6	7	≥ 8

Table 1, continued

Self-Report Validity Indicators			
Variable Response Inconsistency	--	--	≤ 79
True Response Inconsistency	--	--	≤ 79
Infrequency scale (F)	≥ 100	80 - 99	≤ 79
Back infrequency scale (Fb)	≥ 110	90 - 109	≤ 89
Infrequency-psychopathology scale (Fp)	≥ 100	70 - 99	≤ 69
Symptom Validity Scale (FBS)	≥ 28	27	≤ 26

A summed validity score was generated for each individual and used to determine study eligibility. To be retained in the dataset, patients must have obtained a total score of “0” or “1” on their summed validity scale score. In other words, an individual had to be negative on all validity indicators (0) or was allowed to have one “indeterminate” (1) score on either a cognitive or self-report validity indicator.

Measures and Variables

Validity indicators.

Cognitive performance validity. The following forced-choice performance validity tests and internal validity indicator were used to exclude from the study those individuals that exhibited poor performance on measures of cognitive validity.

Portland Digit Recognition Test. The PDRT (Binder, 1993) is a 72-item forced-choice cognitive validity test that measures recognition memory. In each trial, the person is presented with a five-digit string of numbers then instructed to count backwards for a set amount of time (distractor period). After the distractor period has ended, the individual is presented with a card displaying two five-digit strings and asked to identify the string previously shown to them. The first 36 items are referred to as the “Easy” trials whereas the last 36 items are referred to as the “Hard” trials. Trials 1

through 18 have a distractor period of five seconds, Trials 19 through 36 have a distractor period of fifteen seconds, and Trials 37 through 72 have a distractor period of 30 seconds.

The cut-off scores used to determine cognitive performance validity were those based on the five and ten percent false positive error rates established in Binder and Kelly's (1996) sample of 120 no-incentive patients with brain dysfunction (see Table 1). It is important to note that a number of patients in the data set qualified for and received the abbreviated version of the PDRT, which is typically indicative of good effort (Doane, Greve, & Bianchini, 2006). However, to further ensure accurate effort classification for individuals receiving this version, the cutoffs used for the "Easy" items on the full version of the test were used to determine patient inclusion.

Test of Memory Malingering. The TOMM (Tombaugh, 1996, 1997) is another forced-choice measure of cognitive performance validity that employs visual recognition of line drawings of 50 common objects. The test consists of two learning trials (Trials 1 and 2) and a retention trial (Retention). During each of the learning trials, individuals are first shown 50 line drawings one at a time. After all 50 pictures are presented, individuals are then asked to identify the previously presented pictures using a two-choice discrimination format. After a fifteen minute delay, individuals are again asked to identify the previously shown pictures using a two-choice discrimination formation. The scores used to determine performance validity were based on the data provided in the TOMM manual (Tombaugh, 1996).

Reliable Digit Span. The RDS (Greiffenstein, Baker, & Gola, 1994) is a well-studied and well-validated embedded validity indicator derived from the Digit Span

subtest of the Wechsler Adult Intelligence Scale or Wechsler Memory Scale. The RDS is calculated by summing the longest forward and backward digit strings repeated without error over two trials. For this study, scores less than 8 were indicative of “indeterminate” validity performance and scores less than 7 were considered invalid (Suhr & Barrash, 2007).

Self-report validity. The following Minnesota Multiphasic Personality Inventory-2 (MMPI-2; Butcher et al., 1989, 2001) validity scales were used to exclude individuals that were exhibiting excessive psychological exaggeration and/or inconsistent responding that would result in an uninterpretable MMPI-2 profile. The entire test consists of 567 true-false items that yield a number of validity scales and ten clinical scales. All validity scale cutoffs described below are ones designated by the MMPI-2 manual.

Variable response inconsistency (VRIN). This validity scale is comprised of 67 pairs of items that were chosen based on their statistical associations and semantic similarities (Nichols, 2001). The individual’s score on this indicator is based on how consistent the individual is with their responses (Friedman, Lewak, Nichols, & Webb, 2001; Greene, 2000). Someone exhibiting valid performance on this scale would answer items that are similar with the same response (i.e. either both true or both false) and items that are opposite with opposite responses (i.e. one true and one false). A *VRIN T-score* of ≥ 80 is considered in indication of inconsistent responding (Butcher et al., 1989, 2001); as such, individuals scoring above this cut-off were be excluded from the study.

True response inconsistency (TRIN). This validity scale is also a measure of

response consistency. It is similar to *VRIN* in that it is also comprised of item pairs (23); however, unlike *VRIN*, the pairs are solely opposite in content (Graham, 2006).

Therefore, two true responses to item pairs or two false responses to item pairs would be an indication that the individual was answering the items indiscriminately which could potentially lead to an invalid profile (Friedman et al., 2001; Greene, 2000). A *TRIN T-score* of ≥ 80 is considered an indication of inconsistent responding (Butcher et al., 1989, 2001).

Infrequency (F) scale. The F scale is a scale used to detect symptom over-reporting and consists of 60 items (Graham, 2006; Greene, 2000). All of the items are located within the first 361 items of the inventory and were chosen because less than ten percent of the MMPI-2 normative sample endorsed them. As such, a high T-score on this scale would mean that an individual endorses a set of symptoms that only a minority of individuals endorse (Nichols, 2001).

Back infrequency scale (Fb). Since the *F scale* deals primarily with item responses in the first 60% of the MMPI-2, this scale was developed to measure the occurrence of infrequent responding on the remaining 40% of the measure. It consists of 40 items that the MMPI-2 normative sample infrequently endorsed.

Infrequency-psychopathology scale (Fp). Given that some individuals taking the MMPI-2 have severe psychological problems, the test developers thought it was important to develop a scale that measured infrequent response styles in both psychiatric and normative samples (Butcher et al., 1989, 2001). Therefore, this scale was developed as a supplemental measure to the *F scale* and consists of 27 items that

are infrequently endorsed (less than twenty percent) by psychiatric inpatients (Friedman et al., 2000; Graham, 2006; Greene, 2000).

Symptom Validity Scale (FBS). The Symptom Validity Scale (previously referred to as the Fake Bad Scale) was developed by Lees-Haley, English, and Glenn (1991) derived from items on the MMPI-2. The 43 items that comprise FBS are sensitive to exaggeration of complaints associated with physical injury as opposed to psychopathology. The FBS is sensitive to an individual's response set that is goal-directed and designed to: 1) appear psychologically normal (except for the influence of the alleged injury); 2) minimize pre-injury psychopathology; and 3) appear honest and present a plausible degree of injury or disability (Larrabee, 1998). FBS has proven to be powerful at detecting exaggeration of physical complaints across a variety of medical and psychological conditions (For reviews see, Greiffenstein, Fox, & Lees-Haley, 2006; Nelson, Sweet, & Demakis, 2006).

Summary. Patients were retained in the sample if they "passed" (i.e. showed valid performance) on cognitive and self-report validity indicators or had no more than one "indeterminate" finding on either a cognitive or self-report validity indicator. Overall, 83.1%, 66.7%, and 69.2% of the mild TBI, M/S TBI, and CP patients, respectively, exhibited valid performance on all validity indicators. In terms of cognitive validity performance, 11.3% of the mild TBI patients, 20.0% of the M/S TBI patients, and 18.0% of the CP patients obtained an "indeterminate" score. For self-report validity, 5.6% of the mild TBI patients, 13.3% of the M/S TBI patients, and 12.8% of the CP patients obtained a score in the "indeterminate" range.

Spontaneously-reported symptoms. During the clinical interview portion of the neuropsychological or pain psychological evaluations, patients were asked to list symptoms that they were experiencing. Patients first provided their symptom report without being cued/prompted (spontaneously-reported) and then were asked about symptom experiences within certain domains. Forty-one possible symptoms (see Table 5 in the Results section for the list of symptoms) were listed in the dataset that could be divided into three main functional domains: cognitive, psychological, and somatic. Each item was coded in the dataset as either being endorsed (“1”) or not (“0”). Four scores were generated from the responses by summing the responses from the cognitive, psychological, and somatic lists, as well as a “total” score for the total number of items endorsed. These variables were then used to see relationships, if any, amongst the other variables of interest in the study. It is important to note that data was available for all TBI patients but only 86 CP patients; therefore, analyses that examined group differences on these variables were based on the smaller sample size.

Psychological variables. Four Minnesota Multiphasic Personality Inventory-II clinical scale *T*-scores were examined in this study. According to the MMPI-2 manual, *T*-scores of 45 to 54 indicate “Average” psychological functioning, 55 to 64, 65 to 74, and greater than 75 are indicative of moderate, high, and very high clinical elevations on these scales, respectively.

Hypochondriasis (Hs; Scale 1). This scale consists of 32 items that are designed to address an individual’s preoccupation and concern over their general bodily functioning (Butcher et al., 2001).

Depression (D; Scale 2). This scale consists of 57 items that measures symptomatic depression. Some items reflect general feelings of demoralization (e.g., discouragement, pessimism, hopelessness) as well as depressive cognitive, somatic, and emotional complaints (Butcher et al., 2001).

Hysteria (Hy; Scale 3). This scale consists of 60 items. Some of the items on this scale are designed to measure specific physical complaints but most of the items measure how one perceives their problems and how they cope with such problems (Butcher et al., 2001). Individuals that exhibit high *T*-scores on this scale are often described as being self-centered with a high need for approval and attention and are often in denial about their maladaptive coping responses when confronted with stress (Friedman et al., 2001; Greene, 2000)

Psychasthenia (Pt; Scale 7). This scale consists of 48 items that characterizes an individual's more generalized anxiety (Friedman et al., 2001). This scale also taps into abnormal fears, self-criticism, difficulties in concentration, and guilty feelings (Greene, 2000).

Working Attention variables. The following two WAIS-3 variables served represented "working attention" in the study. For the purposes of this study, the demographically-adjusted *T*-scores, which adjust for age, education level, ethnicity, and gender were used. Use of the *T*-scores helped control for any influences demographic factors may have had on tests scores as well as provide a way to compare patients' performances to normal.

WAIS-III Working Memory Index. The Working Memory Index (WMI) of the WAIS-III "reflects a memory-related ability that requires the holding of information

'online' so that manipulations or calculations can be performed (analogous to a mental scratch pad)" (Sattler, 2001; p. 387-8) and is considered one of two standardized measures of WM (the other being the WMI of the WMS-3; (Parente et al., 1999). It is comprised of three subtests: Digit Span, Letter-Number Sequencing, and Arithmetic.

WAIS-III Processing Speed Index. The Processing Speed Index (PSI) of the WAIS-3 "reflects a hypothesized performance-related ability involved in perceptual processing and speed as reflected by both mental and psychomotor performance" (Sattler, 2001; p. 388). It is comprised of two subtests, Digit-Symbol-Coding and Symbol Search.

Chapter 3: Results

Validity Indicators

Analyses of variance (ANOVAs) were conducted to examine performance on cognitive and self-report validity indicators as function of clinical group. The groups did not differ on any of the cognitive validity indicators. Significant group differences were found on TRIN, the Fp scale, and FBS of the MMPI-2. Although post-hoc comparisons failed to show significant group differences for TRIN, Tukey B post-hoc comparisons showed that the mild TBI and CP groups had significantly lower scores on Fp and significantly higher scores on FBS than the moderate-severe TBI group. These differences are not in the elevated range although the FBS scores obtained for the mild TBI and CP groups are at the low end of the range that is commonly associated with somatization. Table 2 provides the means, standard deviations, and information from the ANOVAs.

Table 2.

Means, standard deviations, and analyses of variance results for cognitive and self-report validity indicators as a function of clinical group

	Mild TBI		M/S TBI		Chronic Pain		F	p ≤	partial η ²
	M	(sd)	M	(sd)	M	(sd)			
Cognitive									
PDRT Easy*	31.0	(3.0)	31.3	(2.9)	31.4	(2.7)	0.7	ns	.006
TOMM Trial 2	49.8	(0.6)	49.8	(0.4)	49.8	(0.5)	0.3	ns	.003
TOMM Ret	49.7	(0.6)	49.8	(0.4)	49.8	(0.5)	1.1	ns	.009
RDS	9.2	(1.6)	9.5	(1.9)	9.3	(1.7)	0.4	ns	.003
Symptom									
VRIN	52.7	(10.3)	51.9	(9.2)	53.1	(9.6)	0.3	ns	.002
TRIN	56.3	(9.0)	55.9	(9.6)	52.9	(10.6)	3.3	.04	.026
F	55.0	(11.4)	55.8	(10.7)	56.1	(10.6)	0.2	ns	.002
Fb	56.2	(13.8)	53.9	(13.8)	58.0	(14.0)	1.5	ns	.012
Fp	50.5	(7.7) ^a	55.8	(10.0) ^b	51.1	(8.9) ^a	5.9	.003	.046
FBS	21.1	(3.9) ^b	15.5	(5.0) ^a	20.5	(4.3) ^b	27.2	.001	.182

Note. TBI = traumatic brain injury; M/S = moderate-severe; CP = chronic pain; M = mean; sd = standard deviation; PDRT = Portland Digit Recognition Test; TOMM = Test of Memory Malinger; Ret = Retention; RDS = reliable digit span; VRIN = variable response inconsistency; TRIN = true response inconsistency; F = infrequency scale; Fb = back infrequency scale; Fp = infrequency-psychopathology scale; FBS = symptom validity scale

* the Abbreviated PDRT was administered to 101 patients, therefore, only data for the "Easy" trial was available for everybody, and thus, used in the group analyses

^{ab} row means with the same letter are not statistically different from each other

Sample Characteristics

Group Analyses. Descriptive statistics were evaluated for age, education, time since injury, ethnicity, and gender to determine if the groups were significantly different on demographic characteristics. Significant group differences were found for age, education, and time since injury using ANOVAs. Specifically, the mild TBI and CP groups were older than the M/S TBI group, the mild TBI group had the highest average level of education, and both TBI groups exhibited significantly less time between injury

and evaluation than the CP group. Chi-square analyses revealed no significant group differences for gender or race as a function of clinical group.

To control for those differences found to be significant, the demographically-adjusted T-scores, which adjust for gender, race, age, and education, were used for Working Memory and Processing Speed Index scores. To ensure demographic adjustments, correlations between demographic variables and WAIS scores were conducted and found to be non-significant for each clinical group (see Appendix D for the specific correlations). The results of the demographic analyses are presented in Table 3.

Table 3.
Demographic analyses as a function of clinical group

	Mild TBI	M/S TBI	Chronic Pain			<i>partial</i>
	<i>M (sd)</i>	<i>M (sd)</i>	<i>M (sd)</i>	<i>F</i>	<i>p ≤</i>	<i>η²</i>
N	71	45	133			
Age	42.5 (11.7) ^a	33.5 (10.8) ^b	43.1 (9.0) ^a	15.8	.001	.11
Education	12.6 (1.4) ^a	12.3 (1.5) ^{ab}	11.9 (1.3) ^b	6.4	.002	.05
Time Since Inj.	26.0 (18.2) ^a	23.8 (29.5) ^a	37.2 (30.7) ^b	6.00	.003	.04
	%	%	%	<i>χ²</i>	<i>p ≤</i>	
Gender (male)	63.4	75.6	59.4	3.8	ns	
Race (white)	76.1	77.8	77.4	.06	ns	

Note. TBI = traumatic brain injury; M/S = moderate/severe; M = mean; sd = standard deviation; inj. = injury

^{ab} row means with the same letter are not statistically different

Preliminary Analyses

Correlations. Relationships among working attention variables, psychological scales, and spontaneously-reported symptoms (cognitive, psychiatric, somatic) were examined using Pearson correlations. Correlations were calculated for each group

separately so as to see if relationships between variables differed as a function of clinical group. Table 4 presents the results of the correlational analyses.¹

With the exception of a small correlation between Scale 3 (Hysteria) and the WMI in the CP sample ($r = .173, p < .05$), none of the correlations between WAIS variables and MMPI-2 scales were significant for the three groups. In terms of spontaneously-reported symptoms and WAIS variables, a negative correlation existed between spontaneously-reported cognitive symptoms and WMI in the M/S group ($r = -.347, p < .05$), whereas significant relationships between somatic symptoms and WMI scores ($r = -.271, p < .05$) and PSI scores ($r = -.238, p < .05$) were found in the CP group. Correlations were non-significant between any of the spontaneously-reported symptoms and WAIS variables in the mild TBI group.

Larger group correlational differences were observed in relation to spontaneously-reported symptoms and MMPI-2 scales. For the mild TBI group, the only correlation to reach statistical significance was between spontaneously-reported somatic symptoms and MMPI-2 Scale 1 (Hypochondriasis; $r = .250, p < .05$). In contrast, a higher number of significant correlations were observed within the M/S TBI and CP groups. In the M/S TBI group, cognitive symptoms and Scale 7 (Psychasthenia) were positively correlated ($r = .338, p < .05$), and psychological and somatic symptom report rates were positively correlated with all MMPI-2 scales. In the CP group, cognitive symptoms were correlated with Scales 2 (Depression) and 3 and showed a trend towards being associated with Scale 7, psychological symptoms were correlated with Scale 2, and somatic symptoms were correlated with all MMPI-2 scales.

¹ The correlation analyses demonstrating the relationships between 1) WMI and PSI and 2) Hs, D, Hy, Pt with each other are discussed in the modeling portion of the Results but are shown in the comprehensive correlations table.

Table 4.

Pearson product-moment correlations between WMI, PSI, MMPI-2 Scales, and spontaneously-reported symptoms broken down by clinical group

Mild TBI								
	WMI T-score	PSI T-score	Scale 1	Scale 2	Scale 3	Scale 7	Cognitive Sx	Psych. Sx
PSI T-score	.517**							
Scale 1	.012	-.034						
Scale 2	.097	.165	.381**					
Scale 3	.089	.088	.738**	.434**				
Scale 7	.139	.006	.271*	.567**	.359**			
Cognitive Sx	-.003	.022	-.007	-.160	-.089	.035		
Psych. Sx	-.003	-.088	.187	.166	.192	.086	0.154	
Somatic Sx	.132	-.072	.250*	-.017	.106	.009	.372**	-.047
Moderate-Severe TBI								
	WMI T-score	PSI T-score	Scale 1	Scale 2	Scale 3	Scale 7	Cognitive Sx	Psych. Sx
PSI T-score	.327*							
Scale 1	-.042	.055						
Scale 2	-.174	.166	.594**					
Scale 3	-.133	.093	.802**	.740**				
Scale 7	-.088	.171	.616**	.791**	.722**			
Cognitive Sx	-.347*	-.102	.227	.292	.173	.338*		
Psych. Sx	-.064	.227	.470**	.563**	.506**	.637**	.324*	
Somatic Sx	.014	.008	.428**	.496**	.354*	.432**	.223	.337*
Chronic Pain								
	WMI T-score	PSI T-score	Scale 1	Scale 2	Scale 3	Scale 7	Cognitive Sx	Psych. Sx
PSI T-score	.387**							
Scale 1	.038	-.090						
Scale 2	.098	-.051	.475**					
Scale 3	.173*	-.033	.707**	.560**				
Scale 7	.046	-.036	.483**	.695**	.393**			
Cognitive Sx	.025	-.001	.152	.262*	.312**	.210		
Psych. Sx	-.028	-.025	-.060	.300**	.190	.157	.126	
Somatic Sx	-.271*	-.238*	.232*	.281**	.288**	.241*	.236*	.008

Note. TBI = traumatic brain injury; WMI = Working Memory Index; PSI = Processing Speed Index; Scale 1 = hypochondriasis; Scale 2 = depression; Scale 3 = hysteria; Scale 7 = psychasthenia; sx = symptoms.

* correlations are significant at the $p < .05$ level; ** correlations are significant at the $p < .01$ level

Spontaneously-reported symptoms. ANOVAs were conducted to determine if there were group differences in the total number and types of symptoms that patients endorsed. As can be seen from Table 5, the groups statistically differed in their report of symptoms in all three domains and the overall number of symptoms that were endorsed. Overall, the mild TBI group endorsed more symptoms compared to the M/S TBI and CP groups.

Table 5.

Means, standard deviations, and ANOVA results for spontaneously-reported symptoms as a function of clinical group

Symptom Domain	Clinical Group			F	p ≤	partial η ²
	Mild TBI	M/S TBI	Chronic Pain*			
	M (sd)	M (sd)	M (sd)			
Cognitive	1.4 (1.4) ^a	1.3 (1.1) ^a	0.2 (0.6) ^b	29.0	.001	.23
Psychiatric	1.2 (1.3) ^{ab}	0.8 (1.2) ^a	1.7 (1.4) ^b	6.3	.002	.06
Somatic	1.7 (1.2) ^a	1.0 (1.3) ^b	0.8 (0.9) ^b	13.5	.001	.12
Total	4.6 (2.7) ^a	3.6 (2.8) ^b	3.2 (1.9) ^b	6.6	.002	.06

Note. TBI = traumatic brain injury; M/S = moderate/severe; M = mean; sd = standard deviation

* analyses for the CP group was based on $n=86$ (47 missing cases)

^{ab} row means with same letters are not statistically different

Next, chi-square analyses were conducted to examine whether the symptom endorsement rates for individual symptoms varied by clinical group. Table 6 presents the proportion of patients in each clinical group that endorsed specific symptoms and the results of the chi-square analyses. As can be seen, the TBI groups endorsed significantly more cognitive symptoms than the CP group, especially for attention and concentration and recent memory. Interestingly, although not statistically different, a higher percentage of mild TBI patients reported problems for these two symptoms than the M/S TBI group. The groups did not differ on most of the psychiatric symptoms

although a significantly higher proportion of the mild TBI and CP groups endorsed depression than the M/S TBI group. A higher percentage of the CP group endorsed insomnia and sexual dysfunction than the TBI groups. Finally, endorsement rates on somatic symptoms were similarly low across the three groups, with the exception of headaches, in which 64% of the mild TBI group reported experiencing headaches.

Table 6.
Cross-tabs of the proportions of each clinical group endorsing specific cognitive, psychiatric, and somatic symptoms

	Mild TBI	M/S TBI	CP*	χ^2	$p \leq$
<u>Cognitive Symptoms</u>					
attention & concen.	38.0 ^a	28.9 ^a	9.3 ^b	18.6	.001
dysarthria	4.2	2.2	0.0	3.6	ns
stuttering	2.8	0.0	0.0	3.7	ns
exp. language	7.0	8.9	1.2	4.8	ns
comp. language	7.0 ^a	0.0 ^b	1.2 ^b	6.4	.041
word finding	14.1 ^a	8.9 ^a	1.2 ^b	9.6	.008
recent memory	52.1 ^a	60.0 ^a	8.1 ^b	48.7	.001
remote memory	7.1 ^a	11.1 ^a	0.0 ^b	8.8	.012
reading	4.2	6.7	1.2	2.9	ns
computation	2.8	2.2	0.0	2.3	ns
<u>Psychiatric Symptoms</u>					
depression	28.2 ^{ab}	17.8 ^a	41.9 ^b	8.5	.014
anxiety	23.9	8.9	17.4	4.3	ns
emotional lability	1.4	11.1	4.7	5.5	ns
irritability	33.8	35.6	30.2	0.4	ns
hallucinations	1.4	0.0	0.0	1.9	ns
personality change	2.8	2.2	1.2	0.6	ns
insomnia	14.1 ^b	4.4 ^a	36.0 ^c	21.0	.001
sex dysfunction	7.0 ^a	2.2 ^a	27.9 ^b	20.7	.001
suicidal ideation	2.8	0.0	4.7	1.4	ns
panic	1.4	2.2	2.3	0.2	ns

Table 6, continued

<u>Somatic Symptoms</u>					
visual acuity	17.1 ^a	15.6 ^a	3.5 ^b	8.6	.014
diplopia	5.6	6.7	1.2	3.2	ns
triplopia	0.0	0.0	1.2	1.4	ns
blind spots	0.0	0.0	0.0	--	ns
light sensitivity	2.8	2.2	2.3	.05	ns
hearing acuity	4.2	8.9	1.2	4.7	ns
tinnitus	4.2	2.2	0.0	3.6	ns
paresthesias	2.8	0.0	2.3	1.2	ns
smell/taste change	2.8 ^a	15.6 ^b	0.0 ^a	17.5	.001
numbness	16.9 ^b	2.2 ^a	23.3 ^b	9.6	.008
fatigue	7.0	11.1	2.3	4.3	ns
dizziness	29.6 ^a	15.6 ^{ab}	7.0 ^b	14.3	.001
spells	4.2	0.0	0.0	5.6	ns
headaches	63.6 ^a	11.1 ^b	22.1 ^b	43.2	.001
tremors	2.8	0.0.	2.3	1.2	ns
g.i./nausea	7.0	2.2	2.3	2.7	ns
rashes	0.0	0.0	0.0	-	ns
edema	0.0	0.0	2.3	2.7	ns
bloating	0.0	0.0	0.0	--	ns
chest pain	0.0	0.0	1.2	1.4	ns
chills/fever	0.0	2.2	1.2	1.4	ns

Note. TBI = traumatic brain injury; M/S = moderate-severe; CP = chronic pain; X^2 = chi-square; concen. = concentration; exp. = expressive; comp. = comprehensive; g.i. = gastrointestinal problems

*Spontaneously-reported symptom information was not collected for 47 CP patients.

^{abc} row percentages with the same letter are not statistically different

Psychological scales. A MANOVA was performed to examine clinical group differences on psychological variables. Four dependent variables from the MMPI-2 were used: Scales 1 (Hypochondriasis), 2 (Depression), 3 (Hysteria), and 7 (Psychasthenia). Preliminary assumption testing was conducted to check for normality, linearity, outliers, homogeneity of variance-covariance matrices, equality of error variances, and multicollinearity. Assumption violations were noted for homogeneity of covariance

matrices [*Box's M* = 40.5, $F(20, 73633) = 2.7, p \leq .001$] and the error variances associated with Hypochondriasis [$F(2,246) = 8.6, p \leq .001$] and Depression [$F(2,246) = 3.7, p \leq .025$]. Given these violations, Pillai's Trace values were examined and the alpha level for Hypochondriasis and Depression were adjusted for the follow-up ANOVAs.

The results of the MANOVA showed that there was a statistically significant difference between the clinical groups on the combined dependent variables: $F(8,488) = 9.5, p \leq .001$; *Pillai's Trace* = .27; *partial eta*² = .13. Follow-up ANOVAs with Tukey B post-hoc comparisons showed that groups significantly differed on all of the MMPI scales. Generally, the scores for the mild TBI and CP group did not differ from each other and were significantly higher than the scores produced by the M/S TBI group for all of the scales. Refer to Table 7 for a summary of the means, standard deviations, and results from the ANOVAs.

Table 7.

Means, standard deviations and analyses of variance results for MMPI-2 psychological variables as a function of clinical group

	Mild TBI		M/S TBI		Chronic Pain		<i>F</i>	<i>p</i> ≤	<i>partial</i> η^2
	<i>M</i>	(<i>sd</i>)	<i>M</i>	(<i>sd</i>)	<i>M</i>	(<i>sd</i>)			
Hypochondriasis	75.1	(11.6) ^b	60.4	(13.6) ^a	76.1	(8.5) ^b	39.4	.001	.24
Depression	72.2	(10.6) ^b	63.0	(11.4) ^a	73.9	(13.0) ^b	14.0	.001	.10
Hysteria	75.4	(13.1) ^b	58.7	(14.3) ^a	76.4	(14.1) ^b	28.9	.001	.19
Psychasthenia	65.5	(10.8) ^b	54.5	(13.7) ^a	64.2	(13.5) ^b	11.8	.001	.09

Note. TBI = traumatic brain injury; M/S = moderate/severe; M = mean; sd = standard deviation

^{abc} row means and standard deviations with the same letter do not statistically differ

The psychological variables were further analyzed to establish the clinical meaningfulness of the scores. Table 8 shows the percentage of individuals in each clinical group that obtained T-scores of < 65, 65 to 74, and ≥ 75 . The mild TBI and CP groups did not statistically differ on any of the MMPI-2 scales. For three of the scales, Hypochondriasis, Depression, and Hysteria, approximately half of the mild TBI and CP groups obtained scores ≥ 75 , while less than a quarter of the M/S TBI group obtained scores at this level.

The percentage of each group scoring at or above “high” T-scores (≥ 65) and “very high” T-scores (≥ 75) were also examined for combinations of psychological scores. The percentages in the “Combined Scales” section of Table 8 represent individuals that scored at or above the designated T-score level for each of the scales examined. For example, around 75% of individuals in the mild TBI and CP groups showed T-scores ≥ 65 on the Hypochondriasis and Hysteria scales (referred to as “somatization” in the table). As can be seen, the proportions of patients in the mild TBI and CP groups did not statistically differ from each other but did from the proportion of the M/S TBI group on almost all of the combinations that were examined. The exception was the “very high” T-score level for elevations on all the scales; for this variable, only 10% of each of the groups showed elevations.

Table 8.

Cross-tabulations of individuals in each clinical group obtaining scores at various cutoffs on individual MMPI-2 scales and combination scales.

Individual Scales		Mild TBI	M/S TBI	CP	χ^2	$p \leq$
Hypochondriasis	< 65	18.3 ^b	68.9 ^c	5.3 ^a	84.3	.001
	65 to 74	28.2 ^b	8.9 ^a	36.1 ^b		
	≥ 75	53.5 ^b	22.2 ^a	58.6 ^b		
Depression	< 65	23.9 ^a	73.3 ^b	27.8 ^a	36.1	.001
	65 to 74	29.6 ^a	11.1 ^b	26.3 ^a		
	≥ 75	46.5 ^a	15.6 ^b	45.9 ^a		
Hysteria	< 65	21.1 ^a	73.3 ^b	24.1 ^a	43.4	.001
	65 to 74	25.4 ^b	8.9 ^a	21.1 ^b		
	≥ 75	53.5 ^b	17.8 ^a	54.9 ^b		
Psychasthenia	< 65	46.5 ^a	84.4 ^b	51.9 ^a	18.7	.001
	65 to 74	32.4 ^b	6.7 ^a	27.8 ^b		
	≥ 75	21.1	8.9	20.3		
Combined Scales						
Somatization *	≥ 65	74.6 ^b	17.8 ^a	74.4 ^b	51.7	.001
	≥ 75	42.3 ^b	15.6 ^a	42.9 ^b	11.5	.003
Somat. + Dep.	≥ 65	63.4 ^b	13.3 ^a	62.4 ^b	36.2	.001
	≥ 75	25.4	13.3	28.6	4.2	ns
All Scales	≥ 65	40.8 ^b	11.1 ^a	39.8 ^b	13.7	.001
	≥ 75	9.9	8.9	10.5	.10	ns

Note. TBI = traumatic brain injury; M/S = moderate/severe; CP = chronic pain; χ^2 = chi-square; somat. = somatization; dep. = depression

* somatization = scales 1 & 3

^{abc} row percentages with the same letter are not significantly different from each other

Working memory and processing speed. A MANOVA was performed to examine clinical group differences on working memory and processing speed. For all analyses examining WAIS performance, the M/S TBI group was separated into a Moderate TBI ($n = 19$) and Severe TBI group ($n = 26$) so as to determine the effect that injury severity had on WAIS performance.

Preliminary assumption testing was conducted to check for normality, linearity, outliers, homogeneity of variance-covariance matrices, equality of error variances, and multicollinearity. Using a chi-square critical value associated with two dependent variables ($\chi^2_{\text{critical value}} = 13.8$), two individuals were identified as multivariate outliers based on their Mahalanobis distances values (27.2, 15.4). Assumption violations were also noted for homogeneity of covariance matrices [*Box's M* = 24.6, $F(9, 31412) = 2.7$, $p \leq .004$]. Removal of the two outliers did not affect the statistical outcome so they were left in the dataset.

The results of the MANOVA showed that the clinical groups were statistically different on the combined dependent variables: $F(6, 488) = 2.5$, $p < .02$; *Pillai's Trace* = .06; *partial eta*² = .03). Follow-up ANOVAs with Tukey B post-hoc comparisons showed that groups did not significantly differ on WM ($F[3, 246] = 0.9$, $p = \text{ns}$, *partial eta*² = .01) but did significantly differ on PS ($F[3, 246] = 3.5$, $p < .02$, *partial eta*² = .04). Specifically, the Severe TBI group had significantly lower PS scores ($M = 37.9$, $sd = 12.1$) than the other groups (see means and standard deviations for these groups in the t-test table below). Interestingly, the Moderate TBI group scored the highest on PS ($M = 46.2$, $sd = 11.6$) although statistical significance was only observed between the Moderate and Severe TBI groups.

Next, the means of each of the group's scores were examined using t-tests to see if they were significantly different from normal. The results indicated that the mild TBI and CP groups scored significantly lower than normal on WMI while the mild TBI, Severe TBI, and CP groups scored significantly lower than normal on PS.

Subsequently, another set of t-tests were conducted to see if group means were significantly different from the "impairment" cutoff (T-score = 40). For this set of analyses, the Severe TBI group was not significantly different than impairment level on PSI; for the remaining analyses, each of the groups scored significantly higher than a T-score of 40. Table 9 provides the means and standard deviations for each of the variables by clinical group and summarizes the results from the two sets of t-tests.

Table 9.
T-tests examining deviations from normal (T-score = 50) and "impairment" (T-score = 40) level for WMI and PSI T-scores by clinical group

	<i>Mean</i>	<i>(sd)</i>	Test Value = 50		Test Value = 40	
			<i>t-value</i>	<i>p</i> ≤	<i>t-value</i>	<i>p</i> ≤
WMI T-score						
Mild TBI	44.6	(8.4)	-5.5	.001	4.6	.001
Mod TBI	47.7	(8.3)	-1.2	ns	4.0	.001
Sev TBI	46.2	(11.6)	-1.7	ns	2.7	.012
CP	45.3	(8.3)	-6.5	.001	7.3	.001
PSI T-score						
Mild TBI	43.0	(8.4)	-7.0	.001	3.0	.004
Mod TBI	46.2	(11.6)	-1.4	ns	2.3	.031
Sev TBI	37.9	(12.1)	-5.1	.001	-0.9	ns
CP	42.8	(7.9)	-10.6	.001	4.0	.001

Note. Sd = standard deviation; WMI = Working Memory Index; PSI = Processing Speed Index; TBI = traumatic brain injury; M/S = moderate-severe; CP = Chronic Pain

Finally, as with the MMPI variables, group differences were examined via chi-square analyses to see the proportion of patients in each clinical group that scored at or below 1 (T-score ≤ 40), 1.5 (T-score ≤ 35), and 2 (T-score ≤ 30) standard deviations

below the mean (T-score = 50) on the individual variables and the combined set. The proportion of patients in each clinical group was statistically different for PSI scores at each of the examined cutoffs. At each cutoff, the Severe TBI group had a higher percentage of patients scoring at the cutoffs than the other clinical groups. See Table 10 for the percentage of individuals in each clinical group that scored at the designated cutoffs.

Table 10.

Percentage of patients in each clinical group that scored at or below 1, 1.5, or 2 standard deviations below the mean on Working Memory and Processing Speed scores

T-score	Mild TBI	Mod TBI	Sev TBI	CP	χ^2	$p \leq$
WMI \leq 40	29.6	15.8	30.8	31.6	2.0	ns
WMI \leq 35	16.9	10.5	19.2	10.5	2.6	ns
WMI \leq 30	2.8	5.3	7.7	0.8	5.3	ns
PSI \leq 40	32.9	31.6	61.5	44.4	7.6	ns
PSI \leq 35	14.3 ^a	15.8 ^{ab}	38.5 ^b	16.5 ^a	8.2	.042
PSI \leq 30	2.9 ^a	10.5 ^{ab}	19.2 ^b	4.5 ^a	10.4	.015
Both \leq 40	18.6	10.5	26.9	19.5	7.1	ns
Both \leq 35	5.7	5.3	11.5	2.3	8.4	ns
Both \leq 30	1.4 ^{ab}	5.3 ^a	7.7 ^a	0.0 ^b	12.9	.044

Note. TBI = traumatic brain injury; M/S = moderate-severe; CP = Chronic Pain; χ^2 = chi-square; ns = non-significant; WMI = Working Memory Index; PSI = Processing Speed; \leq = less than or equal to

Regressions

Next, six (two per clinical group) sequential hierarchical multiple regressions were conducted to determine if any of the four psychological variables significantly predicted working memory or processing speed performance. The psychological variables were entered in two steps. The Hypochondriasis and Hysteria scales were entered into the first step followed by Depression and Psychasthenia (Step 2).

The only sequential regression to approach statistical significance was the one predicting WM performance in the CP sample. The results of step 1 indicated that the variance accounted for (R^2) with the first two independent variables equaled .04 (adjusted $R^2 = .03$), which showed a trend towards significance ($F [2, 130] = 3.0, p = .053$). Examination of the variables in the first step showed that Hysteria was the only statistically significant predictor ($b = .173, \beta = .292, t = 2.41, p < .02$). The addition of Depression and Psychasthenia in the second step did not improve R^2 ($R^2 = .04$, adjusted $R^2 = .02, F [2,128] = .02$). Interestingly, this relationship appeared to go in the opposite direction as expected as increases in Hysteria were associated with higher WM scores.

Next, six standard multiple regressions were conducted for each clinical group to determine if spontaneously-reported cognitive, psychiatric, or somatic symptoms significantly predicted working memory or processing speed performance. The regression equation predicting WM performance from spontaneously-reported symptoms for the CP sample was the only one to approach statistical significance ($R = .28, R^2 = .08, Adjusted R^2 = .05, F [3,82] = 2.48, p = .067$). Examination of the predictors identified somatic symptoms as a significant predictor of WM score ($b = -2.52, \beta = -.294, t = -2.7, p \leq .008$). This finding indicates that the higher the somatic symptom report, the lower the WM score.

Structural Equation Modeling Analysis

The regressions showed some promising results in regards to the relationship between psychological variables and spontaneously-reported symptoms with working

memory performance. However, regression is limited in the sense that one cannot consider the relationship between predictor variables, variables cannot be latent constructs, and one cannot analyze multiple dependent variables in the same analysis. One significant weakness with conducting regression analyses is their vulnerability to multicollinearity. As can be seen in Table 4, moderately strong to very strong correlations were observed between all of the MMPI-2 scales for each of the clinical groups with the correlation between Hypochondriasis and Hysteria being the largest. The Working Memory and Processing Speed Indices showed weak ($r_{M/S\ TBI} = .327$; $R_{CP} = .387$) to moderate ($r_{mild\ TBI} = .517$) correlations, and thus, did not pose a significant threat to regression analyses.

In order to conduct modeling analyses, the variables being studied have to be related in some way. Examination of the correlation table (Table 4) shows weak correlations existed between the spontaneously-reported symptoms and WAIS variables as well as the MMPI-2 scales and WAIS variables. Nevertheless, given the limits of regression, an exploratory model was analyzed using the Chronic Pain sample in order to examine if the relationships could be better elucidated. The exploratory model examined how predictive somatization (a latent variable with Hypochondriasis and Hysteria as indicators), emotional distress (a latent variable with Depression and Psychasthenia as indicators) and spontaneously-reported somatic symptoms (a measured indicator) were of Working Memory Index and Processing Speed Index T-scores. Specifically, it was hypothesized that higher scores on MMPI-2 scales and higher levels of symptom report would predict decreases in working attention (as represented by working memory and processing speed) performance. After post hoc

model modifications, the resulting model was the best fitting model ($\chi^2 [11] = 14.3$, $p = ns$; $CFI = .98$; $RMSEA = .06$). See Figure 1 for the values associated with the standardized regression paths and covariances. For the purposes of graphically representing the relationships in the M/S TBI group, a similar model was estimated for this group and was found to be a good-fitting model ($\chi^2 [11] = 6.3$, $p = ns$; $CFI = 1.00$; $RMSEA = .00$). Due to the small sample sizes of each of the groups and the exploratory nature of the modeling analyses, models presented in this document are strictly for informational purposes.

Figure 1.
 Model representing the relationship between somatization, emotional distress, and symptom report on working attention in the chronic pain sample

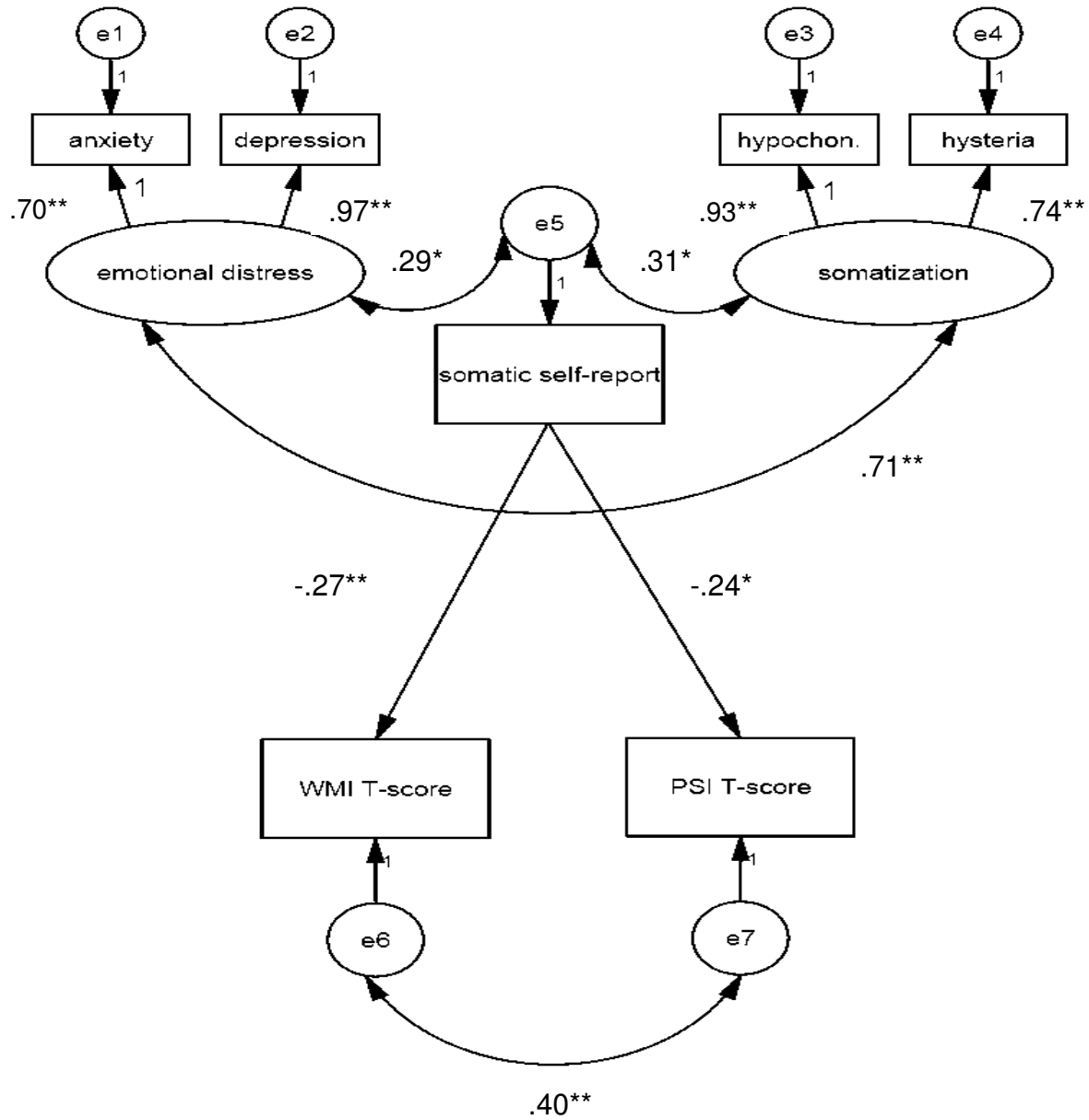
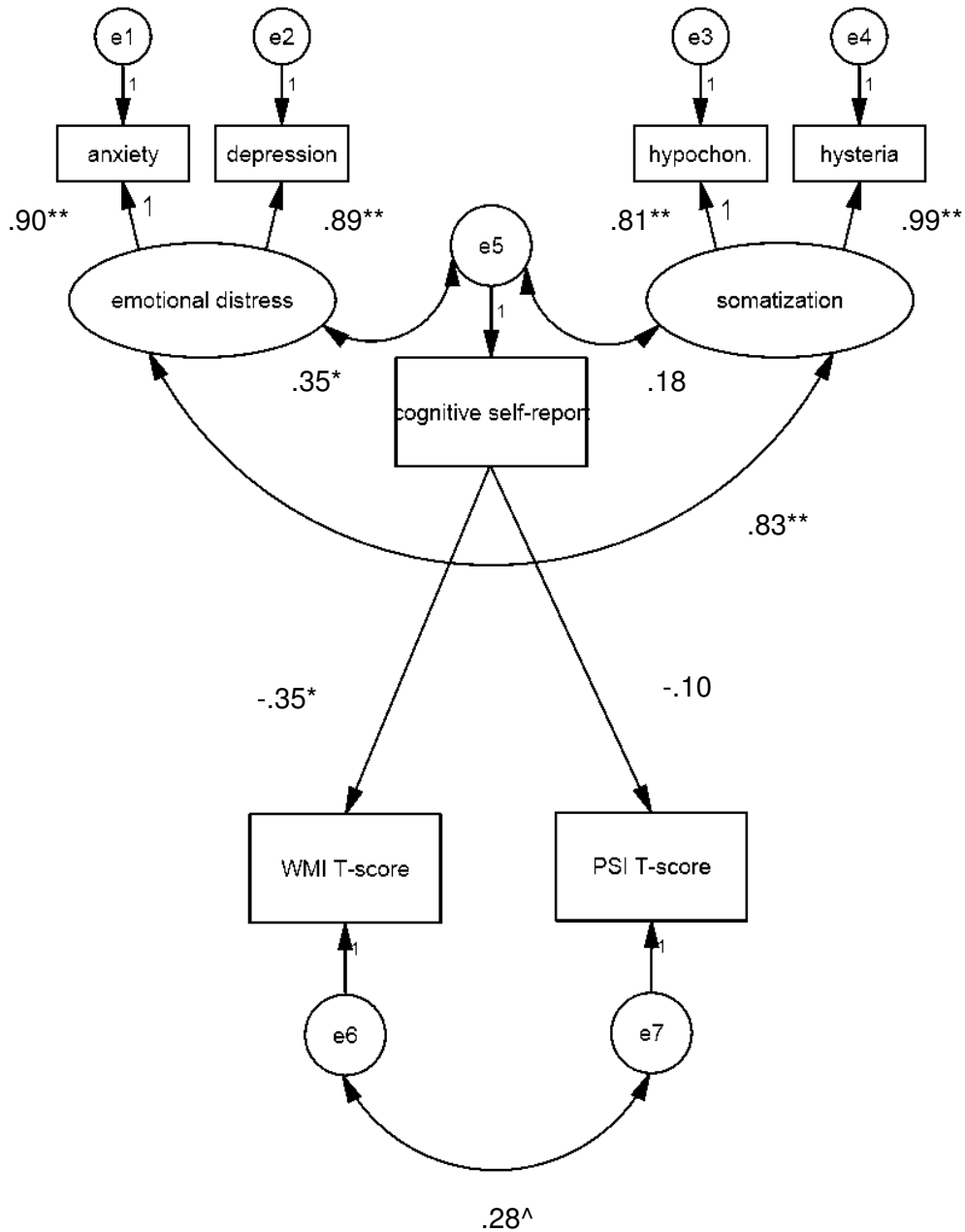


Figure 2.
 Model representing the relationship between somatization, emotional distress, symptom report, and working attention in the M/S TBI group



Follow-up analyses

Pain subgroups. Additional analyses were conducted by breaking the CP up into subgroups to see if different relationships between the variables being studied emerged. The CP group was divided into two groups based on those who did ($n = 88$) and did not ($n = 45$) have objective evidence of spinal pathology. In terms of demographics, the CP/positive findings group was significantly older ($M = 45.7$, $s.d. = 9.0$) than the CP/no findings group ($M = 41.7$, $s.d. = 9.8$; $F [1,131] = 6.0$, $p < .02$, *partial eta*² = .04). There were no significant differences between the groups for education ($M_{CP/no\ findings} = 11.9$, $s.d. = 1.2$; $M_{CP/findings} = 11.9$, $s.d. = 1.5$; $F [1,131] = .02$, $p = ns$, *partial eta*² = .00), time since injury ($M_{CP/no\ findings} = 39.3$, $s.d. = 33.0$; $M_{CP/findings} = 33.0$, $s.d. = 25.6$; $F [1,131] = 1.3$, $p = ns$, *partial eta*² = .01), gender (CP/no findings = 59.1% male; CP/findings = 60.0% male; $X^2 [1] = .01$, $p = ns$), or race (CP/no findings = 73.9% White, CP/findings = 84.4% White; $X^2 [3] = 2.7$, $p = ns$).

In terms of validity hits, a comparable proportion of individuals in each group were negative on all validity indicators ($X^2 [1] = 1.3$, $p = ns$). Specifically, 65.9% of the CP/no findings group and 75.6% of the CP/findings group were negative on all cognitive and self-report indicators. For those that had an “indeterminate” score, 10.2% of the CP/no findings and 17.8% of the CP/findings groups obtained one “indeterminate” score on a self-report validity indicator; these group percentages were not statistically different ($X^2 [1] = 1.3$, $p = ns$). In contrast, 23.9% of the CP/no findings and 6.7% of the CP/findings groups obtained an “indeterminate” score on a cognitive validity indicator, and this did differ by group $X^2 [1] = 6.0$, $p < .02$).

Group performance on individual cognitive and self-report indicators showed that the groups only differed on VRIN and TRIN of the MMPI-2. For each indicator, the CP/findings group had significantly higher scores on both variables ($M_{VRIN} = 56.0$, $s.d. = 10.1$; $F [1,131] = 6.2$, $p < .02$, $partial\ eta^2 = .05$; $M_{TRIN} = 55.5$, $s.d. = 9.3$; $F [1,131] = 4.1$, $p < .05$, $partial\ eta^2 = .03$) than the CP/no findings group ($M_{VRIN} = 51.7$, $s.d. = 9.1$; $M_{TRIN} = 51.6$, $s.d. = 11.1$). However, these group differences were not clinically meaningful as these scores are still reflective of consistent reporting on the MMPI-2.

No significant differences between the pain groups was found for the main variables of interest: spontaneously-reported symptoms ($F [3,82] = .05$, $p = ns$, $partial\ eta^2 = .002$, $Wilk's\ lambda = .998$), the MMPI-2 scales ($F [4,128] = .25$, $p = ns$, $partial\ eta^2 = .008$, $Wilk's\ lambda = .992$), or the WAIS variables ($F [2, 130] = .13$, $p = ns$, $partial\ eta^2 = .002$, $Wilk's\ lambda = .998$).²

Subtle differences were observed for the correlations between WAIS and MMPI-2 variables. None of the correlations between WAIS and MMPI-2 variables were significant for the CP/no findings group. Conversely, the CP/findings group exhibited a marginally significant correlation between WM and Depression ($r = .275$, $p < .07$) and a significant relationship between WM and Hysteria ($r = .386$, $p < .001$). However, Fisher r-to-z calculations showed that the correlations were not significantly different between the groups (WM & Depression: $z = -1.46$, $p = ns$; WM & Hysteria: $z = -1.83$, $p < .07$).

MMPI-2 scale elevations. A MANOVA was performed to examine clinical group differences on the remaining MMPI-2 scales not included in the study. This was done,

² Due to the small sample size that had spontaneously-reported symptom data in the CP/findings group ($n = 15$), chi-square analyses examining frequency of individual symptom report as well as correlations were not conducted. The results (tables) of the chi-squares examining subgroup differences on MMPI-2 scales and WAIS scores are available in Appendix D.

in part, to examine whether mild TBI and CP groups could be characterized more by somatizing and depressive elevations, or by generally elevated psychopathology. Six dependent variables from the MMPI-2 were used: Scales 4 (Psychopathic Deviate [Pd]), 5 (Masculinity-Femininity [Mf]), 6 (Paranoia [Pa]), 8 (Schizophrenia [Sc]), 9 (Hypomania [Ma]), and 0 (Social Introversion [Si]). The results of the MANOVA showed that there was a statistically significant difference between the clinical groups on the combined dependent variables: $F(18, 726) = 1.9, p \leq .02$; *Wilk's lambda* = .9; *partial eta*² = .05. Follow-up ANOVAs with Tukey B post-hoc comparisons showed that groups significantly differed on Paranoia, Schizophrenia, and Social Introversion. The scores for the M/S TBI group were significantly lower than the scores of the remaining groups on each of these scales. Refer to Table 12 for a summary of the means, standard deviations, and results from the ANOVAs.

Prescription use. Finally, relationships among working attention variables and the total number of prescriptions a person reported taking were examined to see if prescription drug usage had any relationship with working memory or processing speed performance. Correlations were calculated for each group separately so as to see if relationships between variables differed as a function of clinical group. None of the correlations for total prescription usage and WMI or PSI scores were significant (CP: $r_{wm} = .043, r_{ps} = -.071$; Mild TBI: $r_{wm} = .006, r_{ps} = -.003$; M/S TBI: $r_{wm} = -.022, r_{ps} = -.011$).

Table 11.

Means, standard deviations, and analyses of variance results for the remaining MMPI-2 psychological scales as a function of clinical group.

	Mild TBI <i>M(sd)</i>	M/S TBI <i>M(sd)</i>	CP/no findings <i>M(sd)</i>	CP/findings <i>M(sd)</i>	<i>F</i>	<i>p</i> ≤	<i>partial</i> η^2
Psycho. Dev.	56.7(9.8)	57.6(10.9)	60.6(11.2)	59.2(12.8)	1.9	ns	.02
Masc. - Fem.	47.0(8.1)	48.0(9.4)	49.7(10.9)	48.7(9.7)	1.1	ns	.01
Paranoia	57.2(10.9) ^{ab}	51.9(10.6) ^a	58.2(13.9) ^b	57.9(13.7) ^b	2.8	.04	.03
Schizophrenia	64.6(11.5) ^b	57.3(12.2) ^a	65.3(13.1) ^b	64.6(12.7) ^b	4.7	.003	.05
Hypomania	52.8(12.9)	52.4(11.5)	51.3(10.6)	52.8(10.7)	0.3	ns	.00
Social Introv.	55.5(10.1) ^b	49.9(9.0) ^a	54.6(10.4) ^b	54.0(10.4) ^{ab}	3.1	.028	.04

Note. TBI = traumatic brain injury; M/S = moderate/severe; CP = chronic pain; Psycho. Dev. = Psychopathic Deviate; Masc. - Fem. = Masculinity-Femininity; Introv. = Introversion
^{ab}: row means with the same letter do not statistically differ

Chapter 4: Discussion

There is a large body of research demonstrating that persisting symptoms after mild brain injury and chronic pain are the results of psychosocial contributors rather than neurological or physiological factors. A large portion of this research has examined the significant role that cognitive and/or symptom exaggeration has on persisting symptoms. In these studies, emphasis is placed on examining how individuals classified as “poor effort” and/or “symptom exaggerators” perform against comparison or normative groups. However, “poor effort” and “symptom exaggeration” does not account for persisting symptoms in all individuals; thus, identifying other psychosocial mechanisms contributing to symptom chronicity has been warranted.

The only way to be able to dissociate genuine levels of psychological impairment someone has post-injury from those that are inflated due to symptom exaggeration is to control for exaggeration. This study sought to uniquely add to the literature by examining the relationships between symptom self-report, scores on psychological scales, and objective cognitive tests of working attention while explicitly controlling for confounding factors such as cognitive and self-report exaggeration, as well as demographic influences. Specifically, the goals of the study included: 1) identify and compare the self-report rates of cognitive, psychiatric, and somatic problems in three populations, mild TBI, M/S TBI, and CP; 2) compare clinical groups' scores on scales representing Hypochondriasis [Scale 1], Depression [Scale 2], Hysteria [Scale 3], and Psychasthenia (anxiety; Scale 7)]; 3) determine the association between subjective report of cognitive problems and objective evidence of working attention function (as measured by the T-scores associated with Working Memory and Processing Speed

Indexes of the WAIS-3) and determine if the associations differed as a function of clinical group; and, 4) examine the association of somatization, depression, and anxiety with subjective and objective evidence of working attention problems and distinguish clinical group differences, if any.

Overview of Findings

One purpose of the study was to examine the extent of working attention deficits within mild TBI, M/S TBI, and CP. Within the TBI groups, the oft-cited positive correlation between injury severity and residual cognitive deficits reported in the literature (Dikmen et al., 1995, 2009; Schretlen & Shapiro, 2003) was evident in this study. In particular, a “dose-response” effect was observed for processing speed performance; mild and moderate TBI patients performed relatively normal (Binder, Iverson, & Brooks, 2009), whereas the severe TBI group exhibited evidence of impaired performance. The processing speed findings corroborate results from other research groups who have found that non-verbal tasks are more sensitive to brain dysfunction than verbal tasks (see Axelrod, Fichtenberg, Leithen, Czamota, & Stucky, 2001; Fisher, Ledbetter, Cohen, Marmor, & Tulskey, 2000; Langeluddecke & Lucas, 2003 for WAIS-3 studies using M/S TBI patients). Comparison of the TBI group’s scores with CP showed that the CP group performed similarly to mild TBI patients and scored within the normal range on working attention. The findings from the three groups support the notion that physiological factors have a residual effect on cognitive performance in severe TBI patients but not in CP or mild TBI groups.

Although the CP and mild TBI groups performed relatively normally, a small proportion of individuals in each group (~ 20 to 30%) scored at or below one standard

deviation below the mean on working memory, processing speed, and the working attention composite. These proportions were not significantly different from the proportions observed in the moderate or severe TBI groups. Since physiological factors most likely do not account for these findings, psychological factors were examined in these groups.

Before examining the impact psychological complications may have had on working attention, it was important to assess the prevalence of psychological elevations in the three groups after carefully controlling for self-report exaggeration. As expected, examination of the group means on the four psychological variables of interest showed that the mild TBI and CP groups did not differ from each other on any of the scales but did significantly differ from the M/S TBI group. The effects for individual scales were highest for Hypochondriasis and Hysteria, followed by Depression, and Psychasthenia.

Inspection of the percentages of individuals in each group scoring at T-scores < 65 (“normal” to “moderate” clinical range), 65 to 74 (“high” clinical range), or ≥ 75 (“very high” clinical range) on individual scales showed that a similarly high percentage of patients in the mild TBI and CP groups showed “high” to “very high” clinical elevations compared to the M/S TBI group, which tended to score in the “normal” to “moderate” clinical ranges. The estimates presented in Table 8 are consistent with the findings of Dersh et al., (2006), Riley, Robins, Geisser, & Wittmer, (1993), Mayer et al., (2008) and Porter-Moffitt et al., (2006). Additionally, elevations on multiple scales were common in the mild TBI and CP groups. Supplemental analyses looking at the remaining six scales of the MMPI-2 showed elevations on Paranoia, Schizophrenia, and Social Introversion for the two groups, but not at the levels observed for Scales 1, 2, and 3, indicating that

psychological complications in these groups are primarily comprised of somatizing and depressive components.

In terms of self-report, it was hypothesized that mild TBI and CP patients would spontaneously-report more problems (particularly cognitive) than the M/S TBI group. This supposition was only partially supported. The mild TBI group reported significantly more “total” symptoms than the M/S TBI patients, which was expected. However, they also reported significantly more “total” symptoms than the CP group, which was an unexpected finding.

Examination of the cognitive domain demonstrated that mild TBI patients reported as many cognitive problems as the M/S group and significantly more cognitive problems than the CP group. When individual cognitive symptoms were examined, the CP group reported problems with attention, concentration and recent memory at significantly lower levels than the mild TBI group. This is in contrast with research reporting high rates of cognitive symptom endorsement in CP patients (see Appendix C for the studies employing CP patients and the prevalence of cognitive symptom report). This divergence in findings may be, in part, due to lack of adequate symptom exaggeration assessment in previous studies utilizing CP patients. Alternately, methodological issues in the current study may have accounted for this discrepancy in findings (see methodological considerations below).

Correlational and regression analyses were conducted to examine the various relationships between MMPI-2 scales, spontaneously-reported symptoms, and working attention performance. The results of the analyses show that although the mild TBI group reported more total symptoms but a similar rate of cognitive symptoms to the M/S

TBI group, these were not associated with psychological elevations on the MMPI-2 or objective cognitive performance. In contrast, significant relationships were observed between self-reported symptoms, MMPI-2 scales, and working memory performance in the M/S TBI group and CP groups.

Interpretation of Findings

What accounts for the difference in findings between the mild TBI and CP groups? The lack of relationship between cognitive symptom report and working attention scores in this study is consistent with a number of studies that have shown either a very small or no relationships between subjective report and objective cognitive functioning when the influence of effort and exaggeration is statistically controlled in mild TBI (Grisart, Van der Linden, & Masquelier, 2002; Mooney & Speed, 2001; Mooney, Speed, & Sheppard, 2005). Since exaggeration was controlled in this study, cognitive symptom report rates and cognitive deficits on objective measures were not inflated to the extent that they are in studies that do not control for exaggeration.

Conversely, psychological factors do appear to influence both symptom report and cognitive performance in the CP group. Researchers in the area of chronic pain have theorized that negative affect (depression, anxiety) and somatization contribute to symptom (in particular, cognitive) chronicity by causing an increased over-focus of physical symptoms which then detrimentally affects other cognitive processes (Brown, 2004; Iezzi et al., 1999; Seminowicz & Davis, 2006; Sullivan et al., 2002; Turk, 2004). In other words, attentional resources are allocated to monitoring their symptoms and this affects their ability to attend to other tasks (Brown, 2000; Grisart et al., 2002).

In a study investigating the attentional functioning in patients diagnosed with fibromyalgia, rheumatoid arthritis, or musculoskeletal pain, 60 percent of the patients had at least one score in the clinically impaired range and all three groups of chronic pain patients had impaired functioning on tests of everyday attention (Dick, Eccleston, & Crombez, 2002). These findings are consistent with earlier findings that showed greater performance deficits on complex attention-demanding tasks in patients with severe chronic pain versus normal controls (Crombez, Eccleston, Baeyens, & Eelen, 1998; Eccleston, 1994, 1995).

Summary

In summary, mild TBI patients reported cognitive symptoms at similar levels to the M/S TBI group; however, symptom report did not translate to working attention deficits and the group as a whole performed within the normal range of performance on this composite. In the M/S TBI group, processing speed appeared to be significantly affected by injury severity while working memory performance averaged around normal. However, symptom report, psychological complications, and working memory scores were significantly correlated with each other implying that psychological issues and associated symptoms may have a detrimental effect on working memory performance in some individuals. A similar pattern of relationships was observed for the CP group. While this group also averaged normal performance on working attention, psychological elevations and an over-focus of somatic symptoms may have distracted/preoccupied some individuals to the extent that attentional performance was slightly affected.

Clinical Implications

This study adds to the literature by providing insight into the cognitive and psychological functioning of individuals who present for clinical evaluations with persisting problems and who have passed cognitive and self-report validity indicators. In agreement with a number of meta-analyses, reviews, and commentaries that have been conducted examining expected neuropsychological outcome after TBI, this study found that mild and moderate TBI patients evaluated for persisting symptoms, and who pass validity markers, exhibit no evidence of objective working attention deficits. The study also shows that CP patients perform similarly to these patients. Therefore, when/if cognitive deficits are reported by a patient, it is most likely that they are accounted for by psychological distress (Binder, 1997; Iezzi et al., 1999; Stulemeijer et al., 2007).

The psychological profiles of the mild TBI and CP patients in this study were marked by elevations on a number of scales, especially scales 1, 2, and 3. Elevations on psychological scales, particularly ones that represent somatization, have been found to be associated with a greater likelihood of future disability (Davis et al., 2000; Graham, 2006; McBeth et al., 2001; Sullivan et al., 2002), higher levels of perceived disability (Alschuler et al., 2008; Seminowicz & Davis, 2006), and poorer response to treatment (Davis et al., 2000; Kidner, Gatchel, & Mayer, 2010).

As such, assessing psychological complications is as necessary as evaluating response validity during an evaluation. This is especially important when someone is experiencing medically unexplained symptoms or symptoms in the absence of medical findings. Ultimately, this information can enable clinicians to identify individuals that are “at risk” for developing persisting symptoms and can serve as a foundation for the

development of preventative techniques (e.g., educating the patient as to expected outcome, developing a therapeutic strategy) that will potentially lessen the likelihood of someone having a poor post-injury outcome and developing a chronic condition.

Considerations and Limitations

This study is not without methodological limitations. First, almost all of the patients in this study were involved in a medico-legal process and therefore, unlikely to be representative of the general population of TBI and CP patients. Being involved in litigation introduces additional psychosocial factors that make it difficult to analyze and interpret the unique influences of individual psychosocial contributors of chronic/persisting symptoms. Therefore, future studies looking at psychological prevalence rates in compensation-seeking and no-incentive pain patients passing self-report validity markers are necessary. On the other hand, the samples utilized in this study are likely representative of patients who fail to recover as expected and therefore, the results of this study can be applied to patients being evaluated in similar contexts/circumstances.

In this study, precautions were taken to filter out individuals in the TBI groups that had significant pain complaints. Nevertheless, two-thirds of the mild TBI sample reported experiencing headaches. This is consistent with other studies which have also found high comorbidity rates of headache in mild TBI patients (Martelli et al., 1999; Mooney et al., 2005; Nicholson & Martelli, 2004; Smith-Seemiller et al., 2003). Future studies should aim to compare this study's findings to groups of patients with low versus high reports of headaches to see if the relationships differ.

Measurement issues in the current study likely contributed to lower symptom report rate, particularly in the CP group. Many of the studies summarized in Appendix C utilized inventories that required the individual to endorse symptom(s) from a list and report on their frequency and severity. In the current study, symptoms were identified from the interview portion of the clinical report and they were coded as “absent” or “present” if the person spontaneously-reported having the problem (i.e. without being cued or prompted). Data entry was dependent on the researcher’s interpretation of the symptom reported; consequently, data entry errors (e.g., mislabeling a symptom as “other” rather than placing it in the “attention/concentration” category) could have contributed to inaccurate symptom report findings. Additionally, there were a disproportionate number of somatic symptoms that were coded in the data set (20 somatic symptoms versus 10 cognitive and 10 psychiatric). Moreover, examination of the somatic symptom list (see Table 6) shows a strong bias towards TBI-related somatic symptoms (e.g., diplopia, smell/taste change, visual/hearing acuity) and a relative lack of pain-related somatic symptoms. Therefore, it is likely that somatically-related complaints were underestimated for the CP group.

It is also worth mentioning that practice effects were not examined in this study. It is not uncommon for individuals being assessed in an incentivized context to undergo multiple evaluations. Since the WAIS is one of the most ubiquitously-used measures in neuropsychological evaluations (Rabin et al., 2005), previous exposure to the test could have familiarized patients with test content, and thus, affected performance on the measure during subsequent evaluations. As such, there is the possibility that some patients’ scores were reflective of prior knowledge of the test material and not

necessarily an indication of “normal” performance. This is especially applicable for Processing Speed scores as Basso, Carona, Lowery, & Axelrod (2002) found that scores on this index significantly improved over 3- and 6- month testing intervals.

In this study, total prescription drug use was not found to be associated with working memory or processing speed scores. However, the calculation may be an underestimate of actual prescription use since calculations were based on the number of drug “categories” an individual reported taking medicine from and not the number of drugs in each category a person may have been prescribed. In other words, a patient may have reported taking two medications but these were counted once in the dataset because both were categorized as “antidepressants.” Future research that examines individual prescription use in more detail, along with combinations of prescription drug use, and how they may affect cognitive performance, is warranted.

Finally, the MMPI-2 is a widely used measure of psychological functioning; however one limitation of the measure is the presence of high correlation between most of the clinical scales. The Minnesota Multiphasic Personality Inventory-2-Restructured Form (MMPI-2-RF; Tellegen & Ben-Porath, 2008) is a revised version of the MMPI-2 consisting of 338 items taken from the MMPI-2 used to restructure or develop new and revised scales. This was done to “preserve the important descriptive properties of the MMPI-2 clinical scales while enhancing their distinctiveness” (Tellegen et al., 2003; p. 10). The restructuring resulted in clinical scales that are considerably less intercorrelated. Therefore, validating this study using the RC scales may help to clarify the role that specific psychological factors have on persisting symptoms.

Conclusion

This study sought to examine the effects that psychological factors have on symptom report and “working attention” performance. As expected, psychological elevations were observed for the mild TBI and CP groups at similarly high clinical levels compared to the M/S TBI comparison group. However, psychological elevations were not significantly associated with symptom self-report and “working attention” performance in the mild TBI, thus implying that persisting problems in this group may be the result of cognitive and symptom exaggeration rather than psychological influences. Contrastingly, scores on psychological scales were significantly associated with self-reported symptoms and working memory performance in the CP group indicating that psychological overlay may detrimentally affect cognitive performance in some individuals. Further research is recommended so as to further elucidate the observed differences between the mild TBI and CP groups.

REFERENCES

- Alexander, M.P. (1992). Neuropsychiatric correlates of persistent postconcussive syndrome. *Journal of Head Trauma Rehabilitation, 7*, 60-69.
- Alexander, M.P. (1995). Mild traumatic brain injury: Pathophysiology, natural history, and clinical management. *American Academy of Neurology, 45*, 1253-1260.
- Allen, L.M., Conder, R.L., Green, P., & Cox, D.R. (1997). *CARB '97 manual for the Computerized Assessment of Response Bias*. CogniSyst, Inc.: Durham, NC.
- Alschuler, K.N., Theisen-Goodvick, M.E., Haig, A.J., & Geisser, M.E. (2008). A comparison of the relationship between depression, perceived disability, and physical performance in persons with chronic pain. *European Journal of Pain, 12*, 757-764.
- American Board of Clinical Neuropsychology (2007). American Academy of Clinical Neuropsychology (AACN) practice guidelines for neuropsychological assessment and consultation. *The Clinical Neuropsychologist, 21*, 209-231.
- American Psychiatric Association. (2000). *Diagnostic and Statistical Manual (4th edition - text revised)*. Washington, DC: American Psychiatric Association.
- Arbisi, P.A., & Butcher, J.N. (2004). Psychometric evidence on detection of malingering of pain: Use of the Minnesota Multiphasic Personality Inventory - 2. *Clinical Journal of Pain, 20*, 383-391.
- Arciniegas, D.B., Anderson, C.A., Topkoff, J., & McAllister, T.W. (2005). Mild traumatic brain injury: A neuropsychiatric approach to diagnosis, evaluation, and treatment. *Neuropsychiatric Disease and Treatment, 1*, 311-327
- Atkinson, R.C. & Shiffrin, R.M. (1968). Human memory: A proposed system and its control processes. In K.W. Spence & J.T. Spence (Eds.). *The Psychology of learning and motivation (vol. 2)*. New York: Academic Press.
- Arpino, L., Iavarone, A., Pariato, C., & Moraci, A. (2004). Prognostic role of depression after lumbar disc surgery. *Neurological Science, 25*, 145-147.
- Axelrod, B.N., Fichtenberg, N.L., Liethen, P.C., Czamoto, M.A., & Stucky, K. (2001). Performance characteristics of postacute traumatic brain injury patients on the WAIS-III and WMS-III. *The Clinical Neuropsychologist, 15*, 516-520.
- Baddeley, A. (1986). Working memory and comprehension. In D. Broadbent, J. McGaugh, M. Kosslyn, N. Mackintosh, E. Tulving, & L. Weiskrantz (Eds.). *Working Memory*. Oxford, England: Oxford University Press.
- Baddeley, A. & Hitch, G. (1974). Working memory. In G. Bower (Ed.). *The psychology of learning and motivation (vol. 8)*. (pp. 47-90). New York: Academic Press.
- Basso, M.R., Carona, F.D., Lower, N., & Axelrod, B.N. (2002). Practice effects on the WAIS-III across 3- and 6- month intervals. *The Clinical Neuropsychologist, 16*, 57-63.
- Behrmann, M. & Haimson, C. (1999). The cognitive neuroscience of visual attention. *Current Opinion in Neurobiology, 9*, 158-163.
- Belanger, H.G., Curtiss, G., Demery, J.A., Lebowitz, B.K., & Vanderploeg, R.D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *Journal of the International Neuropsychological Society, 11*, 215-227.

- Bernstein, D.M. (1999). Subject Review: Recovery from mild head injury. *Brain Injury*, 13, 151-172.
- Bianchini, K.J., Curtis, K.L., & Greve, K.W. (2006). Compensation and malingering in traumatic brain injury: A dose-response relationship? *The Clinical Neuropsychologist*, 20, 831-847.
- Bianchini, K.J., Etherton, J.L., & Greve, K.W. (2004). Diagnosing cognitive malingering in patients with work-related pain: Four cases. *Journal of Forensic Neuropsychology*, 4, 65-85.
- Bianchini, K.J., Greve, K.W., & Glynn, G. (2005). On the diagnosis of malingered pain-related disability: Lessons from cognitive malingering research. *The Spine Journal*, 5, 404-417.
- Bianchini, K. J., Mathias, C. W., & Greve, K. W. (2001). Symptom Validity Testing: a critical review. *The Clinical Neuropsychologist*, 15, 19-45.
- Binder, L.M. (1986). Persisting symptoms after mild head injury: A review of the Postconcussion Syndrome. *Journal of Clinical and Experimental Neuropsychology*, 8, 323-346.
- Binder, L. M. (1993). Portland Digit Recognition Test manual (2nd ed.). Portland, OR: Private Publication.
- Binder, L.M. (1997). A review of mild head trauma. Part II: Clinical Implications. *Journal of Clinical and Experimental Neuropsychology*, 19, 432-457.
- Binder, L.M. (2005). Forensic assessment of medically unexplained symptoms. In G.J. Larrabee (Ed.). *Forensic neuropsychology: A scientific approach* (pp. 298-333). New York: Oxford University Press.
- Binder, L.M. & Kelly, M.P. (1996). Portland Digit Recognition Test performance by brain dysfunction patients without financial incentives. *Psychological Assessment*, 3, 403-409.
- Binder, L.M. & Rohling, M.L. (1996). Money matters: A meta-analytic review of financial incentives on recovery after closed-head injury. *American Journal of Psychiatry*, 153, 7-10.
- Binder, L.M., Iverson, G.L., & Brooks, B.L. (2009). To err is human: "Abnormal" neuropsychological test scores and variability are common in healthy adults. *Archives of Clinical Neuropsychology*, 24, 31-46.
- Binder, L.M., Rohling, M.L., & Larrabee, G.J. (1997). A review of mild head trauma. Part I: Meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology*, 19, 421-431.
- Bohnen, N., Twijnstra, A., & Jolles, J. (1993). Persistence of postconcussional symptoms in uncomplicated mildly head-injured patients: A prospective cohort study. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 6, 193-200.
- Bohnen, N.I., Jolles, J., Twijnstra, A., Mellink, R., & Wijnen, G. (1995). Late neurobehavioral symptoms after mild head injury. *Brain Injury*, 9, 27-33.
- Boone, K. B. (2007). *Assessment of Feigned Cognitive Impairment: A Neuropsychological Perspective*. New York: Guilford Press.
- Boone, K.B. (2009). The need for continuous and comprehensive sampling of effort/response bias during neuropsychological evaluations. *The Clinical Neuropsychologist*, 23, 729-741.

- Brown, R.J. (2000). Psychological mechanisms of medically unexplained symptoms: An integrative conceptual model. *Psychological Bulletin*, 130, 793-812.
- Brown, R.J. (2004). Psychological mechanisms of medically unexplained symptoms: An integrative conceptual model. *Psychological Bulletin*, 130, 793-812.
- Brown, S.C., Glass, J.M., & Park, D.C. (2002). The relationship of pain and depression to cognitive function in rheumatoid arthritis patients. *Pain*, 96, 279-284.
- Brox, J.I., Storheim, K., Holm, I., Friis, A., & Reikeras, O. (2005). Disability, pain, psychological factors and physical performance in healthy controls, patients with sub-acute and chronic low back pain: A case-control study. *Journal of Rehabilitative Medicine*, 37, 95-99.
- Bush, S.S., Ruff, R.M., Troster, A.I., Barth, J.T., Koffler, S.P., Pliskin, N.H....Silver, C.H. (2005). Symptom validity assessment: Practice issues and medical necessity NAN Policy and Planning Committee. *Archives of Clinical Neuropsychology*, 20, 419-426.
- Butcher, J.N. (2001). *The Minnesota Report: Adult Clinical System – Revised*. Minneapolis: Pearson Assessment.
- Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegren, A., & Kaemmer, B. (1989). *MMPI-2: Manual for administration and scoring*. Minneapolis, MN: University of Minnesota.
- Byrne, B.M. (2001). *Structural Equation Modeling with AMOS*. Mahwah, NJ: Lawrence Erlbaum Associates, Inc.
- Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L., et al. (2004). Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, (43 Suppl), 84-105.
- Celestin, J., Edwards, R.R., & Jamison, R.N. (2009). Pretreatment psychosocial variables as predictors of outcomes following lumbar surgery and spinal cord stimulation: A systematic review and literature synthesis. *Pain Medicine*, 10, 639-653.
- Chambers, C.D. & Mattingly, J.B. (2005). Neurodisruption of selective attention: Insights and implications. *Trends in Cognitive Neuroscience*, 9, 542-550.
- Chan, R.C.K. (2001). Base rate of post-concussion symptoms among normal people and its neuropsychological correlates. *Clinical Rehabilitation*, 15, 266-273.
- Chan, R.C.K. (2001). Attentional deficits in patients with post-concussion symptoms : A componential perspective. *Brain Injury*, 15, 71-94.
- Chan, R.C.K. (2002). Attentional deficits in patients with persisting postconcussive complaints: A general deficit or specific component deficit? *Journal of Clinical and Experimental Neuropsychology*, 24, 1081-1093.
- Cicerone, K.D. (1996). Attention deficits and dual task demands after mild traumatic brain injury. *Brain Injury*, 10, 79-89.
- Cicerone, K.D. & Azulay, J. (2002). Diagnostic utility of attention measures in postconcussion syndrome. *The Clinical Neuropsychologist*, 16, 280-289.
- Conder, R.L., Allen, L.M., & Cox, D.R. (1992). *Computerized Assessment of Response Bias*. CogniSyst, Inc.: Durham, NC.

- Crisp, R. (2005). Key factors related to vocational outcome: Trends for six disability groups. *Journal of Rehabilitation, 71*, 30-37.
- Crombez, G., Eccleston, C., Baeyens, F., & Eelen, P. (1998). Attentional disruption is enhanced by the threat of pain. *Behaviour Research and Therapy, 36*, 195-204.
- Curtis, K. L., Greve, K. W., & Bianchini, K. J. (October, 2006). Attention and memory dysfunction in pain patients while controlling for effort on the California Verbal Learning Test - II. *Archives of Clinical Neuropsychology, 21*, 544-545.
- Davis, P.J., Reeves, J.L., Hastie, B.A., Graff-Radford, S.B., & Naliboff, B.D. (2000). Depression determines illness conviction and pain impact: A structural equation modeling analysis. *Pain Medicine, 1*, 238-246.
- Dersh, J., Gatchel, R. J., Mayer, T., Polatin, P., & Temple, O. R. (2006). Prevalence of psychiatric disorders in patients with chronic disabling occupational spinal disorders. *Spine, 31*, 1156-1162.
- Dick, B., Eccleston, C., & Crombez, G. (2002). Attentional functioning in fibromyalgia, rheumatoid arthritis, and musculoskeletal pain patients. *Arthritis and Rheumatism, 47*, 639-644.
- Dikmen, S.S., Temkin, N.R., Machamer, J.E., Holubkov, A.L., Fraser, R.T., & Winn, H.R. (1994). Employment following traumatic head injuries. *Archives of Neurology, 51*, 177-186.
- Dikmen, S.S., Machamer, J.E., Winn, H.R., & Temkin, N.R. (1995). Neuropsychological outcome at 1-year post head injury. *Neuropsychology, 9*, 80-90.
- Dikmen, S.S., Corrigan, J., Levin, H.S., Machamer, J., Stiers, W., & Weiskopf, M.G. (2009). Cognitive outcome following traumatic brain injury. *Journal of Head Trauma Rehabilitation, 24*, 1-9.
- DiStefano, G. & Radanov, B.P. (1995). Course of attention and memory after common whiplash: A two-year prospective study with age, education, and gender pair-matched patients. *Acta Neurologica Scandinavica, 91*, 346-352.
- Driver, J. (2001). A selective review of selective attention research from the past century. *British Journal of Psychology, 92*, 53-78.
- Dunn, J.T., Lees-Haley, P.R., Brown, R.S., Williams, C.W., English, L.T. (1995). Neurotoxic complaint base rates of personal injury claimants: Implications for neuropsychological assessment. *Journal of Clinical Psychology, 51*, 577-584.
- Eccleston, C. (1994). Chronic pain and attention: A cognitive approach. *British Journal of Clinical Psychology, 33*, 535-547.
- Eccleston, C. (1995). Chronic pain and distraction: An experimental investigation into the role of sustained and shifting attention in the processing of chronic persistent pain. *Behavioral Research and Therapy, 33*, 391-405.
- Eccleston, C. & Crombez, G. (1999). Pain demands attention: A cognitive-affective model of interruptive function of pain. *Psychological Bulletin, 125*, 356-366.
- Evered, L., Ruff, R., Baldo, J., & Isomura, A. (2003). Emotional risk factors and postconcussional disorder. *Assessment, 10*, 420-427.
- Fee, C.R.A. & Rutherford, W.H. (1988). A study of the effect of legal settlement on post-concussion symptoms. *Archives of Emergence Medicine, 5*, 12-17.
- Fishbain, D.A., Lewis, J.E., Gao, J., Cole, B., & Rosomoff, R.S. (2006). Chronic pain and the measurement of personality: Do states influence traits? *Pain Medicine, 7*, 509-529.

- Fishbain, D.A., Lewis, J.E., Gao, J., Cole, B., & Rosomoff, R.S. (2009). Is chronic pain associated with somatization/hypochondriasis? An evidence-based structured review. *Pain Practice, 9*, 449-467.
- Fisher, D.C., Ledbetter, M.F., Cohen, N.J., Marmor, D., & Tulskey, D.S. (2000). WAIS-III/WMS-III profiles for mildly to severely brain-injured patients. *Applied Neuropsychology, 7*, 126-132.
- Fox, D.D., Lees-Haley, P.R., Earnest, K., & Dolezal-Wood, S. (1995a). Post-Concussive symptoms: Base rates and etiology in psychiatric patients. *The Clinical Neuropsychologist, 9*, 89-92.
- Fox, D.D., Lees-Haley, Earnest, K., & Dolezal-Wood, S. (1995b). Base rates of postconcussive symptoms in health maintenance organization patients and controls. *Neuropsychology, 9*, 606-611.
- Frenchman, K.A.R., Fox, A.M. & Maybery, M.T. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychology, 27*, 33-351.
- Friedman, A.F., Lewak, R., Nichols, D.S., & Webb, J.T. (2001). *Psychological Assessment with the MMPI-2*. Mahwah, New Jersey: Lawrence Erlbaum Associates, Inc.
- Gaetz, M. (2004). The neurophysiology of brain injury. *Clinical Neurophysiology, 115*, 4-18.
- Garden, N. & Sullivan, K.A. (2010). An examination of the base rates of post-concussion symptoms: The influence of demographics and depression. *Applied Neuropsychology, 17*, 1-7.
- Garden, N., Sullivan, K.A., Lange, R.T. (2010). The relationship between personality characteristics and postconcussion symptoms in a nonclinical sample. *Neuropsychology, 24*, 168-175.
- Gasquoine, P.G. (2000). Postconcussional symptoms in chronic pain. *Applied Neuropsychology, 7*, 83-89.
- Gatchel, R.J. (2004). Psychosocial factors that can influence the self-assessment of function. *Journal of Occupational Rehabilitation, 14*, 197-206.
- Gatchel, R.J. & Dersh, J. (2002). Psychological disorders and chronic pain: Are there cause and effect relationships. In D.C. Turk & R.J. Gatchel (Eds.), *Psychological Approaches to Pain Management: A practitioner's handbook* (2nd ed., pp. 30-51). New York: Guilford Press.
- Gatchel, R.J., Mayer, T.G. & Eddington, A. (2006). MMPI disability profile: The least known, most useful screen for psychopathology in chronic occupational spinal disorders. *Spine, 31*, 2973-2978.
- Gatchel, R. J., Bernstein, D., Stowell, A. W., & Pransky, G. (2008). Psychosocial differences between high-risk acute vs. chronic low back pain patients. *Pain Practice, 8*, 91-97.
- Geisser, M.E., Robinson, M.E., Keefe, F.J., & Weiner, M.L. (1994). Catastrophizing, depression, and the sensory, affective and evaluating aspects of chronic pain. *Pain, 59*, 79-84.
- Geisser, M.E., Robinson, M.E., Miller, Q.L., & Bade, S.M. (2003). Psychosocial factors and functional capacity evaluation among persons with chronic pain. *Journal of Occupational Rehabilitation, 13*, 259-276.

- Gervais, R.O., Rohling, M.L., Green, P., & Ford, W. (2004). A comparison of WMT, CARB, and TOMM failure rates in non-head injury disability claimants. *Archives of Clinical Neuropsychology*, *19*, 475-487.
- Goldstein, E.B. (2002). *Sensation and Perception (6th ed.)*. Pacific Grove, CA: Wadsworth Group.
- Gouvier, W.D., Cubic, B., Jones, G., Brantley, P., & Cutlip, Q. (1992). Postconcussion symptoms and daily stress in normal and head-injured college populations. *Archives of Clinical Neuropsychology*, *7*, 193-211.
- Graham, J.R. (2006). *MMPI-2. Assessing Personality and Psychopathology (4th ed.)*. New York: Oxford University Press.
- Green, P. & Iverson, G.L. (1998). Exaggeration of anosmia in 80 litigating head injury cases. *Archives of Clinical Neuropsychology*, *13*, 138.
- Green, P. & Iverson, G.L. (2001). Effects of injury severity and cognitive exaggeration on olfactory deficits in head injury compensation claims. *Neurorehabilitation*, *16*, 237-243.
- Green, P., Allen, L.M., & Astner, K. (1996). *The Word Memory Test: A user's guide to the oral and computer-administered forms, US version 1.1*. CogniSyst, Inc.: Durham, NC.
- Green, P., Rohling, M.L., Iverson, G. L. & Gervais, R.O. (2003). Relationships between olfactory discrimination and head injury severity. *Brain Injury*, *17*, 479-496.
- Green, P., Rohling, M.L., Lees-Haley, P.R. & Allen, L.M. (2001). Effort has a greater effect on test scores than severe brain injury in compensation claimants. *Brain Injury*, *15*, 1045-1060.
- Greene, R.L. (2000). *The MMPI-2: An Interpretive Manual (2nd ed.)*. Needham Heights, Massachusetts: Allyn & Bacon.
- Greiffenstein, M.F., Baker, W.J. (2001a). Comparison of premorbid and postinjury MMPI-2 profiles in late postconcussion claimants. *The Clinical Neuropsychologist*, *15*, 162-170.
- Greiffenstein, M.F., & Baker, W.J. (2001b). Descriptive analysis of premorbid MMPI-2 profiles in chronic postconcussive claimants. *Journal of the International Neuropsychological Society*, *7*, 181.
- Greiffenstein, M.F., Baker, W.J., & Gola, T. (1994). Validation of malingered amnesic measures with a large clinical sample. *Psychological Assessment*, *6*, 218-224.
- Greiffenstein, M.F., Fox, D., & Lees-Haley, P.R. (2007). The MMPI-2 Fake Bad Scale in detection of noncredible brain injury claims. In K.B. Boone (Ed.). *Assessment of feigned cognitive impairment*, (pp. 210-235. New York: The Guilford Press.
- Grisart, J., Van der Linden, M., & Masquelier, E. (2002). Controlled processes and automaticity in memory functioning in fibromyalgia patients: Relation with emotional distress and hypervigilance. *Journal of Clinical and Experimental Neuropsychology*, *24*, 994-1009.
- Gunstad, J. & Suhr, J.A. (2004). Cognitive factors in Postconcussion Syndrome symptom report. *Archives of Clinical Neuropsychology*, *19*, 391-405.
- Guo, H., Tanaka, S., Cameron, L.L., Seligman, P.J., Behrens, V.J., Ger, J., Wild, D.K., Putz-Anderson, V. (1995). Back pain among workers in the United States: National estimates and workers at high risk. *American Journal of Industrial Medicine*, *28*, 591-602.

- Guo, H., Tanaka, S., Halperin, W.E., Cameron, L.L. (1999). Back pain prevalence in US industry and estimates of lost workdays. *American Journal of Public Health, 89*, 1029-1035.
- Hart, R.P., Martelli, M.F., & Zasler, N.D. (2000). Chronic pain and neuropsychological functioning. *Neuropsychology Review, 10*, 131-149.
- Hart, R.P., Wade, J.B., & Martelli, M.F. (2003). Cognitive impairment in patients with chronic pain: The significance of stress. *Current Pain and Headache Reports, 7*, 116-126.
- Heilbronner, R.L., Sweet, J.J., Morgan, J.E., Larrabee, G.J., Millis, S.R. & Conference Participants (2009). American Academy of Clinical Neuropsychology Consensus Conference Statement on the neuropsychological assessment of effort, response bias, and malingering. *The Clinical Neuropsychologist, 23*, 1093-1129.
- Hess, D.W., Marwitz, J.H., & Kreutzer, J.S. (2003). Neuropsychological impairments after spinal cord injury: A comparative study with mild traumatic brain injury. *Rehabilitation Psychology, 48*, 151-156.
- Iezzi, T., Archibald, Y., Barnett, P., Klinck, A., & Duckworth, M. (1999). Neurocognitive performance and emotional status in chronic pain patients. *Journal of Behavioral Medicine, 22*, 205-216.
- Ingebrigsten, T. (1998). Quantification of post-concussion symptoms 3 months after minor head injury in 100 consecutive patients. *Journal of Neurology, 245*, 609-612.
- Iverson, G.L. (2005). Outcome from mild traumatic brain injury. *Current Opinions in Psychiatry, 18*, 301-317.
- Iverson, G.L. (2006). Misdiagnosis of the persistent syndrome in patients with depression. *Archives of Clinical Neuropsychology, 21*, 303-310.
- Iverson, G.L. (2007). Identifying exaggeration and malingering. *Pain Practice, 7*, 94-102.
- Iverson, G.L. (2010). Mild traumatic brain injury meta-analyses can obscure individual differences. *Brain Injury, 24*, 1246-1255.
- Iverson, G.L. & Lange, R.T. (2003). Examination of "Postconcussion-Like" symptoms in a healthy sample. *Applied Neuropsychology, 10*, 137-144.
- Iverson, G.L. & McCracken, L.M. (1997). 'Postconcussive' symptoms in persons with chronic pain. *Brain Injury, 11*, 783-790.
- Iverson, G.L., Brooks, B.L., Collins, M., & Lovell, M.R. (2006). Tracking neuropsychological recovery following concussion in sports. *Brain Injury, 20*, 1335-1344.
- Iverson, G.L., King, R.J., Scott, J.G., & Adams, R.L. (2001). Cognitive complaints in litigating patients with head injuries or chronic pain. *Journal of Forensic Neuropsychology, 2*, 21-32.
- Iverson, G.L., Lange, R.T., Brooks, B.L., & Rennison, V.L. A. (2010). "Good old days" bias following mild traumatic brain injury. *The Clinical Neuropsychologist, 24*, 17-37.
- Jacobson, R.R. (1995). The post-concussional syndrome: Physiogenesis, psychogenesis, and malingering. An integrative model. *Journal of Psychosomatic Research, 39*, 675-693.
- Jarrold, C. & Towse, J.N. (2006). Individual differences in working memory. *Neuroscience, 139*, 39-50.

- Jones, R.D. & Rizzo, M. (2004). Head trauma and traumatic brain injury. In M. Rizzo & P.J. Eslinger (Eds.). *Principles and practice of behavioral neurology and neuropsychology* (pp. 615-634). Philadelphia, PA: Elsevier, Inc.
- Karzmark, P., Hall, K., & Englander, J. (1995). Late-onset post-concussion symptoms after mild brain injury: The role of premorbid, injury-related, environmental, and personality factors. *Brain Injury, 9*, 21-26.
- Kashluba, S., Casey, J.E., & Paniak, C. (2006). Evaluating the utility of ICD-10 diagnostic criteria for postconcussion syndrome following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 12*, 111-118.
- Keefe, F.J., Rumble, M.E., Scipio, C.D., Giordano, L.A., & Perri, L.M. (2004). Psychological aspects of persistent pain: Current state of the science. *The Journal of Pain, 5*, 195-211.
- Kendall, E. (2003). Predicting vocational adjustment following traumatic brain injury: A test of psychosocial theory. *Journal of Vocational Rehabilitation, 19*, 31-45.
- Kendall, E. & Terry, D.J. (1996). Psychosocial adjustment following closed head injury: A model for understanding individual differences and predicting outcome. *Neuropsychological Rehabilitation, 6*, 101-132.
- Kennedy, J.E., Clement, P.F., & Curtiss, G. (2003). WAIS-III processing speed index scores after TBI: The influence of working memory, psychomotor speed, and perceptual processing. *The Clinical Neuropsychologist, 17*, 303-307.
- Kessler, R.C., Berglund, P., Demler, O., Jin, R., & Walters, E.E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry, 62*, 593-602.
- Kidner, C., Gatchel, R., & Mayer, T. (2010). MMPI disability profile is associated with degree of opioid use in chronic work-related musculoskeletal disorders. *The Clinical Journal of Pain, 26*, 9-15.
- Krieter, S. & Niev, D. (2007). Pain: Clinical updates. *International Association for the Study of Pain, XV*, 1-4.
- Kwok, F.Y., Lee, T.M.C., Leung, C.H.S., & Poon, W.S. (2008). Changes in cognitive functioning following mild traumatic brain injury over a 3-month period. *Brain Injury, 22*, 740-751.
- Lange, R.T., Iverson, G.L. & Rose, A. (2011). Depression strongly influences postconcussion symptom reporting following mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 26*, 127-137.
- Lange, R.T., Iverson, G.L., Brooks, B.L., & Rennison, V.L.A. (2010). Influence of poor effort on self-reported symptoms and neurocognitive test performance following mild traumatic brain injury. *The Journal of Clinical and Experimental Neuropsychology, 32*, 961-972.
- Langeluddecke, P.M., & Lucas, S.K. (2003). Wechsler Adult Intelligence Scale – Third Edition findings in relation to severity of brain injury in litigants. *The Clinical Neuropsychologist, 17*, 273-284.
- Larrabee, G.J. (2003b). Exaggerated MMPI-2 symptom report in personal injury litigants with malingered neurocognitive dysfunction. *Archives of Clinical Neuropsychology, 18*, 673-686.
- Larrabee, G.J. (2003c). Exaggerated pain in litigants with malingered neurocognitive dysfunction. *The Clinical Neuropsychologist, 17*, 395-401.

- Larrabee, G.J. (2005). Assessment of malingering. In G. J. Larrabee (ed.), *Forensic neuropsychology: a scientific approach* (pp. 115-158). New York, NY: Oxford University Press.
- Larrabee, G.J. & Berry, D.T.R. (2007). Diagnostic classification statistics and diagnostic validity of malingering assessment. In G.J. Larrabee (Ed.), *Assessment of malingered neurocognitive* (pp. 14-26). New York: Oxford University Press.
- Lamberty, G. L. (2008). *Understanding somatization in the practice of neuropsychology*. New York: Oxford University Press.
- Lavie, N. (2005). Distracted and confused? Selective attention under load. *Trends in Cognitive Neuroscience*, 9, 75-82.
- Lavie, N. (2006). The role of perceptual load in visual awareness. *Brain Research*, 1080, 91-100.
- Lebovits, A.H. (2000). The psychological assessment of patients with chronic pain. *Current Review of Pain*, 4, 122-126.
- Lees-Haley, P.R. & Brown, R.S. (1993). Neuropsychological complaint base rates of 170 personal injury claimants.
- Lees-Haley, P. R., English, L. T., & Glenn, W. J. (1991). A fake bad scale for the MMPI-2 for personal injury claimants. *Psychological Reports*, 68, 203-210.
- Lees-Haley, P.R., Fox, D.D., & Courtney, J.C. (2001). A comparison of complaints by mild brain injury claimants and other claimants describing subjective experiences immediately following their injury. *Archives of Clinical Neuropsychology*, 16, 689-695.
- Linton, S.J. (2000). A review of the psychological risk factors in back and neck pain. *Spine*, 25, 1148-1156.
- Lishman, W.A. (1988). Physiogenesis and psychogenesis in the 'post-concussional syndrome.' *British Journal of Psychiatry*, 153, 460-469.
- Lynch, W.J. (2004). Determination of effort level, exaggeration, and malingering in neurocognitive assessment. *Journal of Head Trauma Rehabilitation*, 19, 277-283.
- MacLeod, A.D.S. (2010). Post concussion syndrome: The attraction of the psychological by the organic. *Medical Hypotheses*, 74, 1033-1035.
- Malec, J.F., Brown, A.W., Leibson, C.L., Flaada, J.T., Mandrekar, J.N., Diehl, N.N. et al. (2007). The Mayo classification system for traumatic brain injury severity. *Journal of Neurotrauma*, 24, 1417-1424.
- Malojic, B., Mubrin, Z., Coric, B., Susnic, M., & Spilich, G.J. (2008). Consequences of mild traumatic brain injury on information processing assessed with attention and short-term memory tasks. *Journal of Neurotrauma*, 25, 30-37.
- Martelli, M.F., Grayson, R.L., & Zasler, N.D. (1999). Posttraumatic headache: Neuropsychological and psychological effects and treatment implications. *Journal of Head Trauma Rehabilitation*, 14, 49-69.
- Mathias, J.L., Beall, J.A., & Bigler, E.D. (2004). Neuropsychological and information processing deficits following mild traumatic brain injury. *Journal of the International Neuropsychological Society*, 10, 286-297.
- Mayer, T. G., Towns, B. L., Neblett, R., Theodore, B. R., & Gatchel, R. J. (2008). Chronic widespread pain in patients with occupational spinal disorders: prevalence, psychiatric comorbidity, and association with outcomes. *Spine (Phila Pa 1976)*, 33, 1889-1897.

- McBeth, J., Macfarlane, G.J., Benjamin, S., & Silman, A.J. (2001). Features of somatization predict the onset of chronic widespread pain. *Arthritis and Rheumatism*, *44*, 940-946.
- McCallister, T.W., & Arciniegas, D. (2002). Evaluation and treatment of postconcussive symptoms. *NeuroRehabilitation*, *17*, 265-283.
- McCracken, L.M., & Iverson, G.L. (2001). Predicting complaints of impaired cognitive functioning in patients with chronic pain. *Journal of Pain Symptom Management*, *21*, 392-396.
- McCrea, M. (2008). *Mild traumatic brain injury and postconcussion syndrome*. New York: Oxford University Press.
- McCrea, M., Iverson, G.L., McAllister, T.W., Hammeke, T.A., Powell, M.R., Barr, W.B., & Kelly, J.P. (2009). An integrated review of recovery after mild traumatic brain injury (MTBI): Implications for clinical management. *The Clinical Neuropsychologist*, *23*, 1368-1390.
- Mendelson, G. (1984). Follow-up studies of personal injury litigants. *International Journal of Law and Psychiatry*, *7*, 179-187.
- Mendelson, G. (1995). Compensation neurosis' revisited: Outcome studies of the effects of litigation. *Journal of Psychosomatic Research*, *39*, 695-706.
- Mersky, H., Bogduk, N. (Eds.). (1994) *Classification of chronic pain (2nd ed.)*. Seattle, WA: IASP Press (pp. 209-214).
- Meyers, J.E. & Diep, A. (2000). Assessment of malingering in chronic pain patients using neuropsychological tests. *Applied Neuropsychology*, *7*, 133-139.
- Meyers, J.E. & Rohling, M.L. (2004). Validation of the Meyers Short Battery on mild TBI patients. *Archives of Clinical Neuropsychology*, *19*, 637-651.
- Meyers, J.E., Volbrecht, M., Axelrod, B.N., Reinsch-Boothby, L. (2011). Embedded symptom validity tests and overall neuropsychological test performance. *Archives of Clinical Neuropsychology*, *26*, 8-15.
- Mickeviciene, D., Scradar, H., Obelieniene, D., Surkiene, D., Kunickas, R., Stover, L.J., & Sand, T. (2004). A controlled prospective inception cohort study on the post-concussion syndrome outside the medicolegal context. *European Journal of Neurology*, *11*, 411-419.
- Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine. Definition of mild traumatic brain injury. (1993). *Journal of Head Trauma Rehabilitation*, *8*, 86-87.
- Miller, L. (2001). Not just malingering: Syndrome diagnosis in traumatic brain injury litigation. *Neurorehabilitation*, *16*, 109-122.
- Miller, L.J. & Donders, J. (2001). Subjective symptomatology after traumatic head injury. *Brain Injury*, *15*, 297-304.
- Mittenberg, W., & Strauman, S. (2000). Diagnosis of mild head injury and the Postconcussion syndrome. *Journal of Head Trauma Rehabilitation*, *15*, 783-791.
- Mittenberg, W., DiGiulio, D., Perrin, S., & Bass, A.E. (1992). Symptoms following mild head injury: Expectation as aetiology. *Journal of Neurology, Neurosurgery, and Psychiatry*, *55*, 200-204.
- Mittenberg, W., Patton, C., Canyock, E.M., & Condit, D.C. (2002). Base rates of malingering and symptom exaggeration. *Journal of Clinical and Experimental Neuropsychology*, *24*, 1094-1102.

- Mooney, G. & Speed, J. (2001) The association between mild traumatic brain injury and psychiatric conditions. *Brain Injury*, 15, 865-877.
- Mooney, G., Speed, J. & Sheppard, S. (2005). Factors related to recover about mild traumatic brain injury. *Brain Injury*, 19, 975-987.
- Moore, E.L., Terryberry-Spohr, L., & Hope, D.A. (2006). Mild traumatic brain injury and anxiety sequelae: A review of the literature. *Brain Injury*, 20, 117-132.
- Nichols, D.S. (2001). *Essentials of MMPI-2 Assessment*. New York: John Wiley & Sons, Inc.
- Nicholson, K. (2000). Pain, cognition, and traumatic brain injury. *Neurorehabilitation*, 14, 95-103.
- Nicholson, K. & Martelli, M.F. (2004). The problem of pain. *Journal of Head Trauma Rehabilitation*, 19, 2-9.
- Nicholson, K., Martelli, M.F., & Zasler, N. D. (2001). Does pain confound interpretation of neuropsychological test results? *NeuroRehabilitation*, 16, 225-230.
- Panayiotou, A., Jackson, M., & Crowe, S.F. (2010). A meta-analytic review of the emotional symptoms associated with mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 32, 463-473.
- Paniak, C., Reynolds, S., Toller-Lobe, G., Melnyk, A., Nagy, J., & Schmidt, D. (2002). A longitudinal study of the relationship between financial compensation and symptoms after treated mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 24, 187-193.
- Pankratz, L. (1979). Symptom validity testing and symptom retraining: Procedures for the Assessment and Treatment of functional sensory deficits. *Journal of Consulting and Clinical Psychology*, 47, 409-410.
- Pankratz, L. (1983). A new technique for the assessment and modification of feigned memory deficit. *Perceptual and Motor Skills*, 57, 367-372.
- Pappagallo, M. & Werner, M. (2008). *Chronic Pain: A primer for physicians*. Chicago, IL:Remedica.
- Parente, R. & Herrmann, D. (1996). *Retraining cognition: Techniques and applications*. Gaithersburg, MD: Aspen Publishers, Inc.
- Parente, R., Kolakowsky-Hayner, S., Krug, K., & Wilk, C. (1999). Retraining working memory after traumatic brain injury. *NeuroRehabilitation*, 13, 157-163.
- Ponsford, J., Willmott, C., Rothwell, A., Cameron, P., Kelly, A., Nelms, R., Curran, C., & Ng, K. (2000). Factors influencing outcome following traumatic brain injury in - adults. *Journal of the International Neuropsychological Society*, 6, 568-579.
- Porter-Moffitt, S., Gatchel, R. J., Robinson, R. C., Deschner, M., Posamentier, M., Polatin, P., et al. (2006). Biopsychosocial profiles of different pain diagnostic groups. *Journal of Pain*, 7, 308-318.
- Rabin, L.A., Barr, W.B., & Burton, L.A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: A survey of INS, NAN, and APA Division 40 members. *Archives of Clinical Neuropsychology*, 20, 33-65.
- Radanov, B., Dvorak, J., & Valach, L. (1992). Cognitive deficits in patients after soft tissue injury of the cervical spine. *Spine*, 17, 127-131.
- Raskin, S.A., Mateer, C.A., & Tweeten, R. (1998). Neuropsychological assessment of individuals with mild traumatic brain injury. *The Clinical Neuropsychologist*, 12, 21-30.

- Reynolds, S., Paniak, C., Toller-Lobe, G., & Nagy, J. (2003). A longitudinal study of compensation-seeking and return to work in a treated mild traumatic brain injury sample. *Journal of Head Trauma Rehabilitation, 18*, 139–147.
- Riley, J. L., III, Robinson, M. E., Geisser, M. E., & Wittmer, V. T. (1993). Multivariate cluster analysis of the MMPI-2 in chronic low-back pain patients. *Clinical Journal of Pain, 9*, 248-252.
- Rohling, M.L., Allen, L.M., & Green, P. (2002). Who is exaggerating cognitive impairment and who is not? *CNS Spectrums, 7*, 387-395.
- Rose, J.M. (2005). Continuum of care model for managing mild traumatic brain injury in a worker's compensation context: A description of the model and its development. *Brain Injury, 19*, 39-53.
- Rudy, T.E., Lieber, S.J., Boston, J.R., Gourley, L.M. & Baysal, E. (2003). Psychosocial predictors of physical performance in disabled individuals with chronic pain. *Clinical Journal of Pain, 19*, 18-30.
- Ruff, R. (2005). Two decades of advances in understanding of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 20*, 5-18.
- Ruff, R.M., Iverson, G.L., Barth, J.T., Bush, S., Broschek, D.K. and the NAN Policy and Planning Committee (2009). Recommendations for diagnosing a mild traumatic brain injury: A National Academy of Neuropsychology education paper. *Archives of Clinical Neuropsychology, 24*, 3-10.
- Ryan, L.M. & Warden, D.L. (2003). Post concussion syndrome. *International Review of Psychiatry, 15*, 310-316.
- Sattler, J.M. (2001). *Assessment of children: Cognitive applications (4th ed.)*. San Diego, CA: Author.
- Satz, P.S., Alfano, M.S., Light, R., Morgenstern, H., Zaucha, K., Asarnow, R.F., & Newton, S. (1999). Persistent post-concussive syndrome: A proposed methodology and literature review to determine the effects, if any, of mild head and other bodily injury. *Journal of Clinical and Experimental Neuropsychology, 21*, 620-628.
- Sawchyn, J.M., Brulot, M.M., Strauss, E. (2000). Note on the use of Postconcussion Syndrome Checklist. *Archives of Clinical Neuropsychology, 15*, 1-8.
- Scherer, M. & Madison, C.F. (2005). Moderate and severe traumatic brain injury. In G.J. Larrabee (Ed.). *Forensic neuropsychology: A scientific approach* (pp. 237-270). New York: Oxford University Press.
- Schnurr, R.F. & MacDonald, M.R. (1995). Memory complaints in chronic pain. *The Clinical Journal of Pain, 11*, 103-111.
- Schretlen, D.J. & Shapiro, A.M. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry, 15*, 341-349.
- Seminowicz, D.A., & Davis, K.D. (2006). Cortical responses to pain in healthy individuals depends on pain catastrophizing. *Pain, 120*, 297-306.
- Serino, A., Ciaramelli, E., Santantonio, A.D., Malagu, S., Servadei, F., & Ladavas, E. (2006). Central executive system impairment in traumatic brain injury. *Brain Injury, 20*, 23-32.
- Slesinger, D., Archer, R.P., Duane, W. (2002). MMPI-2 characteristics in a chronic pain population. *Assessment, 9*, 406-414.

- Slick, D. J., Sherman, E. M. S., & Iverson, G. L. (1999). Diagnostic criteria for malingered neurocognitive dysfunction: Proposed standards for clinical practice and research. *The Clinical Neuropsychologist*, *13*, 545–561.
- Smith-Seemiller, L., Fow, N.R., Kant, R., & Franzen, M.D. (2003). Presence of postconcussion syndrome in patients with chronic pain vs mild traumatic brain injury. *Brain Injury*, *17*, 199-206.
- Stevens, A., Friedel, E., Mehren, G., & Merten, T. (2008). Malingering and uncooperativeness in psychiatric and psychological assessment: Prevalence and effects in a German sample of claimants. *Psychiatry Research*, *157*, 191-200.
- Stulemeijer, M., Vos, P.E., Bleijenberg, G., & van der Werf, S.P. (2007). Cognitive complaints after mild traumatic brain injury: Things are not always what they seem. *Journal of Psychosomatic Research*, *63*, 637-645.
- Suhr, J.A. & Barrash, J. (2007). Performance on standard attention, memory, and psychomotor speed tasks as indicators of malingering. In G.J. Larrabee (Ed.) *Assessment of malingered neuropsychological deficits*, (pp. 131-170). New York: Oxford University Press.
- Suhr, J.A. & Gunstad, J. (2002). Postconcussive symptom report: The relative influence of head injury and depression. *Journal of Clinical and Experimental Neuropsychology*, *24*, 981-993.
- Suhr, J.A. & Gundstad, J. (2007). Coaching and malingering: A review. In G.J. Larrabee (Ed.). *Assessment of malingered neuropsychological deficits*, (pp. 287-311). New York: Oxford University Press.
- Sullivan, M.J., Stanish, W., Sullivan, M.E., & Tripp, D. (2002). Differential predictors of pain and disability in patients with whiplash injuries. *Pain Research and Management*, *7*, 68-74.
- Sweet, J.E. & Sweet, J.J. (2009). *Neuropsychology of malingering casebook*. (J.E. Morgan & J.J. Sweet, Eds.). New York: Psychology Press.
- Tellegen, A. & Ben-Porath, Y.S. (2008). *MMPI-2-RF Minnesota Multiphasic Personality Inventory-2-Restructured Form Technical Manual*. Minneapolis: University of Minnesota Press.
- Tellegen, A., Ben-Porath, Y.S., McNulty, J.L., Aribisi, P.A., Graham, J.R., & Kaemmer, B. (2003). *MMPI-2 Restructured Clinical (RC) scales: Development, validation, and interpretation*. Minneapolis: University of Minnesota Press.
- Tiersky, L.A., Cicerone, K.D., Natelson, B.H., & DeLuca, J. (1998). Neuropsychological functioning in chronic fatigue syndrome and mild traumatic brain injury: A comparison. *The Clinical Neuropsychologist*, *12*, 503-512.
- Tombaugh, T. (1996). *Test of Memory Malingering manual*. New York, NY: MultiHealth Systems.
- Tombaugh, T. N. (1997). The Test of Memory Malingering (TOMM): Normative data from cognitively intact and cognitively impaired individuals. *Psychological Assessment*, *9*, 260–268.
- Trahan, D.E., Ross, C.E., & Trahan, S.L. (2001). Relationships among post-concussional-type symptoms, depression, and anxiety in neurologically normal young adults and victims of mild brain injury. *Archives of Clinical Neuropsychology*, *16*, 435-445.

- Tsanadis, J., Montoya, E., Hanks, R.A., Millis, S.R., Fichtenberg, N.L., & Axelrod, B.N. (2008). Brain injury severity, litigation status, and self-report of postconcussive symptoms. *The Clinical Neuropsychologist*, 22, 1080-1092.
- Tunks, E.R., Crook, J., & Weir, R. (2008). Epidemiology of chronic pain with psychological comorbidity: Prevalence, risk, course, and prognosis. *Canadian Journal of Psychiatry*, 53, 224-234.
- Turk, D.C. (2002). A diathesis-stress model of chronic pain and disability following traumatic injury. *Pain Research Management*, 7, 9-19.
- Turk, D.C. (2004). Understanding pain sufferers: The role of cognitive processes. *The Spine Journal*, 4, 1-7.
- Vanderploeg, R.D., Curtiss, G., & Belanger, H.G. (2005). Long-term neuropsychological outcomes following mild traumatic brain injury. *Journal of the International Neuropsychological Society*, 11, 228-236.
- Vecera, S.P. & Rizzo, M. (2004). Attention: Normal and disordered processes. In M. Rizzo & P.J. Eslinger (Eds.). *Principles and Practice of behavioral neurology and neuropsychology*. (pp. 223-245).
- Wade, J.B. & Hart, R.P. (2002). Attention and the stages of pain processing. *Pain Medicine*, 3, 30-38.
- Wade, J.B., Dougherty, L.M., Archer, C.R., & Price, D.D. (1996). Assessing the stages of pain processing: A multivariate analytical approach. *Pain*, 68, 157-167.
- Wang, Y., Chan, R.C.K., & Deng, Y. (2006). Examination of postconcussion-like symptoms in healthy university students: Relationships to subjective and objective neuropsychological function. *Archives of Clinical Neuropsychology*, 21, 339-347.
- Wechsler, D. A. (1997a). *Wechsler adult intelligence scale-III*. New York, NY: Psychological Corporation.
- Williams, D. H., Levin, H. S., & Eisenberg, H. M. (1990). Mild head injury classification. *Neurosurgery*, 27, 422-428.
- Williams, W.H., Potter, S., & Ryland, H. (2010). Mild traumatic brain injury and Postconcussion Syndrome: A neuropsychological perspective. *Journal of Neurology, Neurosurgery, and Psychiatry*, 81, 1116-1122.
- Willmott, C., Ponsford, J., Hocking, C., & Schonberger, M. (2009). Factors contributing to attentional impairments after traumatic brain injury. *Neuropsychology*, 23, 424-432.
- Wong, J.L., Regennitter, R.P., & Barrios, F. (1994). Base rate and simulated symptoms of mild head injury among normals. *Archives of Clinical Neuropsychology*, 9, 411-425.
- Wood, R.L.L. (2004). Understanding the 'miserable minority': A diathesis-stress paradigm for post-concussional disorder. *Brain Injury*, 18, 1135-1153.
- Wood, R.I. & Rutterford, N.A. (2006). The effect of litigation on long term cognitive and psychosocial outcome after severe brain injury. *Archives of Clinical Neuropsychology*, 21, 239-246.

Appendix A

Brain injury severity classification systems

				may not be transient	
	13-15	≤ 30 minutes	≤ 24 hours	* 1 (or more) of following: LOC, PTA, confusion or disorientation, or other transient neurological abnormalities such as focal signs, seizure, intracranial lesions not requiring surgery. * plus GCS * these manifestations cannot be due to: drugs, alcohol, medication caused by other injuries caused by other problems caused by a penetrating injury	WHO, 2004
Moderate	9-12	20 minutes to 36 hours	1 - 7 days		Stein, 1996
Severe	3-8	> 36 hours	> 7 days		Stein, 1996

Note. GCS = Glasgow Coma Scale; LOC = loss of consciousness; PTA = post-traumatic amnesia; ACRM = American Congress of Rehabilitation Medicine; WHO = World Health Organization

Appendix B

Diagnostic and Statistical Manual of Mental Disorders – 4th ed. Text-Revised criteria for Postconcussional Disorder.

- A. A history of head trauma that has caused significant cerebral concussion. Note: the manifestations of concussion include loss of consciousness, post-traumatic amnesia, and, less commonly, posttraumatic onset of seizures. The specific method of defining this criterion needs to be established by further research.
- B. Evidence from neuropsychological testing or quantified cognitive assessment of difficulty in attention (concentrating, shifting focus of attention, performing simultaneous cognitive tasks) or memory (learning or recalling information)
- C. Three (or more) of the following occur shortly after the trauma and last at least three months:
 - (1) becoming fatigued easily
 - (2) disordered sleep
 - (3) headache
 - (4) vertigo or dizziness
 - (5) irritability or aggression on little or no provocation
 - (6) anxiety, depression, or affective lability
 - (7) changes in personality (e.g., social or sexual inappropriateness)
 - (8) apathy or lack of spontaneity
- D. The symptoms in Criteria B and C have their onset following head trauma or else represent a substantial worsening of preexisting symptoms.
- E. The disturbance causes significant impairment in social or occupational functioning and represents a significant decline from a previous level of functioning. In school-age children, the impairment may be manifested by a significant worsening in school or academic performance dating from the trauma.
- F. The symptoms do not meet criteria for Dementia Due to Head Trauma and are not better accounted for by another mental disorder (e.g., Amnesic Disorder Due to Head Trauma, Personality Change Due to Head Trauma)

Note. American Psychiatric Association. (2000). *Diagnostic and Statistical Manual, Fourth Edition, Text Revision*. Washington, DC: American Psychiatric Association (pages 761-2).

Appendix C

Compilation of studies examining the frequencies of self-reported symptoms meeting criteria for Post-Concussional Disorder

Investigators	sample(s)	n	Criteria C Symptoms for Post-Concussional Disorder							Cognitive Symptoms		
			fatigue	d/o sleep	h/a	vertigo/ dizzy	irr./ agg.	anx.*	dep.*	attn/ conc.	memory	other
Chan (2001a)	healthy volunteers	85	53.5	50.6	40.0	31.8	43.6	-	31.8	58.9	-	58.9
Dunn et al., (1995)	P.I. psych	156	71.2	81.4	76.9	41.0	62.8	86.5	76.3	71.2	45.5	-
	head injured/toxic	68	55.9	39.7	57.4	27.9	30.9	55.9	41.2	33.8	23.5	-
	family med controls	113	36.6	29.5	50.4	21.2	26.5	40.7	26.5	21.2	12.4	-
Fox et al., (1995a)	non-lit Psychiatric	329	-	-	-	-	51.0	65.0	-	42.0	-	-
Fox et al., (1995b)	non-lit psychotherapy	397	55.0	-	52.0	30.0	55.0	-	-	45.0	31.0	-
	neurology	104	52.0	-	49.0	30.0	41.0	-	-	34.0	36.0	-
	normal controls	292	34.0	-	43.0	24.0	33.0	-	-	19.0	18.0	-
Garden & Sullivan (2010)	healthy volunteers	96	24.0	27.1	28.1	7.3	20.8	18.8	17.7	20.7	9.4	5.2
Garden, Sullivan, & Lange (2010)	healthy volunteers	93	24.0	27.0	28.0	7.0	22.0	19.0	18.0	21.0	22.0	5.0
Iverson (2006)	depression	64	57.8	53.1	28.1	10.9	35.9	35.9	56.3	46.9	42.2	-
Iverson et al., (2010)	mild TBI post-injury	90	90.0	81.1	95.6	77.8	72.2	68.9	66.7	82.2	75.6	61.1
	mild TBI retro. pre-injury	90	20.0	17.8	25.6	3.3	11.1	8.9	11.1	2.2	2.2	2.2
	healthy controls	177	39.0	32.8	26.6	17.5	29.9	22.6	25.4	27.1	23.7	16.4
Iverson & Lange (2003)	healthy volunteers	104	75.7	62.1	52.4	41.7	71.8	63.1	61.2	61.2	50.5	-
Kashluba, Casey, & Paniak (2006)	mild TBI (one month)	110	90.0	72.0	76.0	59.0	61.0	63.0	40.0	63.0	74.0	-
	mild TBI (three month)	110	59.0	50.0	58.0	27.0	56.0	51.0	39.0	42.0	48.0	-
	control (one month)	118	33.0	47.0	58.0	22.0	47.0	60.0	33.0	35.0	47.0	-
	control (three month)	118	36.0	40.0	59.0	16.0	47.0	58.0	37.0	37.0	50.0	-
Lange et al., (2010)	good effort mTBI	48	59.6	48.9	63.8	40.4	34.0	21.3	34.0	46.8	34.0	-
	poor effort mTBI	15	86.7	80.0	73.3	46.7	66.7	60.0	60.0	73.3	73.3	-

continued

Investigators	sample(s)	n	Criteria C Symptoms for Post-Concussional Disorder							Cognitive Symptoms		
			fatigue	d/o sleep	h/a	vertigo/ dizzy	irr./ agg.	anx.*	dep.*	attn/ conc.	memory	other
Lange, Iverson, & Rose (2011)	mild TBI no depression	37	37.8	37.8	45.9	24.3	29.7	13.5	8.1	32.4	32.4	21.6
	mild TBI depressed	23	69.6	60.9	69.6	43.5	52.2	60.9	56.5	65.2	47.8	34.8
	depressed outpatient	58	62.1	58.6	29.3	12.1	39.7	39.7	62.1	51.7	46.6	25.9
	healthy control	72	2.8	2.8	4.2	0.00	4.2	0.00	0.00	0.00	1.4	0.00
Lees-Haley et al., (2001)	P.I. Other Injury	66	-	-	77.0	44.0	62.0	85.0	82.0	56.0	36.0	65.0
	P.I. mild TBI	24	-	-	75.0	54.0	46.0	42.0	38.0	65.0	42.0	67.0
Mittenberg et al., (1992)	simulating controls	223	47.2	-	80.0	63.3	50.0	68.1	67.6	66.8	-	-
	mixed-severity TBI	100	63.9	-	59.1	52.0	65.9	58.3	63.2	70.5	-	-
Sawchyn et al., (2000)	college students	326	53.0	-	27.0	14.0	30.0	29.0	-	40.0	18.0	-
Smith-Seemiller et al., (2003)	chronic pain	63	90.0	97.0	71.0	40.0	86.0	-	84.0	78.0	67.0	-
	mild TBI	32	81.0	72.0	81.0	56.0	78.0		63.0	94.0	94.0	-
Trahan et al., (2001)	mild head injury	40	35.0	25.0	19.0	7.0	21.0	29.0	7.0	30.0	-	25.0
	depression	56	68.0	55.0	37.0	20.0	52.0	74.0	50.0	54.0	-	25.0
	normal	496	16.0	16.0	3.0	4.0	9.0	16.0	6.0	18.0	-	12.0
Tsanadis et al., (2008)	mod-severe TBI	133	46.0	11.0	45.0	22.0	39.0	42.0	39.0	53.0	55.0	24.0
	poor effort mTBI	25	84.0	76.0	80.0	50.0	80.0	76.0	71.0	88.0	92.0	44.0
Wang et al., (2006)	college students	124	76.9	50.4	35.5	32.2	42.1	-	37.2	58.7	-	45.5

Note. Criteria C and cognitive symptoms are based on the the diagnostic criteria for Post-Concussion Disorder specified in the DSM-IV TR. The values listed represent the percentage of individuals in the sample that endorsed the symptoms at a level of at least moderate severity on the self-reported post-concussion questionnaires utilized in the study. Apathy and change in personality were not included in the table due to an inadequate number of individuals endorsing these symptoms. * Anxiety and depression are listed together in the DSM-IV TR criteria but were reported separately for the purposes of this table. d/o = disorder; h/a = headache; irr./agg. = irritability/aggression; anx = anxiety; dep = depression; attn/conc. = attention/concentration; P.I. = personal injury; psych = psychiatric group; med = medical; non-lit = non-litigating; retro. = retrospective; TBI = traumatic brain injury.

Appendix D

Correlations between demographic variables and WMI and PSI

		Chronic Pain		Mild TBI		M/S TBI	
		WMI	PSI T-	WMI	PSI T-	WMI	PSI T-
		score	score	score	score	score	score
age	<i>r</i>	.047	.018	-.177	-.182	-.111	.031
education	<i>r</i>	.089	.018	.016	.144	.179	.148
gender	<i>r</i>	.090	.146	-.05	-.081	.219	-.28
race	<i>r</i>	.091	-.033	-.078	-.213	-.083	-.186

Note. All correlations were non-significant. Gender was coded as: 0 = male, 1 = female; race was coded as: White = 1, not White = 0.

Chronic Pain Subgroup Comparison on MMPI-2 Individual Scales and Combination Scales

Cross-tabs of individuals in pain subgroups obtaining scores at various cutoffs on individual MMPI-2 scales and combination scales.

Individual Scales		CP/no findings	CP/findings	χ^2	$p \leq$
Hypochondriasis	< 65	5.7	4.4	0.5	ns
	65 to 74	34.1	40.0		
	≥ 75	60.2	55.6		
Depression	< 65	29.5	24.4	0.5	ns
	65 to 74	25.0	28.9		
	≥ 75	45.5	46.7		
Hysteria	< 65	23.9	24.4	0.5	ns
	65 to 74	22.7	17.8		
	≥ 75	53.4	57.8		
Psychasthenia	< 65	52.3	51.1	1.5	ns
	65 to 74	25.0	33.3		
	≥ 75	22.7	15.6		

Combined Scales					
Somatization *	≥ 65	75.0	73.3	0.7	ns
	≥ 75	46.6	35.6	7.1	.03
Somat. + Dep.	≥ 65	62.5	62.2	0.7	ns
	≥ 75	31.8	22.2	3.9	ns
All Scales	≥ 65	40.9	37.8	1.5	ns
	≥ 75	14.8	2.2	8.5	ns

Note. TBI = CP = chronic pain; X^2 = chi-square; somat. = somatization; dep. = depression

* somatization = scales 1 & 3

^{ab} row percentages with the same letter are not significantly different from each other

T-Test Comparisons for Chronic Pain Subgroups

T-tests examining deviations from normal (T-score = 50 and "impairment" (T-score = 40) level for WMI and PSI T-scores by clinical group for pain subgroups

	<i>Mean</i>	<i>(sd)</i>	Test Value = 50		Test Value = 40	
			<i>t-value</i>	<i>p</i> ≤	<i>t-value</i>	<i>p</i> ≤
<u>WMI T-score</u>						
CP/no findings	45.3	(8.3)	-5.3	.001	6.0	.001
CP/findings	45.3	(8.5)	-3.7	.001	4.2	.001
<u>PSI T-score</u>						
CP/no findings	42.5	(7.8)	-9.0	.001	3.1	.003
CP/findings	43.2	(8.2)	-5.6	.001	2.6	.012

Note. Sd = standard deviation; CP = Chronic Pain

Examination of Working Attention Impairment in Chronic Pain Subgroups
Percentage of patients in pain subgroups that scored at or below 1, 1.5, or 2 standard deviations below the mean on Working Memory and Processing Speed scores

T-score	CP/no findings	CP/findings	χ^2	$p \leq$
WMI \leq 40	28.4	37.8	1.2	ns
WMI \leq 35	9.1	13.3	0.6	ns
WMI \leq 30	1.1	0.0	0.6	ns
PSI \leq 40	45.5	42.2	0.1	ns
PSI \leq 35	15.9	17.8	.08	ns
PSI \leq 30	4.5	4.4	0.00	ns
Both \leq 40	18.2	22.2	0.3	ns
Both \leq 35	2.3	2.2	0.7	ns
Both \leq 30	5.7	4.4	0.1	ns

Note. TBI = traumatic brain injury; M/S = moderate-severe; CP = Chronic Pain; χ^2 = chi-square; ns = non-significant; WMI = Working Memory Index; PSI = Processing Speed; \leq = less than or equal to

Vita

Kelly Curtis was born in Lake Forest, Illinois and graduated from the University of Illinois, Urbana-Champaign with a Bachelor of Science in Psychology in May of 2000. Kelly began working with Dr. Kevin Greve at the University of New Orleans in the fall of 2003. Kelly received her Master of Science in Applied Biopsychology in August of 2005. She was awarded the Andrew S. Wensel Distinguished Graduate Student Award in April of 2008. She hopes to continue the research related to her dissertation regarding the contributing factors to persisting symptoms in mild brain injury and chronic pain patients. Kelly is looking forward to fulfilling her dream of teaching when she starts her professorship in North Carolina in the fall of 2012.