A Neuropsychosocial Investigation of Persistent Post-Concussion Symptoms After Mild Traumatic Brain Injury: Contributions of Cognitive Impairment, Anxiety Susceptibility, and Identity

by

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B.A., McMaster University, 2004
M.Sc., University of Victoria, 2010

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Abstract

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Objectives: The majority of individuals who sustain a mild traumatic brain injury (mTBI) will experience a full recovery within the first weeks or months post-injury. However, some individuals will experience ongoing difficulties, or persistent post-concussion symptoms (PCS), for years following the injury. To date, most researchers have attributed PCS to either neuropathological factors or to psychogenic factors. Lacking exploration has been the role of psychosocial variables and the consideration of PCS from a more holistic, or 'whole person', perspective. As such, the goal of the current study was to undertake an investigation of persistent PCS using a broad, neuropsychosocial framework. Specifically, this was done by investigating how (a) cognitive functioning, (b) susceptibility to anxiety while in the context of a stressful situation (i.e., anxiety susceptibility), and (c) multiple components of identity (including self-perception, TBI-related self-concept, and TBI-related social identity) influence the severity of persistent PCS. The main underlying assertion to this research is that there are multiple factors that underlie the experience of persistent PCS; a purely neuropathological or psychogenic perspective is not sufficient to understand the complex processes inherent in recovery after mTBI.

Method: The sample consisted of 21 adults, between 20 and 65 years of age, who had sustained an mTBI at least one year earlier. Following a telephone interview to determine eligibility (and a separate telephone interview with a source of collateral information) the participants completed a number of standardized neuropsychological measures and self-report questionnaires during an in-person, one-on-one data collection session.

Results: The only injury-related or demographic variable that had an influence on PCS was injury etiology, whereby individuals with sports related injuries reported significantly less PCS than did those who sustained non-sports related injuries (e.g., motor vehicle accidents). Cognitive functioning had no influence on PCS severity, nor did anxiety susceptibility. However, one's general propensity to experience anxiety (i.e., trait anxiety) was a significant predictor of PCS. Further, multiple aspects of identity influenced PCS with both current self-perception and TBI-related social identity being significant predictors of self-reported PCS severity.

Conclusions: Despite the failure to find any impact of neuropsychological factors on PCS in the current study, other lines of research have demonstrated neuropathological changes associated with mTBI – some of which may be chronic. Therefore, cognitive functioning may not be a sufficiently sensitive indicator of possible neuropathology at more than one year post-injury. On the other hand, the current study demonstrates that psychological and psychosocial factors are highly relevant to recovery and outcome following mTBI, and are significant predictors of PCS severity. Overall, the results support the assertion that recovery after mTBI is complex and that there are multiple factors that underlie persistent PCS. Further, the study demonstrates the importance of conceptualizing the process of recovery from a broad, neuropsychosocial perspective. Implications for treatment interventions and future research are discussed.

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Acknowledgments

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Of course, this dissertation would not have been possible without all of the individuals who gave their time and energy to participate in this study. I hope that this project can, in some small way, benefit them and others on their journey of recovery.

Finally, a special thank you to the friends and family who have encouraged and supported me throughout the journey that is graduate school – especially Carmen, Aunt Ann and Aunt Bette. It means the world!

Dedication

This dissertation is dedicated to my family.

To my parents, who taught me to work hard and to persevere through adversity, who always believed in me and my abilities, and who have always provided me with unconditional love and unwavering support. Thank you for everything.

And to Joshua – my partner through it all – whose love, support, and encouragement know no bounds. You have sacrificed so much to help make this all possible, and I'm so lucky to be on this journey with you. Now it's your turn! <4

Introduction

Traumatic brain injury (TBI) is a major health concern in all industrialized nations causing a significant degree of disability, subsequent social service usage, and even death in severe cases (Tator, 2010). The majority of TBIs are of mild severity (referred to as mild TBI, or mTBI), with mTBIs making up approximately 70 – 90% of all *treated* brain injuries (Cassidy et al., 2004). Incidence rates of TBI (and mTBI, specifically) within Canada are incomplete, and information for British Columbia (the location of the current study) is not currently available. The Ontario Neurotrauma Foundation (ONF, 2013) cites an incidence rate for mTBI of between 493 and 653 per 100,000 in Ontario when both hospital-treated cases as well as those presenting to a family physician were included. In 2013 alone there were 148,710 mTBIs diagnosed in Ontario (ONF, 2017) – obviously, this number does not include those individuals who sustained a mTBI but who were not seen or treated and thus did not received a formal diagnosis. This is compared to an estimated incidence rate for dementia (including Alzheimer's Disease) of 500 per 100,000 in Ontario in 2011 (Ng, et al., 2015), and an incidence rate for heart failure of 306 per 100,000 in Ontario in 2007 (Yeung, et al., 2012). Incidence and prevalence figures for mTBI can be quite shocking, which has in part lead to TBI being referred to as a “silent epidemic”; these injuries are surprisingly common, yet, until very recently, the general public had very little knowledge or understanding of brain injury and the range of impacts that they can cause (Langlois, Marr, Mitchko, & Johnson, 2005).

The large majority of individuals who suffer a single mTBI will experience a full recovery, typically within the first few days to months following the injury (ONF, 2011), with no noticeable long-term difficulties experienced. However, this is not the case for all individuals: approximately 15% of those who sustain an mTBI will report ongoing symptoms long after most experts in the fields of medicine, neurology, and neuropsychology agree that functional recovery should have occurred (Iverson & Lange, 2011a). These symptoms are referred to as post-concussion symptoms and the symptom constellation is often referred to as Post-Concussion Syndrome or Post-Concussion Disorder. If the symptoms remain beyond one year post-injury, they are then referred to as *persistent* post-concussion symptoms (Alexander, 1995).

In recent years, a good deal of research has attempted to establish why some individuals experience persistent symptoms – e.g., what factors predict ongoing symptoms, commonalities among individuals with incomplete recovery and how they are different from those who experience good recovery. A predominant theme in the literature is that the symptoms associated with post-concussion syndrome are not unique to mTBI and that, instead, the symptoms are due to the exacerbation of pre-existing factors (e.g., personality characteristics, depression), poor adjustment following the injury, and/or other comorbidities such as pain, post-traumatic stress disorder, etc. A second (and sometimes related) theme in the literature is that those who report persistent symptoms are feigning their symptoms for some type of secondary gain - particularly when persistent symptoms are reported in the context of injury-related litigation. However, it may be too simplistic to conclude that persistent post-concussion symptoms can be discounted as wholly unrelated to the original mTBI in all individuals. Further, there may be other – heretofore unconsidered – factors that can influence the development of persistent symptoms and it is likely that, for many individuals who sustain a mTBI, there are multiple factors that may play a role in poor recovery. As such, the goal of the current study was to investigate persistent post-concussion symptoms from a broad, 'whole person' perspective, considering the role of neuropsychological, psychological, and psychosocial factors in the experience of ongoing symptoms after mTBI. In other words, a neuropsychosocial approach to understanding persistent post-concussion symptoms was taken.

The current study aims to build on previous literature as well as relevant clinical experience in order to further elucidate factors that may influence the severity of persistent post-concussion symptoms. Specifically, objective cognitive functioning, susceptibility to anxiety while in a cognitively demanding situation, and multiple aspects of identity were investigated as to their influence on the severity of self-reported persistent post-concussion symptoms after mTBI. Moreover, the study aimed to include individuals from across the spectrum of recovery in order to speak to factors that differentiate those who experience a quick and full recovery from those who experience an incomplete recovery with ongoing difficulties.

Background on TBI

Definition and characterization of TBI.

Traumatic brain injury can be defined as damage to the brain that results from a blunt impact to, or affecting, the head (Ng et al., 2015). There are three typical mechanisms of damage: (1) rapid acceleration of the head due to a physical blow from a blunt object (e.g., a fist); (2) rapid deceleration of the head due to contact with a blunt and relatively stable object (e.g., a steering wheel or the ground); and (3) rapid movement of the brain within the skull (acceleration/deceleration and/or rotation) due to impact to the body (e.g., being hit by another player in a contact sport, whiplash-type injuries in motor vehicle accidents) (Richardson, 2000). The most common causes of TBI in Canada are falls, being struck by something (including sports-related injuries and assault), and motor-vehicle accidents (Colantonio et al., 2010).

The severity of a brain injury is determined by the immediate characteristics and sequelae of the injury (Alexander, 1995). The most common metrics used to determine injury severity include: length of loss of consciousness (LoC), length of post-traumatic amnesia (PTA), observer rating on the Glasgow Coma Scale (GCS, Teasdale & Jennett, 1974), neurological focal signs, as well as the results of neuroimaging (e.g., CT or MRI scan) (ONF, 2013). Based on these injury characteristics, TBI is categorized as mild, moderate, or severe.

Length of LoC is a relatively straightforward indicator of injury severity based on the number of minutes, hours, or days that the individual who sustained the injury was unconscious. Unfortunately, LoC can be difficult to measure in individuals who sustain less severe injuries because, if it occurs, it is (by definition) of shorter duration; as such, consciousness has generally been recovered by the time the patient is admitted to the hospital (if emergency services are even sought out) (Richardson, 2000). Further, individuals who sustain less severe injuries may not fully lose consciousness but instead experience brief periods of an altered state of consciousness (i.e., being dazed, confused, and/or disoriented immediately after the injury), which too is indicative of some degree of insult to the brain (Mateer & D'Arcy, 2000; ONF, 2013). For a diagnosis of a mild TBI, any LoC must last less than 30 minutes (ONF, 2013).

The duration of PTA is also used as an indicator of severity. PTA is a state of altered consciousness often involving confusion, agitation, and, most recognizably, loss of memory (i.e., amnesia). The state of confusion common during PTA can include deficits in orientation, episodes of rambling or meaningless speech, and perseverative speech, thoughts, and actions. Agitation during PTA can include restlessness, emotional lability, disturbed diurnal rhythm, impaired insight, impulsiveness, and verbal and/or physical aggression. The defining characteristic of PTA is a global, episodic, anterograde amnesia that affects the encoding, storage and retrieval of new information. In other words, the person is unable to make memories from moment to moment (see Marshman et al., 2013 for a review of PTA). The duration of PTA is often defined as the period of time between the injury and the appearance of intact memory for new information, not including any time that the person was unconscious or unresponsive[1] and it is generally measured in hours or days (ONF, 2013). Because the duration of PTA must be measured, repeated assessment must take place over time in order to determine true, clinical emergence from PTA (Mateer & D'Arcy, 2000). For mTBI, the PTA cut-off is 24 hours or less (ONF, 2013).

The score obtained on the GCS (Teasdale & Jennett, 1974) is also used as an indicator of injury severity. It is one of the most commonly used rubrics for characterizing injury severity in hospital settings (and in research, when available) (ONF, 2013). Teasdale and Jennett (1974) developed the GCS to provide a consistent, structured system to assess and communicate the depth of impaired consciousness/coma in hospital. The GCS allows a physician/nurse to evaluate three individual aspects of behaviour (motor responsiveness, verbal performance, and eye opening) so as to gauge deterioration or improvement in consciousness during the acute post-injury stage. The patient receives a total score out of 15, with higher scores indicating better functioning. There are, however, some limitations to the GCS and its ability to predict injury severity. For example, its ability to accurately predict overall injury severity has been called into question by findings where a significant proportion of those defined as having a mild TBI based on their GCS score were later found to have much more significant injuries (e.g., evidence of intracerebral lesions on CT scan) (Stein et al.,1993). GCS scores can range from 3 to 15, and mTBI is defined as a score between 13 and 15 (ONF, 2013).

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[1] This is a strict anterograde amnesia definition of PTA (i.e., only includes the period of inability to make new memories subsequent the injury). Some definitions of PTA also include any period of retrograde amnesia, or any loss of memory for events immediately prior to the injury, as well.

While cited less frequently, severity can also be assessed based on the presence and type of neurological focal signs following injury. Neurological focal signs are indicators of damage to the brain, spinal cord or nerves (Alexander, 1995). For example, paralysis, paresis (i.e., significant muscle weakness), and aphasia are all neurological focal signs indicative of damage to specific brain regions and, as such, more severe injury. More general neurological focal signs that may be seen following a milder TBI include headache, pallor, extreme fatigue, excessive sweating (i.e., diaphoresis), transient ataxia (i.e., uncoordinated voluntary movements), vomiting, blurred or double vision, balance problems or dizziness, sensitivity to light or noise, and tinnitus immediately following the injury (Alexander, 1995; ONF, 2013).

Finally, results of neuroimaging have become another way of attempting to determine the severity of brain injury. Individuals brought to hospital reporting a head injury and the presence of other neurological symptoms (e.g., confusion, dizziness, loss of consciousness, etc.) will often receive a CT scan (or MRI scan if required/available). These scans are used to identify the presence of lesions that may need immediate intervention (e.g., intracranial hemorrhages, swelling, etc.) (Niogi & Mukherjee, 2010). However, in the case of mTBI, CT and conventional MRI usually fail to detect evidence of structural brain abnormalities (ONF, 2013).

These variables of LoC, PTA, and GCS are assessed as soon as possible after injury, and the results are used to determine the initial injury severity. Each level of injury severity – mild, moderate, and severe – is defined and diagnosed based on specific values or findings for these immediate post-injury characteristics.

Mild TBI (mTBI): definitions and diagnosis. The criteria for diagnosing mTBI have evolved over time and within the last 25 years or so, a significant effort has been made to come up with a consensus on the definition of mTBI as a distinctive clinical entity. While there is still no universally agreed upon definition, those that are the most commonly used are quite similar. The four major definitions of mTBI have been developed by (1) the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (ACRM MTBI Committee), (2) the Center for Disease Control (CDC) working group, (3) the World Health Organization Collaborating Centre Task Force on Mild Traumatic Brain Injury (WHO Collaborating Centre Task Force), and (4) the Ontario Neurotrauma Foundation (ONF). All four definitions agree that a mild TBI should be diagnosed following a traumatic injury to the head (i.e., the head hits something, something hits the head, or sufficient external physical force to potentially cause internal acceleration/deceleration and/or rotation of the brain) with subsequent evidence of immediate symptoms such as a period of confusion, disorientation, or loss of consciousness; loss/dysfunction of memory for the events surrounding the accident; neurological focal signs such as acute seizures, headache, dizziness, irritability, fatigue, and/or poor concentration; and most indicating that the GCS must be 13 to 15 at 30 minutes following the injury (see Ruff et al., 2009; Iverson & Lange, 2011a; Raskin & Mateer, 1999; ONF, 2013). In general, if an individual meets all of the criteria for a mild TBI as described but also has evidence of damage to the brain on neuroimaging and/or a skull fracture, they will be diagnosed with a *complicated* mTBI (Iverson & Lange, 2011a). The distinction between mild and complicated mild TBI has been made because of evidence demonstrating more significant acute dysfunction and poorer prognosis for those with complicated mTBI (see Iverson & Lange, 2011a).

Despite the efforts of the ACRM, CDC, WHO, and ONF to develop clear and distinct criteria for defining and diagnosing mTBI, there are some ongoing problems with mTBI as a clinical entity. First, there exists a lack of agreement between these groups on the specifics of diagnostic criteria (e.g., see Ruff et al., 2009). For example, some of the criteria explicitly state that not *all* of the above sequelae are required for a diagnosis of mTBI while others do not make such clear distinctions; some are more stringent about the criteria than others (e.g., stating that neurological focal signs *must* be transient following a mild injury). Also, it is notable that only the ONF definition indicates that the immediate symptoms may evolve over time and that, in some cases, the symptoms might be prolonged (ONF, 2013). Therefore, an individual could receive a different diagnosis depending on the criteria used. Second, regardless of the specific criteria used, a very wide range of injury severities are inherently included under the umbrella of "mild" TBI. For example, an individual who experiences a few seconds of confusion and a headache, and another individual who is unconscious for almost 30 minutes and experiences significant post-injury confusion and amnesia for up to a day could both be labeled as having sustained a mild TBI. However, based on this information alone, the prognosis for these two individuals would likely be quite different.

A final problem with the definition of mTBI relates to the variety of terms used to refer to mTBI in both the medical community and in the literature. For example, some frequently used synonyms for mTBI are: concussion, mild head injury, minor brain injury, and minor head injury (von Holst & Cassidy, 2004). Concussion is commonly used in the media, often in association with sports-related injuries, but a specific definition and how, or if, it differs from mTBI is lacking. However, it seems that, within the TBI literature at least, "concussion" is regarded as a very mild TBI. von Holst and Cassidy (2004) refer to concussion as an “unspecific term” used by the lay population to describe a disturbance in neurological function caused by the mechanical force of rapid acceleration/deceleration, usually used to refer to a more mild injury, where the individual may feel dazed or see stars but does not lose consciousness. Similarly, Iverson and Lange (2011a) assert that concussion is the preferred term in sports-related accidents and civilian (as opposed to military or forensic) cases, particularly for injuries that fall on the milder end of the spectrum. However, they also state that that mTBI and concussion can be used interchangeably in most circumstances (Iverson & Lange, 2011a). More recently, Sharp (2015) has reviewed both the historical and contemporary usage of the term 'concussion' and indicates that concussion is currently used in two main ways: (1) to describe a distinct pathophysiological entity, mainly seen in the context of sports-related injuries, and often thought to reflect functional (as opposed to structural) disturbance to the brain, and (2) to describe the constellation of symptoms that arise after different types of TBI. This author argues that both of these contemporary usages are problematic and purports that the term concussion “lacks any diagnostic precision and at worst encourages a lazy diagnostic approach” (Sharp, 2015 p. 174). The use of different terms without a clear definition, to conversely refer to the same and different things, serves to further confuse the topic for all involved including researchers and patients. Therefore, throughout this document, the term mild traumatic brain injury (mTBI) will be used.

Outcomes Following mTBI

An overview of typical and atypical recovery. Despite some difficulties with the definition of mTBI, much is known about the progression and recovery. In the first days following the injury, a range of symptoms are possible. There is often an initial focus on physical symptoms, with awareness of cognitive and emotional symptoms developing somewhat later when the patient has had the opportunity to experience these symptoms in different situations or when they have been pointed out by others. The underlying cause of these very early symptoms is initially unclear: they are potentially caused by brain injury, but could also just as feasibly be the result of injury to several different body systems. For example, headache may be due to injury to the scalp or neck, a neural injury, or – even more likely – a mixture of causes; dizziness may be due to vestibular or cervical injury; and anxiety, mood problems, and irritability may be due to neural injury, pain, pre-existing or new psychological factors, or a combination of any of these. That being said, initial cognitive complaints and sleep-wake disturbances are likely the result of neural injury (Alexander, 1995).

By one month post-injury, the number of complaints has often decreased yet the frequency of complaints will often remain much greater than for controls. Also, the relation of many individual symptoms to their causes may have become clearer (e.g., headaches have unequivocal migranious properties, dizziness may be predictably positional, even irritability may be more accurately ascribed to related mood disorder, executive dysfunction, or psychosocial factors, etc.) (Alexander, 1995). The Ontario Neurotrauma Foundation (ONF, 2011) recommends that specialized assessment(s) take place with individuals continuing to experience symptoms at 1 month post-injury for the purposes of differential diagnosis and in order to ascertain the nature of symptoms and identify those that are potentially treatable.

By three months post-injury, substantial neurological recovery will have occurred (Alexander, 1995). Most studies indicate that the majority of survivors' physical and cognitive deficits have largely resolved by this point (Carroll et al., 2004; Iverson & Lange, 2011a). However, this generalization does not fit all mTBI survivors as some individuals may continue to experience on-going symptoms (Belanger et al., 2005). Within most recent literature, it is not until this point (i.e., three months post injury) that ongoing problems are regarded as comprising post-concussion symptoms (e.g., Bender & Matusewicz, 2013; ONF, 2011; Ruff, 2005).

Over the following 6 to 9 months, recovery will continue to occur. However many survivors – even those with good recovery – may remain susceptible to periodic difficulties, particularly under circumstances of physiological, cognitive, or psychological stress (e.g., alcohol use, sleep deprivation, increased workplace demands). By one year post-injury, the large majority of mTBI survivors will have made a good, or even seemingly complete, recovery. Yet between 10-15% of patients will continue to experience persistent symptoms (Ruff & Jamora, 2009). Those survivors who fail to make a good recovery are referred to by some as the "miserable minority" (first coined by Ruff, Camenzuli, and Mueller, 1996). At this point, approximately one year post injury, ongoing difficulties are regarded as *persistent* post-concussive symptoms (Alexander, 1995).

Importantly, the term "persistent" may be somewhat controversial. Symptoms may be experienced (and reported) as waxing and waning over time, as opposed to persisting in a consistent manner from the time of the initial injury (e.g., Meares et al., 2011). As such, it should not be assumed that *persistent* PCS represents a fixed profile of difficulties experienced over time but, instead, the presence of ongoing difficulties experienced subsequent to an mTBI.

Persistent symptoms and sequelae of mTBI. A variety of persistent difficulties are known to be possible following mTBI, including physical, cognitive, and emotional concerns. Physical symptoms are often the most prominent complaints very early post-injury (Mateer & D'Arcy, 2000) as they are often easily recognized by the survivor and easily described to others (e.g., healthcare professionals). For example, headache is very common following mTBI: in fact, ONF (2011) notes that several researchers have shown post-traumatic headache to be *more* common after mTBI than after severe TBI. Balance disorders of either peripheral (i.e., inner ear) or central (i.e., brain) origin are also common. Symptoms can range from vertigo to problems with dizziness and balance, as well as associated nausea, and can affect the individual's overall mobility and capacity to engage in daily activities (ONF, 2011). A range of vision disorders are also possible after sustaining an mTBI. Survivors may experience vision disturbances including diplopia (double vision), inability to fixate, scanning deficits, poor visual acuity, and increased sensitivity to light (photophobia) (ONF, 2011). Other sensory deficits, including increased sensitivity to sound (hyperacusis) and ringing in the ears (tinnitus), can also occur (Mateer & D'Arcy, 2000). Reports of persistent sleep disturbances, specifically insomnia often characterized by problems initiating and/or maintaining sleep, and subsequent daytime sleepiness are also extremely frequent (ONF, 2011). Finally, fatigue is one of the most pervasive symptoms following TBI. Fatigue is the experience of weariness or tiredness following exertion (physical or mental) and it often results in reduced stamina leading to decreased capacity for work and limited efficiency to (accurately and/or appropriately) respond to stimuli (ONF, 2011). Importantly, following mTBI, fatigue can actually be out of proportion to the amount of exertion whereby extreme fatigue occurs following limited exertion (in duration or intensity); it can even occur in the absence of exertion (ONF, 2011). Therefore, an array of significantly disabling persistent physical symptoms can occur following mild brain injury.

A range of persistent cognitive symptoms are also possible. Following mTBI, the most common deficits are in the domains of attention/concentration (including complex attention, or working memory), information processing speed, new learning/memory, and aspects of executive functioning – particularly cognitive (verbal) fluency (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Karr, Areshenkoff, & Garcia-Barrera, 2014; Mateer & D'Arcy, 2000; Mathias, Beall, & Bigler, 2004; ONF, 2011). However, the cognitive deficits following mTBI can be objectively subtle and difficult to assess with standardized measures, which makes the use of appropriately sensitive neuropsychological measures extremely important (Mateer & D'Arcy, 2000).

Persistent emotional, behavioural, and affective difficulties subsequent to mTBI have also been well documented. Early affective symptoms are quite variable and commonly include irritability, anxiety, emotional lability, depressed mood, and apathy (ONF, 2011). A significant proportion of survivors go on to develop persistent mental health concerns, and both new-onset disorders directly associated with the brain injury and exacerbation of pre-injury mental health conditions or vulnerabilities are possible (ONF, 2011). For example, affective disorders (i.e., anxiety, depression, or adjustment disorder) have been found to be highly prevalent with rates around 43% found in the first year post-injury (Delmonico, Theodore, Sandel, Armstrong, & Camica, 2017) and depression and anxiety are commonly co-morbid after mTBI (Moore et al., 2006; Walker, Franke, McDonald, Sima, & Keyser-Marcus, 2015). The most common symptoms of anxiety following mTBI include free-floating anxiety, fearfulness, intense worry, generalized uneasiness, social withdrawal, inter-personal sensitivity, and anxiety-related dreams (Moore, Terryberry-Spohr, & Hope, 2006). Other emotional and behavioural symptoms such as anger, frustration, and irritability are known to exist beyond the acute post-injury phase; however, little research has been done into these areas and prevalence rates vary greatly between studies that have been conducted (e.g., rates of irritability ranging from 5.3% to 21% at one year post-mTBI) (Hovland & Mateer, 2000; Leuthcke, Bryan, Morrow, & Isler, 2011). Taken together, it is evident that a significant number of individuals with mTBI will experience persistent psychological difficulties after their injury.

Persistent Post-Concussion Syndrome

Definition and diagnosis. As mentioned above, the majority of individuals who sustain a mild brain injury will make a good recovery (i.e., experience no ongoing symptoms), and this is the expected prognosis for individuals who sustain an uncomplicated mTBI. However, for a minority of individuals, generally reported to be between 10% and 20% (Iverson & Lange, 2011a), the symptoms associated with the brain injury persist. When they are still present at one year post injury, these symptoms are described as persistent post-concussion symptoms and an individual is regarded as having persistent post-concussion syndrome if he/she meets the diagnostic criteria (i.e., has a sufficient number of the symptoms).

There are two generally accepted sets of diagnostic criteria for post-concussion syndrome: one is from the World Health Organization in the ICD-10 (2010) and the other is from the American Psychiatric Association (APA) in the DSM-IV-TR (2000). The ICD-10 has termed the condition *Postconcussional Syndrome,* and defines it as, “A syndrome that occurs following head trauma (usually sufficiently severe to results in loss of consciousness) and includes a number of disparate symptoms such as headache, dizziness, fatigue, irritability, difficulty in concentration and performing mental tasks, impairment of memory, insomnia, and reduced tolerance to stress, emotional excitement, or alcohol". The DMS-IV-TR refers to the condition as *Postconcussional Disorder* and requires a history of head trauma plus 3 or more symptoms such as: being easily fatigued; disordered sleep; headache; vertigo or dizziness; irritability or aggression; anxiety, depression, or emotional lability; changes in personality; and apathy. Their criteria also require objective evidence of cognitive deficits (e.g., through neuropsychological testing). The onset of these symptoms must be after the head injury, cause significant impairment in social or occupational functioning, and not be better accounted for by another condition. Postconcussional disorder was defined as a disorder for further investigation in the DSM-IV, meaning that the APA did not believe that sufficient evidence existed to warrant inclusion as an official diagnostic entity. Importantly, it has not been included in the newest edition, the DSM-5 (APA, 2013).

Contributing factors. A number of factors have been found to predict persistent post-concussion syndrome and its symptoms. One of the most consistently significant predictors of post-concussion syndrome is involvement in litigation (e.g., Chan, 2005; Lange, Iverson, & Rose, 2010; Paniak et al., 2002). There are a number of potential reasons for this relationship including: (1) the nature of the process of litigation, whereby survivors are repeatedly required to "prove" their symptoms in order to receive any type of compensation for their suffering; (2) the adversarial relationship that exists between the injured individual and the party that is responsible for payment, such that the lawyer and experts for the responsible party constantly try to discredit the injured individual; and (3) an increased risk of malingering – or feigning symptoms – in order to receive some type of material gain (Bender & Matusewicz, 2013). It is worth noting, however, that not all studies find an effect of litigation or receiving compensation on the report of post-concussion symptoms (e.g., Hou et al., 2012).

Demographic and injury-related factors have also been found to predict post-concussion syndrome. Variables such as older age (Carroll et al., 2004; King & Kirkwilliam, 2011) as well as prior head injury, being a student, sustaining the injury in a motor vehicle accident, and post-injury symptoms of nausea (Carroll et al., 2004) have been implicated. Some studies have also found that gender predicts post-concussion syndrome, although the role of gender is inconsistent: while many studies find female gender more predictive (e.g., as reviewed by Carroll et al., 2004, and Dick, 2009) others have found male gender more predictive (e.g., Chan, 2005). Interestingly, variables related to injury severity are almost never significant predictors (Carroll et al., 2004; Wäljas et al., 2015), although in a study including individuals with both mild and moderate TBI, length of PTA was found to play a significant role in the development of post-concussion syndrome (King, 1996).

A range of personality factors have also been found to impact rates and severity of reported post-concussion symptoms. For example, negative head injury perception (e.g., viewing the injury as being associated with a number of symptoms that are out of the patient's control and have a serious impact on the patient's life) and all-or-nothing behaviour (e.g., "I find myself rushing to get everything done before I crash" and "I have avoided my usual activities") have both been shown to predict post-concussion symptoms at 3 and 6 months post (Hou et al., 2012). Anxiety sensitivity, or the sensitivity to one's own bodily sensations (believed to be a personality characteristic), has also been shown to predict the severity of post-concussion symptoms, as has difficulty in identifying one's own feelings (i.e., alexithymia) (Wood, O'Hagan, Williams, McCabe, & Chadwick, 2014).

Type and severity of cognitive deficits following mTBI can also predict post-concussion syndrome. Deficits in information processing speed (King & Kirkwilliam, 2011; Sterr, Herron, Hayward, & Montaldi, 2006), working memory (Sterr et al., 2006), verbal fluency (King & Kirkwilliam, 2011), divided attention (Sterr et al., 2006), and learning and immediate recall (King & Kirkwilliam, 2011) have all been found to be related to the report of other post-concussion symptoms. Also, more general findings including the number of errors on computerized cognitive tasks have been found to predict post-concussion syndrome (Sterr et al., 2006). In addition to objective findings of cognitive difficulties, individuals who meet criteria for post-concussion syndrome have been found to report more cognitive failures in their everyday life (Sterr et al., 2006).

Psychological and emotional factors have also been shown to have a very strong impact on the development and reporting of post-concussion syndrome. Depression (Hou et al., 2012; King & Kirkwilliam, 2011; McCauley et al., 2001; Wood et al., 2014; Lange, Iverson, & Rose, 2011) and anxiety (Hou et al., 2012; King & Kirkwilliam, 2011) are the most common, and often the most significant, predictors of the severity of post-concussion symptoms. For example, one study found that anxiety symptoms accounted for 45.9% of the variance within reported post-concussion symptoms (King & Kirkwilliam, 2011), while another reported that depression accounted for 52.6% of the variance in post-concussion symptoms in their sample (Wood et al., 2014). Post-traumatic stress symptoms have also been found to have a significant impact on post-concussion syndrome (King & Kirkwilliam, 2011; McCauley et al., 2001).

Despite a growing understanding of the importance of psychosocial factors in health, mortality, morbidity, and function in general (e.g., Berkman, Kawachi, & Glymour, 2014), there has been limited investigation into the impact of psychosocial factors on reports of post-concussion syndrome, and the results thus far are inconsistent. For example, less perceived social support, less satisfaction with support, and lower levels of social integration have all been found to predict post-concussion syndrome (McCauley et al., 2001). However, a more recent study found that social support at baseline did not predict meeting criteria for post-concussion syndrome at either 3 or 6 months post-injury (Hou et al., 2012). Other psychosocial variables have yet to be investigated.

As this brief review illustrates, there has been a notable amount of research conducted to investigate factors that may predict post-concussion syndrome. When taken together, this sample of the existing literature demonstrates that a broad range of factors have been found to influence post-concussion symptoms experienced at 3 or more months post-injury. However, this review also illustrates that there is limited consistency across findings; even for variables that are almost consistently found to predict post-concussion syndrome – such as anxiety – there is evidence refuting this relation (e.g., Wood et al., 2014). The inconsistencies in this literature likely have many sources. For example, there are significant design differences between the studies: some studies investigate the ability of baseline measures to predict later post-concussion syndrome, other studies investigate the role of variables in predicting the severity of post-concussion symptoms, while still others look only at the difference between those who meet syndrome criteria and those who do not. There are also differences related to the diagnostic criteria for post-concussion syndrome that are used across studies. Further, some studies include only mild TBI, while others include mild and moderate, etc. While these varying methods and results can make the literature somewhat unwieldy, the complexity of recovery after mTBI and post-concussion syndrome is evident.

Causes of Post-Concussion Syndrome: The Debate in the Literature

Despite the evidence of a multitude of factors playing a role in the development of post-concussion syndrome, two strong and divergent perspectives of the underlying cause of persistent symptoms have developed. One perspective is that post-concussion syndrome is due to injury-related neuropathology, whereas the other perspective views it as largely psychogenic in origin.

Neuropathological perspective. The perspective that post-concussion syndrome is the result of injury-related neuropathology is based on information regarding the biomechanics of TBI and the subsequent metabolic and pathophysiological changes, and evidence for how these injury mechanisms affect the function and structure of the brain (Martin, 2016; McFarlane & Glenn, 2015). Diffuse axonal injury (DAI), one of the main neuropathologies involved in most TBI, is due to the stretching (and, in some instances, complete shearing) of axons that comprise the brain's deep white matter tracts (Su & Bell, 2016). The initial impact causes shock-waves that pass slowly through the skull, semi-fluid brain, and cerebrospinal fluid-filled ventricles, followed by acceleration/deceleration and/or rotation of the brain within the skull (Martin, 2016). These forces cause the axon to stretch, which disrupts its functioning (through the abrupt disruption cellular homeostasis) and subsequently causes the axon to swell which can lead to the axon's eventual degeneration and detachment, with delayed cell death (or apoptosis) occurring up to several weeks post-injury – even in mTBI (Martin, 2016; McFarlane & Glenn, 2015). The impaired function of damaged neurons can then also impair the function of nearby cells via the inappropriate transmission of metabolites, proteases, and spilt neurotransmitters through the intercellular environment (Martin, 2016). The impact forces can also damage the small blood vessels in the brain, especially those that cross between neural layers or emerge from bone, resulting in petechial hemorrhages and edema (Martin, 2016).

Long before our current imaging techniques, Oppenheimer (1968) visualized DAI during autopsy in patients with confirmed mTBI who died of systemic injury. These findings have now been corroborated using modern imaging techniques. Both magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI), which is an MRI technique able to measure white matter microstructural integrity, have been able to provide evidence of damage consistent with DAI both acutely and more chronically (Ljungqvist et al., 2017; Martin, 2016; Niogi & Mukherjee, 2010). This area of study is still relatively new, but DTI studies have already demonstrated that the most commonly damaged areas in mTBI are the frontal association pathways and the anterior corpus callosum (Niogi & Mukherjee, 2010). Damage to these areas is consistent with many of the cognitive symptoms reported by mTBI survivors (e.g., working memory and attention difficulties).

The neuropathology perspective also cites evidence of more gross brain changes following mTBI. Zhou and colleagues (2013) used MRI to measure brain volume changes over the first year post injury. The individuals with mTBI showed more than twice as much overall brain atrophy when compared to matched controls. Specific locations of significant loss have been implicated in affective and cognitive functioning, including selective depressive symptoms, attention, working memory, executive functioning, processing and responding to error, interference, and regions with many reciprocal connections to frontal systems with involvement in executive functions (Zhou et al., 2013). Other research has explicitly demonstrated a link between structural changes in both white and grey matter evident at one year post-injury and post-concussion symptoms, which the authors argue is suggestive of a neurophysiological basis for these persistent symptoms (Dean, Sato, Vieira, McNamara, & Sterr, 2015).

In addition to structural changes, changes in brain function have also been reported following mTBI. There is mounting evidence that functional MRI (fMRI) is sensitive to the changes in neural function following mTBI (Jantzen, 2010). For example, Hammeke and colleagues (2013) used fMRI to investigate brain functioning in conjunction with cognitive performance. Very acutely, individuals with mTBI were found to have decreased activity in areas associated with attention when compared to non-injured, matched controls, as well as poorer performance on tasks of memory and reaction time. Seven weeks later, while the mTBI group now performed equally well on the cognitive measures as the control group, the mTBI group showed *increased* activity in the brain regions found to be underactive previously. It is assumed that the underactivity of the brain in the first hours after the injury underlie the observed cognitive difficulties (as well as subjective symptom complaints), while the hyperactivation may represent compensatory brain responses. In other words, weeks after mTBI, even though performance on cognitive measures may have improved, it may take more neural "effort" to complete the same task (Hammeke, et al., 2013). There is also evidence suggesting that alterations of functional connectivity within specific neural networks (i.e., those involving the insula, thalamus, and anterior cingulate cortex) may underlie TBI-induced depression (Moreno-López, Sahakian, Manktelow, Menon, & Stamatakis, 2016).

Further evidence for functional changes after mTBI is based on the results of single-photon emission computed tomography (SPECT) scans. In a study by Hattori et al. (2009), differences in activation patterns were seen at six months post-injury when mTBI survivors were compared with healthy controls. The participants with mTBI had decreased cerebellar activation and increased prefrontal activation during a cognitively demanding complex attention task (the Paced Auditory Serial Addition Test; PASAT) compared to the controls. The altered pattern of activation in the mTBI group appeared consistent with their poorer performance on the first trial of the PASAT, as well as their reports of significant cognitive fatigue (i.e., the inability to maintain attention and concentration during a sustained task).

In summary, the perspective that injury-related neuropathology is the underlying cause of post-concussion syndrome is based on a range of evidence for cerebral damage and/or change following even very mild TBI in both the acute and chronic stages. Furthermore, the damage is consistent with many of the symptoms commonly reported and may (at least partially) explain why ongoing symptoms are reported even when cognitive deficits are seemingly absent (particularly when less sensitive/cognitively demanding tasks are used). That being said, a number of questions remain including: are these neurological factors found in all individuals who sustain an mTBI or only patients who report ongoing symptoms? Are the neurological changes sufficient to account for the range of symptoms reported? If not, which symptoms are they likely to be causing? While these questions are beyond the scope of the current study, it is hoped that future research in this area will help to clarify some of these issues.

Psychogenic perspective. The other perspective views post-concussion syndrome as primarily psychogenic, and the result of a multitude of confounding factors. From this perspective, the brain injury is often viewed as an impetus for new or increased psychological dysfunction. It has been purported by supporters of this perspective that spontaneous neurological recovery occurs fairly rapidly in most individuals, so symptomatic and functional recovery is instead hampered by these psychological difficulties (e.g., Silverberg & Iverson, 2011). In fact, most researchers from this perspective seem to question the validity of post-concussion syndrome as a unique clinical entity apart from other psychological diagnoses. Within those who espouse the psychogenic perspective is a subgroup of individuals who believe that post-concussion syndrome is not only psychologically driven, but that it is in fact a type of malingering. This belief is rooted in reports that demonstrate a systematic increase in the number and severity of symptoms reported by individuals who are in the process of litigation (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Iverson & Lange, 2011b). Malingering is an issue among those involved in litigation or compensation suits (Carroll et al., 2004) as these individuals stand to make material gain from reporting more severe symptoms (Iverson & Lange, 2011b). Individuals with mTBI who show suboptimal effort or symptom exaggeration on specifically designed measures (e.g., the Test of Memory Malingering and the Victoria Symptoms Validity Test) also report more post-concussion symptoms, have more self-reported cognitive complaints, and also perform more poorly on objective neuropsychological measures than do individuals without questionable performance on effort measures (Lange, Iverson, Brooks, & Rennison, 2010). The cumulative results of a number of meta-analyses have demonstrated that malingering has a larger effect on neuropsychological functioning than even acute moderate to severe brain injury (Iverson & Lange, 2011a). Findings such as these have led some authors to regard the diagnosis of post-concussion syndrome as completely invalid.

The psychogenic perspective is further bolstered by research demonstrating that post-concussion-like symptoms are reported in a myriad of other populations. For example, neurologically intact individuals with chronic pain, depression (Lange, Iverson, & Rose, 2011), general trauma/minor injury (McCauley, Boake, Levin, Contant, & Song, 2001; Mickevičiene et al., 2004) and even healthy individuals (Dean, O'Neill, & Sterr, 2012; Garden & Sullivan, 2010) have all been found to report post-concussion-like symptoms and sometimes report sufficient symptoms to meet criteria for post-concussion syndrome (minus the requirement for head injury, of course). Despite evidence that a greater number and more severe post-concussion symptoms are reported, and that these symptoms are reported at a higher rate after mTBI than other injuries or within the general population (Carroll et al., 2004), the symptoms – and even the constellation of symptoms – are not unique to TBI. These findings have resulted in many individuals calling into question the validity of post-concussion syndrome following mTBI.

The psychogenic perspective emphasizes the role of comorbid conditions in post-concussion syndrome. A number of studies (including meta-analyses) have demonstrated that the "typical" mTBI survivor no longer shows significant neuropsychological difficulties by 3 months post-injury (Karr, Areshenkoff, & Garcia-Barrera, 2014). Observations such as these led some individuals to believe that, in the absence of co-morbid conditions, the injury itself was unable to cause lasting neurological sequelae (e.g., Lishman, 1988; Mittenberg & Strauman, 2000). As discussed earlier, psychological conditions, particularly depression and anxiety disorders, are common among individuals with mTBI. Further, early psychological distress has been shown to influence both the acute and chronic presentation of post-concussion syndrome (Silverberg & Iverson, 2011). When taken together, these lines of research may suggest that most individuals with mTBI will have recovered (physically and neurologically) from the injury by three months, and that the experience of ongoing post-concussion symptoms is more likely to relate to a comorbid condition (Iverson & Lange, 2011b). However, even if one does not want to make the immediate assumption that comorbid conditions are solely responsible for the experience of persistent post-concussion symptoms, the task of differentiating with certainty the impact of the brain injury from the potential impact of comorbid conditions can be extremely difficult. This is because, in order to confidently link the persistent symptoms to a (now) remote mTBI, it would be necessary to have documented evidence of the presence of the symptoms from the initial weeks post-injury, continuing – with only modest improvements – for the following months (Iverson & Lange, 2011b). Given that most individuals who sustain an mTBI do not seek out immediate medical attention, there can be a complete absence of documentation of early symptoms leaving patient self-report as the only source of information, which can be rife with potential issues.

Those from the psychogenic perspective view the need to rely on self-report as a means of assessing post-concussion symptoms as a significant weakness and argue that self-report measures are inherently susceptible to a number of problems including: (unintentional) symptom exaggeration, symptom misattribution, and/or symptom misinterpretation (Iverson & Lange, 2011b). For example, because cognitive difficulties can be objectively measured (although not infallibly, which is important to remember), *perceived* cognitive functioning can be compared to *objective* cognitive performance in order to assess the accuracy of the former. Studies taking this approach have found that subgroups of mTBI survivors with comorbid conditions (post-traumatic stress disorder or pain) report more significant self-perceived cognitive deficits, yet show no differences on neuropsychological testing when compared to mTBI survivors without these co-morbid conditions (Jamora, Young, & Ruff, 2012; Jamora, Schroeder, & Ruff, 2013). Therefore, it seems plausible that other factors are impacting the perceived cognitive functioning of those who reported more significant difficulties.

In a recent review, Iverson & Lange (2011b) point out a number of factors that can influence the perception and reporting of symptoms. First, personality characteristics and styles influence how an individual responds to an illness, injury, disease, or traumatic event. For example, a symptom that is experienced as overwhelming to one individual may be experienced as mildly irritating to another based on one's tendency to over-emphasize cognitive or physical symptoms. Second, expectations of what will happen after an mTBI can cause misattribution of benign symptoms, experienced by most people at one time or another, to the brain injury (e.g., simple acts of forgetting, such as forgetting a grocery item that was not on a list or forgetting where the car was parked in a large parking garage). Third, a tendency to underestimate past problems or difficulties can make the reported discrepancy between past (i.e., pre-injury) and present (i.e., post-injury) functioning more significant than is factually accurate. This is known as the "good old days" bias and, when combined with the expectation of certain symptoms following mTBI, it can have a significant impact on symptom reporting. Finally, stereotype threat – or the threat or fear of fulfilling a negative stereotype – has been shown to impact aspects of stereotype-related functioning in multiple populations (see Kit, 2008 for a review) and can negatively impact cognitive performance in individuals who have sustained an mTBI (Iverson & Lange, 2011b; Kit, 2008).

In summary, the perspective that post-concussion syndrome is of psychogenic origin is based on a selection of results implicating a variety of confounding factors in the experience and/or report of ongoing post-mTBI symptoms. Some authors have highlighted methodological issues that may call into question some of the assumptions that underlie the psychogenic perspective, including the use of meta-analytical data as proof of the lack of neurologically-based persistent symptoms given that this method is likely to obscure the heterogeneity in recovery (Pertab, James, & Bigler, 2009). That being said, the potential impact of psychological difficulties in recovery and the experience of persistent symptoms after brain injury should not be overlooked.

A compromise in the cause of post-concussion syndrome: the neuropsychosocial perspective. It is likely that neither neuropathological nor psychogenic factors in isolation should be regarded as the "true" underlying cause of persistent post-concussion symptoms for all individuals. The neuropathological perspective disregards the commonly accepted impact that psychological difficulties can have on health-related outcomes in general, and the frequency with which psychological concerns are experienced following mTBI. On the other hand, the psychogenic perspective ignores the fact that some degree of damage has occurred to the brain and that research is beginning to demonstrate lasting structural and functional changes associated with even mild injuries. Further, the role of other neurobiological factors (e.g., brain reserve) in recovery and the experience of ongoing symptoms is unclear. Moreover, both perspectives ignore the impact that psychosocial factors – a relatively newer area of investigation within the mTBI literature - could have on recovery and the experience of persistent symptoms. Most experts now agree that a whole-person, or a neuropsychosocial, perspective of recovery from mTBI and post-concussion symptoms is more appropriate (e.g., Silverberg & Iverson, 2011). From this perspective, there is believed to be an impact of neurological, psychological and psychosocial factors on recovery and persistent post-concussion symptoms for most individuals following mTBI (Hou et al., 2012; Ruff, 2005; Ruff & Jamora, 2009). Due to the complexity of the process of recovery after brain injury, Walsh and colleagues (2014) also argue that research on brain injury should be conducted from an integrated theoretical perspective -

incorporating both the social psychology and clinical neuropsychology perspectives. These authors posit that such an integrated perspective may further our understanding of aspects of brain injury that have, heretofore, remained poorly or partially explained by clinical neuropsychology alone - including the "gap between functional impairment and neurological injury" (p.459) - which may be particularly relevant to mTBI (Walsh et al., 2014). One such psychosocial factor that has recently begun to garner attention in the literature is identity, and the role that it may play in recovery.

Identity as a new contributing factor. Some of the most enduring changes after brain injury can relate to the survivors' subjective experience of who he or she is – in other words, their identity (see Carroll & Coetzer, 2011). Survivors often compare their current, post-injury self to their past, pre-injury self (Muenchberger et al., 2008) with their current self commonly perceived more negatively than their past self (Carroll & Coetzer, 2011). Within mTBI specifically, this can be seen in what has become known as the "good old days" bias which refers to the tendency to view one's past self as healthier and to *under*estimate past problems (Iverson, Lange, Brooks, & Rennison, 2010). In conjunction, individuals with mTBI can become hyper-aware of current deficits and *over*estimate current problems (Sawchyn, Mateer, & Suffield, 2005). Therefore, individuals with mTBI are prone to perceive significant negative changes in self-attributes after the injury.

This line of research (e.g., Carroll & Coetzer, 2011; Muenchberger et al., 2008) has demonstrated that personal factors of identity, such as beliefs about the "self" and one's personal attributes, can be shaken following TBI. However, the investigations that have been undertaken to date have been, by in large, from a perspective of identity that is relatively individualistic; in other words, the social factors that can underlie the ways that one sees and defines themselves have yet to be given much attention within the TBI literature (Walsh, Fortune, Gallagher, & Muldoon, 2014). For example, how identity may be influenced by one's sense of belonging to social groups and their beliefs about what group membership entails.

Walsh and colleagues (2014) argue that the *social identity approach* is well suited to address the 'social' component of the biopsychosocial model in brain injury research. Further, the social identity approach is one of the most common frameworks used in social psychological research. For example, the social identity approach has been used to conceptualize research on topics as varied as television watching and entertainment preferences (Trepte, 2006), occupational identity (e.g., Kreiner, Ashforth, & Sluss, 2006) and leadership (e.g., Sivanathan, Arnold, Turner, & Barling, 2004), as well as health and well-being in various patient populations including some research on individuals with TBI (e.g., Douglas, 2012; Gracey & Ownsworth, 2012).

Jetten, Haslam, and Haslam (2012) describe the social identity approach as a psychological “metatheory” that is comprised of two related theories: *social identity theory* and *self-categorization theory*. Briefly, social identity theory (which has its origins in work by Henri Tajfel, starting from the late 1950s, which was more fully developed with John Turner in the mid- to late 1970s) is a social psychological theory of intergroup relations, group processes, group behaviour, and the sense of self derived from social groups (e.g., Hogg, Terry, & White, 1995; Jetten et al., 2012). People have a number of discrete category memberships that vary in importance in one's self-concept; however, each of these memberships is a social identity that can both describe and prescribe what one should think and feel, and how one should behave (Hogg et al., 1995). For example, being a fan of a sports team may be an important aspect of one's self-concept when in certain situations (e.g., when watching a game with others) and be less important in other situations (e.g., when in a meeting at work); when in situations where this identity (i.e., team fan) is relevant, thoughts, feelings and behaviours become in-group stereotypical and normative (e.g., wearing team memorabilia, joining in on rituals with other fans, celebrating wins and commiserating losses, etc.).

Self-categorization theory (developed by Turner and colleagues in the 1980s and 1990s) extends the ideas regarding the importance of social identity in social behaviour and speaks to the way that individuals define and understand themselves in a given context or situation (Jetten et al., 2012). Humans have a strong tendency to categorize themselves and others into "in-groups" and "out-groups", where those that are like us belong to the in-group, and others belong to the out-group (Hogg et al.,1995). In general, the process of categorization of any stimuli (e.g., tables and chairs) has the effect of accentuating our perception of the *similarities* between stimuli belonging to the *same* category, and our perception of the *differences* between stimuli belonging to *different* categories (Hogg et al.,1995). Similarly, categorization of self and others into in-group and out-groups has the same effect and thus accentuates the ways that individuals fit with our cognitive representation of either group's defining features (and, thus, accentuates our perception of the ways that others are the same as, or different from, us). When a specific social identity becomes salient, self-perception and conduct become "in-group stereotypical and normative" (Hogg et al., 1995, p. 260) – somewhat analogous to the proverb, "When in Rome, do as the Romans do."

Regarding mTBI, St. Claire and Clucas (2012) have argued that illness labels (such as those associated with medical conditions) can be regarded as analogous to group labels, with symptom constellations analogous to the behavioural norms of the group. As such, when an individual self-categorizes as a member of a given illness or diagnostic group, their perception of their own symptoms should converge towards those that are associated with that illness or diagnosis. For example, it has been found that even among cold sufferers – a group with temporary membership and limited self-relevance – more cold-related symptoms were reported when the salience of their self-categorization as a member of the group ‘people with colds’ was primed than do cold-sufferers who do not have this group membership primed (St. Claire & Clucas, 2012).

Therefore, in addition to changes in one's self-perception following a TBI, an individual may begin to self-categorize into new groups while old groups or roles may be abandoned (or made less salient for a time) (Kit, 2009; Muenchberger et al., 2008). One of these new self-categorizations may be that of a TBI survivor. The social identity approach suggests that, the more salient this categorization is to the individual, the more their behaviour and experiences will converge with their cognitive representation what a TBI survivor is like, and the norms for the group may be incorporated into their self-identity.

Despite a strong theoretical basis for how or why identity may function as a determinant of outcome following brain injury, it has yet garnered very limited attention within the literature. That being said, Walsh, Muldoon, Gallagher, and Fortune (2015) have investigated identity following acquired brain injury (ABI) of varying severity. The sample was comprised of individuals between the ages of 20 and 65, an average of seven years post-injury, with a variety of ABI etiology including stroke, motor vehicle collision, falls, tumour, assault, or hypoxia. In the study, Walsh and colleagues (2015) differentiated between affiliative identity (e.g., groups that we belong to such as family, nation, gender) and 'self-as-doer' identity (e.g., identities associated with occupation). They found that, in the context of ABI, the relationship between affiliative identity and emotional status was mediated by social support and self-as-doer identity. In other words, it seems that affiliative identities generate perceived and functional social support, which may facilitate participation in activities that then become internalized as self-as-doer identities, and influence well-being (Walsh et al., 2015). The authors suggest that these results demonstrate that affiliative identity (or identity built on belonging) is necessary, but not sufficient, for positive emotional status post-ABI. Engagement in meaninful activities that facilitate identity construction seems to be required for positive post-ABI adjustment. The authors also purport that these results affirm the utility of social approaches to neuropsychology (Walsh et al., 2015). It is important to note that participants identified their own affiliative and self-as-doer identities via open-ended questions. The identity of self as a brain injury survivor was not studied directly, nor was the influence of these aspects of identity on recovery or post-concussion symptoms.

The Current Study

The literature reviewed above demonstrates that mTBI can result in ongoing cognitive and emotional difficulties in some survivors, and that these variables likely play some role in the experience of persistent post-concussion symptoms. However, this literature also demonstrates that each of these post-injury factors is inconsistently related to persistent post-concussion symptoms. In addition, while persistent problems with self-perception can be experienced after mTBI, the potential impact of identity on post-concussion symptoms has yet to be investigated. Furthermore, the potential relationship between these factors as they relate to persistent symptoms/post-concussion syndrome is even less clear. Despite some experts acknowledging the multifaceted nature of post-concussion syndrome and the need to consider various factors together in order to gain a better understanding of persistent post-concussion symptoms, research into more complex predictors has only recently begun. As such, the current study investigates persistent post-concussion symptoms from a neuropsychosocial perspective with the aim of further elucidating the factors that differentiate those who make a good recovery from those who report ongoing symptoms after mTBI. A within subjects design was used to investigate the relationship that specific variables have with self-reported persistent post-concussion symptoms, as well as exploring potential interactions among the variables in the prediction of the severity of persistent post-concussion symptoms. The specific variables of interest in the current study are: objective cognitive functioning, anxiety susceptibility, and identity and their potential influence on persistent post-concussion symptom severity.

Persistent post-concussion symptoms (PCS). The current study examined the severity of persistent post-concussion *symptoms* (PCS), and *not* the presence or absence of the syndrome for a number of reasons: First, there is no clear consensus on the diagnostic criteria for the syndrome and, depending on the criteria-set used, participants could be categorized differently (i.e., some people who would be diagnosed using one set of criteria would not be diagnosed using the other) making the relevance of the results questionable. Second, using the presence/absence of diagnosis instead of symptom severity limits the applicability of the results by taking heterogeneous individuals with a range of problem frequencies and severities and treating them as homogeneous. Finally, creating dichotomous groups is statistically problematic as it greatly reduces the variability in the data making significant results harder to obtain and limiting the types of analyses that can be performed.

Cognitive functioning. Cognitive functioning in specific domains was assessed in order to investigate their role in predicting persistent PCS. Domains included were attention, processing speed, working memory, and verbal fluency. These domains were chosen because they are the primary areas of cognitive deficit following mTBI.

In general, studies investigating cognitive functioning after mTBI are conflicting. When investigated as a predictor of PCS, the impact of cognitive functioning has been inconsistent. Even investigations of the *presence* of cognitive deficits following mTBI have been inconsistent, and those studies that find no evidence of cognitive difficulties have been used to argue that PCS are invalid. However methodological concerns, primarily the sensitivity of the chosen measures, may (at least in part) account for studies with null results. Therefore, in addition to investigating domains that are commonly affected in mTBI, the measures used in the current study were chosen based on evidence demonstrating that they: a) measure the intended domain; and b) have sufficient sensitivity to detect deficits, if present.

Anxiety susceptibility. The current study looked at what will be referred to “anxiety susceptibility”, which is conceptually different from general anxiety, worry, or stress. We have defined anxiety susceptibility is the likelihood of experiencing anxiety in a particular situation. In the current study, anxiety susceptibility was assessed in the context of a cognitively and emotionally demanding situation, which might be particularly anxiogenic because the person has experienced a brain injury.

The existing literature provides evidence that trait/general anxiety influences the experience of PCS, yet a consensus is lacking because some studies have failed to find a significant effect of anxiety on persistent PCS. Furthermore, the inclusion of anxiety as a predictor is complicated by the fact that anxiety is regarded as a symptom of post-concussion syndrome. Therefore, anxiety susceptibility was investigated in hopes of further clarifying what aspects of anxiety are relevant to PCS, while avoiding the circular logic of using an aspect of the outcome variable as a predictor. It was theorized that context-dependent anxiety susceptibility could be an issue for individuals regardless of one's overall/general level of anxiety because situations that induce feelings of self-doubt, self-consciousness, and performance-related (hyper)vigilance could cause an increase in/exacerbation of perceived anxiety symptoms for individuals with high or low general/trait anxiety.

Identity. The possible effects of mTBI on identity is a relatively new area of investigation, yet evidence demonstrates that negative changes are common, and identity-related difficulties are persistent. However, the influence of post-injury identity on the experience PCS had not been previously investigated.

Multiple components of identity were investigated in the current study, including self-perception based on personal attributes, TBI-related self-concept (or how much the brain injury affects how the person sees themselves), as well as TBI-related social identity (based on self-categorization into, affiliation with, and belonging to the wider TBI community). According to the social identity approach, a strong TBI-related self-identity may influence self-perceived brain injury symptoms and PCS.

Research Hypotheses

Based on a neuropsychosocial perspective, and informed by the current body of literature, a model was proposed for the possible influence of cognitive functioning, anxiety susceptibility, and identity on PCS (see Figure 1). The various aspects of this model were investigated via the following hypotheses:

1. Cognitive impairment, as a potential indication of brain dysfunction, would directly predict self-reported PCS severity, with higher levels of cognitive impairment associated with more severe PCS;

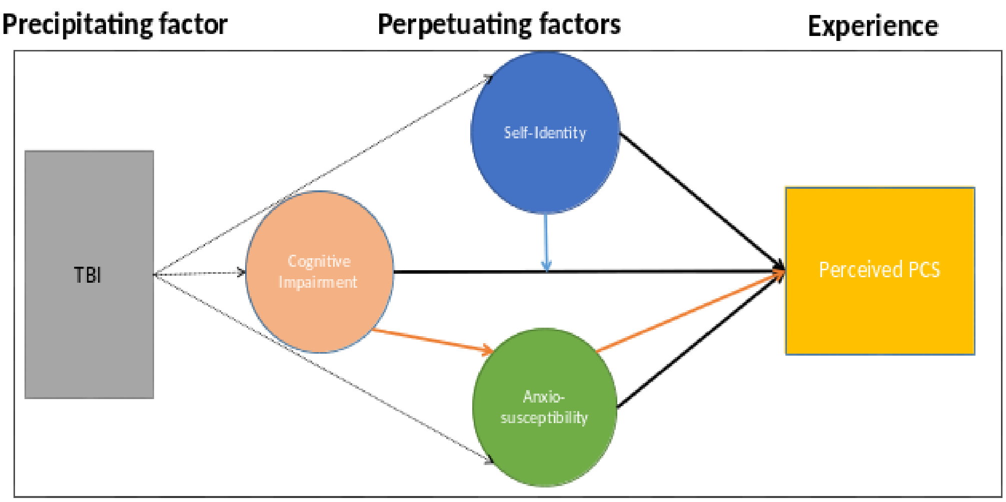

Figure 1. Model displaying the proposed effects of, and relations between, cognitive functioning/impairment, anxiety susceptibility, and identity on perceived PCS. The grey arrows represent effects that will not be investigated, the black arrows represent direct effects, the orange arrow represents a mediation effect, and the blue line represents a moderation effect.

2. Anxiety susceptibility would directly predict PCS severity, with those who experience higher levels of anxiety susceptibility reporting more PCS. In addition, the strength of anxiety susceptibility as a predictor of PCS compared to a more common type of anxiety (i.e., the level of general, or trait, anxiety an individual experiences) was also investigated;
3. Anxiety susceptibility would moderate the relationship between cognitive impairment and PCS such that individuals with cognitive difficulties may be more susceptible to anxiety in cognitively demanding situations and this combination of experiences would likely influence the experience of PCS;

4. The various aspects of identity (i.e., self-perception, TBI-related self-concept, and TBI-related social identity) would directly predict PCS severity with those with more negative self-perception and/or stronger TBI-related identity reporting more severe PCS;
5. Identity would moderate the relationship between cognitive functioning and PCS such that individuals with more negative self-perception and/or stronger TBI-related identity would report more severe PCS regardless of cognitive functioning, but those with less negative self-perception and/or weaker TBI-related identity would only report more severe PCS if they also had poor cognitive functioning; those with less negative self-perception and/or weaker TBI-related identity would report less PCS if they had good cognitive functioning.

Methods

Participants

Given the purpose of this study was to investigate neuropsychosocial factors that may play a role in persistent PCS following mTBI in adults, all participants had to be between the ages of 17 and 65 and be at least one year post-injury with confirmed mild TBI. For the purposes of the present study, mTBI was classified using the diagnostic criteria agreed upon by the ACRM MTBI Committee, the CDC working group, and the WHO Collaborating Centre Task Force (see Iverson & Lange, 2011a). Namely, a traumatic injury to the head (or involving the head through processes such as abrupt acceleration and/or deceleration of the body) with subsequent evidence of immediate symptoms such as: a period of confusion, disorientation, or loss of consciousness lasting less than 30 minutes; any loss or dysfunction of memory for the events surrounding the accident, with PTA lasting less than 24 hours; and/or neurological focal signs such as seizure, headache, dizziness, irritability, fatigue, and/or poor concentration immediately following the injury or in the acute post-injury period (i.e., the subsequent hours and days). In the current study, information on injury severity was acquired through participant self-report and corroborated by an informant who knew the participant well at the time of the injury. Medical documentation was not collected due to practical reasons (i.e., the cost and time needed to request documentation from family physicians and emergency room departments) and therefore information on the participants' GCS rating was not obtained. Some of the participants expressed uncertainty regarding their length of PTA or LoC, which is typical of individuals with brain injury (e.g., Kit, 2009). However, all individuals who were unable to provide a length of PTA or LoC, did endorse a period altered consciousness (e.g., feeling “foggy,” “hazy,” or confused) immediately following the injury.

Interested individuals were excluded from participation if they: (1) had sustained a non-traumatic brain injury (e.g., stroke, encephalitis); (2) had sustained a mTBI less than one year prior; (3) had any history of a severe TBI; (4) had a history of any other major neurological illness or disorder (e.g., Multiple Sclerosis, Parkinson’s disease, brain tumour); (5) had any other major medical conditions that can affect cognition (e.g., Type II diabetes); (6) were currently involved in litigation related to the injury; (7) had a history of severe psychiatric illness (e.g., schizophrenia) or current substance abuse/ dependence; and (8) reported clinically significant symptoms of Post-Traumatic Stress Disorder (based on a total score greater that 1.60 on the Impact of Event Scale – Revised; Weiss & Marmar, 1997).

Recruitment. Three main recruitment strategies were used in this study. First, the majority of participants were recruited via posters and brochures that were placed in locations around the Greater Victoria Area including the Victoria Brain Injury Society, Victoria General Hospital's Outpatient Neurorehabilitation Unit, public recreation centers, Public Health Units, and the offices of various health care practitioners (e.g., Clinical Psychologists, Neurologists, Physiotherapists). Materials (both hard and electronic copies) were also provided to a number of local sports associations, including the Gorge Soccer Association, Vancouver Island Soccer League, Lower Island Soccer Association, and the Westshore Rugby Football Club. Second, advertisements were placed in the CFB Esquimalt Naval Base newspaper (“The Lookout”) and also on various websites including UsedVictoria, Craig's List, and Kijiji. The third recruitment strategy made use of the Island Health Permission to Contact (PTC) program. Individuals, at the time of their contact with an Island Health service or program, provide consent to be contacted for future research projects that they may be eligible for. The PTC program manager was provided with the general eligibility criteria and then contacted all program participants who met these criteria via email. For all three methods of recruitment, interested individuals then had to contact the researcher themselves, via email or telephone, in order to initiate participation.

Power analysis. An *a priori* power analysis was used to inform the minimum sample size needed for this study, drawing on relevant literature for similar studies on mTBI. Within the literature, studies that included correlations between variables as similar as possible to those being investigated in this study were found. Whenever possible, studies including a mild TBI sample were used; however, some variables have yet to be investigated in a population of TBI survivors using similar methods (e.g., qualitative vs. quantitative studies of social identity). The open software program G*Power (Faul, Erdfelder, Buchner, & Lang, 2009) was used to determine the sample size needed to obtain an effect size of 0.61 (a moderate effect size), with 2-tailed $\alpha = .05$, for a multivariate regression analysis. The results indicated that a sample size of at least 19 participants was required, based on the evidence available within the recent literature.

Measures

Participants completed a number of self-report questionnaires (see Appendix) and neuropsychological assessment measures. The measures included in the study were chosen for a variety of reasons including acceptable psychometric properties (including sensitivity to the effects of mTBI, whenever possible), prior use in TBI-related research, and administration/completion time.

Questionnaires.

Center for Epidemiologic Studies Depression Scale (CES-D). The CES-D (Radloff, 1977) is a 20-item measure of one's current level of depressive symptomatology. Participants are asked to rate how often they felt or experienced what is described in the item (e.g., “I was bothered by things that usually don't bother me) over the past week. Each item is rated on a scale from 0 (Rarely or None of the time; < 1 day) to 3 (Most or All of the time; 5 – 7 days) with a score range of 0 to 60 and higher scores indicating greater frequency/severity of depressive symptomatology.

The CES-D was designed for use in the general population. Therefore, its main purpose is not to diagnose depression, but instead to measure a variety of symptoms associated with a diagnosis of clinical depression with an emphasis on negative affect (or depressed mood). The major components of depressive symptomatology included are: depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, psychomotor retardation, loss of appetite, and sleep disturbance. These components were identified via review of the clinical literature and factor analytic studies (Radloff, 1977). A cutoff score of 16 has been used by various studies to distinguish individuals with clinical depression from those without depression (e.g., see Boyd, Meissman, Thompson, & Myers, 1982). Administration of the CES-D to probability samples of households designed to be representative of two US communities (Kansas City, Missouri and Washington County, Maryland; $N = 2514$) resulted in a mean total score of 9.25 (SD = 8.58), with 19% of the sample scoring at or above a total score of 16 (Radloff, 1977).

Initial investigations of the CES-D found it to have good discriminability between psychiatric inpatient and general population samples, with patient populations scoring significantly higher than groups of individuals from the general population, as well as moderate discriminability among different levels of severity within patient groups (r = 0.56 for patient CES-D scores and nurse-clinician ratings of depression severity, Radloff, 1977). Further, the CES-D was found to have good internal consistency in both general and patient populations (Cronbach's α = 0.84 - 0.85 for general population, α = 0.90 for patient population; Radloff, 1977), in adults with systemic sclerosis (Cronbach's α = 0.88; Thombs et al., 2008), in older adults with and without cognitive impairment (Cronbach's α = 0.88; Ros et al., 2011), and also in a sample of individuals with mild to moderate TBI (Cronbach's α = 0.93; McCauley et al., 2006).

Collective Self-Esteem Scale for TBI – Identity subscale (CSE-TBI-I). The *Collective Self-Esteem Scale* (Luhtanen & Crocker, 1992) was developed to assess individual differences in collective, rather than personal, self-esteem or "the positivity of one's social, or collective, identity" (p. 302) based on Social Identity Theory. The full measure has 16 items across four subscales: Membership Esteem, Public Collective Self-Esteem, Private Collective Self-Esteem, and Importance to Identity. Responses to all items are on a 7-point Likert-type scale where 1 = strongly disagree and 7 = strongly agree (Luhtanen & Crocker, 1992). Principal components analysis using varimax rotation showed that 72.3% of the total variance was accounted for by the four factors; this factor structure was supported via confirmatory factor analysis using maximum likelihood estimation (Luhtanen & Crocker, 1992). Also, across three studies, the CSE had good internal consistency (Cronbach's α = 0.85 – 0.88; Luhtanen & Crocker, 1992).

As originally designed, the CSE attempts to assess individuals' levels of social identity based on their memberships in ascribed groups pertaining to gender, race, religion, ethnicity, and socioeconomic class with the aim of creating a single measure that could capture general, cross-group social identity (Luhtanen & Crocker, 1992). However, the measure has since been modified for use with specific groups, including TBI survivors (CSE-TBI; see Kit, 2009). In the current study, only the *Importance to Identity* subscale was used, in order to assess how important having sustained a TBI is to the participants' self-concept (e.g., Having a brain injury is an important part of my self-image). The scale includes four items, two of which are reverse coded. Possible total scores range from 4 to 28, with higher scores indicating stronger TBI-related self-concept. A principal components factor analysis, conducted by Luhtanen and Crocker (1992), found that the factor loadings for the four *Identity* subscale items were between 0.72 and 0.77; reliability analyses also indicated that the subscale is internally consistent (Cronbach's α's = 0.73 and 0.86 across three separate studies).

Head Injury Semantic Differential Scale – III (HISDS-III). The HISDS (Tyerman & Humphrey, 1984) is a measure of an individual's self-perception based on the semantic differential paradigm. The updated version of the scale is comprised of 20 adjective pairs, one “negative” and one “positive” (e.g., Unhappy – Happy), addressing aspects of personality thought to be pertinent to brain injury. Items are rated on a 7-point scale (1 = negative pole, 7 = positive pole); higher scores indicate more positive views of self. In the current study, the total score represented the mean of the ratings provided. This was done in order to aid interpretation through maintaining the original 7-point scale (i.e., total scores from 1 to 3 represent more negative self-perception, total scores of 4 are neutral, and total scores from 5 to 7 represent more positive self-perception). Three versions of the scale are available: past (i.e., prior to the injury), present, and future self. In the current study, the past (i.e., HISDS-pre) and present (i.e., HISDS-post) versions were utilized. In order to assess the change in self-perception since the injury, a difference score was calculated (HISDS-diff = [HISDS-post] – [HISDS-pre]) such that higher difference scores indicate more positive post-injury self-perception and lower difference scores indicate more negative post-injury self-perception. Carroll & Coetzer (2011) report good internal validity for this measure, with Cronbach's $\alpha = 0.93$ for the past version and 0.92 for present version, which was similar to the values found in previous studies of adults with TBI.

Impact of Event Scale – Revised (IES-R). The IES (Horowitz, Wilner, & Alvarez, 1979) was developed as a short self-report measure designed to assess the degree of symptomatic responses to a specific traumatic exposure as they were experienced in the previous seven days. The IES-R was designed to assess the three symptoms clusters necessary to meet the diagnostic criteria for post-traumatic stress disorder (PTSD) in the DSM-III (American Psychiatric Association, 1980), namely: Intrusion, or the experience of intrusive thoughts and feelings; Avoidance, or the avoidance, denial, or blocking of thoughts and images; and Arousal, or physiological hyperarousal (e.g., increased startle reflex, restlessness, vigilance) (Weiss, 2004).

The IES-R (Weiss & Marmar, 1997) includes 22 items, each rated on a 0 (Not at all) to 4 (Extremely) scale to indicate the degree of *distress* the symptom caused the examinee over the past 7 days. Total scores are presented as the mean of all responses, thus allowing identification of the general degree of distress and symptomatology reported (Weiss, 2004).

The IES-R total score showed high internal consistency for Vietnam veterans (Cronbach's $\alpha = 0.96$; Creamer, Bell, & Failla, 2003), survivors of acute lung injury (Cronbach's $\alpha = 0.96$; Bienvenu, Williams, Yang, Hopkins, & Needham, 2013), and survivors of major motor vehicle accidents (Cronbach's $\alpha = 0.95$; Beck et al., 2008). The construct validity, based on correlations between the IES-R and other measures of PTSD, is acceptable: for example, IES-R total scores were strongly correlated with scores on the PTSD Checklist (*r* = 0.84; Creamer et al., 2003) and also with scores on the Clinician Administered PTSD Scale (CAPS) (*r* = 0.80; Bienvenu et al., 2013).

For the current study, a total mean score on the IES-R of 1.60 was used to determine eligibility such that individuals who scored above the cutoff of 1.60 were ineligible for the full study. This cutoff was chosen based on previous research with various populations (e.g., Bienvenu, Williams, Yang, Hopkins, & Needham, 2013; Sumpter & McMillan, 2005; Wade, Hardy, Howell, & Mythen, 2013) and was the cut-off used by all studies published between 2008 and 2012 that used the IES-R as a measure of PTSD symptomatology (Wade et al., 2013). Further, based on measures of sensitivity, specificity, positive and negative predictive values, and likelihood ratios, Bienvenu and colleagues (2013) found that a total mean score of 1.60 was optimal for purposes of screening for full diagnosis.

Neurobehavioural Symptom Inventory (NSI). The NSI (Cicerone & Kalmar, 1995) is a 22-item questionnaire designed to assess the severity of PCS, or the degree to which the participant has been bothered or distressed by symptoms often experienced after a mild TBI, over the past two week period. Participants rate each symptom on a five point scale (from 0 to 4), with a description for each point on the rating scale based on the symptom's frequency, the degree of functional disruption it causes, and the participant's level of concern or perceived need for help regarding the symptom. For example, a rating of 0 is defined as "None; rarely if ever present; not a problem at all" and a rating of 4 is defined as "Very severe; almost always present and I have been unable to perform at work, school, or home; I can probably not function without help". Possible total scores range from 0 to 88. Cluster analysis yielded four sets of symptoms: an affective cluster, a cognitive cluster, a somatic cluster, and a sensory cluster (Cicerone & Kalmar, 1995). Raw scores based on the sum of all responses for the full measure (i.e., total) and the respective items for the Cognitive cluster (or subscale) were used in the current analyses.

The NSI is commonly used by the United States' Department of Veterans Affairs to measure post-concussive symptoms as a part of its comprehensive traumatic brain injury evaluation, leading to the development of normative data (Soble et al., 2014). These norms indicate that an NSI total score from 58 to 88 represent the 1st percentile; scores from 46 to 57 represent the 2nd - 4th percentile; scores from 34 to 45 represent the 5th - 9th percentile; scores from 25 to 33 represent the 10th - 24th percentile; scores from 11 to 24 represent the 25th - 50th percentile; and NSI total scores between 0 and 10 represent those at or above the 51st percentile (Soble et al., 2014). Therefore, NSI total scores at or below 24 can be regarded as within the average range (i.e., > 25th percentile) for the mTBI population. It is important to note, however, that these norms are based on the scores obtained by members of the Florida National Guard who sustained an mTBI while deployed (and screened negative for PTSD). Unfortunately, no other normative data currently exist.

Regarding the measure's psychometric properties, the NSI total score has been found to have high internal consistency in a large sample of veterans, with Cronbach’s α ranging from 0..93 to 0.95 (Soble et al., 2014 and King et al., 2012, respectively), as well as in a sample of community-living mTBI survivors (Cronbach’s α = 0.96; Silva, Barwick, Kretzmer, Vanderploeg, & Belanger, 2013). High test-retest reliability has also been demonstrated for the NSI with an inter-test interval of 7 days ($r = 0.94$; Silva et al., 2013).

Numerical Pain Rating Scale (NPRS). The NPRS is a measure of perceived pain intensity. Participants are asked to rate their current level of pain on a numerical point scale (from 0 to 10) with extreme anchors of 'no pain' to 'extreme pain'. Because the NPRS includes only one question, it provides a quick and easy measure of subjective level of pain in the moment. This approach was appropriate for this particular study given that pain was not a major focus of the research. Instead, level of in-the-moment pain was assessed because of the known impact that pain can have on reports of mood (e.g., Blågestad, Pallesen, Grønli, Tang, & Nordhus, 2016) and cognitive functioning (e.g., van der Leeuw et al., 2016). As such, it was important to assess the level of pain experienced at the time of the face-to-face session in order to rule it out as a factor influencing performance.

The test-retest reliability for the NPRS has been demonstrated to be moderate to high, ranging from 0.67 to 0.96 (see Kahl & Cleland, 2005 for a review). Further, the NPRS's convergent validity with the Visual Analogue Scale of pain have ranged from 0.79 to 0.95 (Kalh & Cleland, 2005) thus indicating that these two measures - which purport to assess the same phenomenon – do, in fact, measure the same construct and yield similar results.

State-Trait Anxiety Inventory (STAI). The STAI (Form Y; Spielberger, 1983) was developed to assess both *state* and *trait* anxiety. State anxiety exists at a given moment in time and at a particular level of intensity. It is transitory and subject to the influence of relevant situational factors present in the moment (Spielberger, 1983). Trait anxiety, on the other hand, is conceptualized as relatively stable anxiety-proneness. It is an enduring tendency to perceive the world in a certain way (i.e., to perceive stressful situations as dangerous or threatening) and react or behave in a specified manner with predictable regularity (i.e., to consistently respond to stressful situations with elevations in anxiety intensity) (Spielberger, 1983).

The STAI includes two separate self-report scales: the STAI-State (STAI-S) to assess state anxiety and the STAI-Trait (STAI-T) to assess trait anxiety. The STAI-S consists of 20 statements that evaluate how the participant feels "*right now*, at this moment". Each statement is rated on a 4-point scale, from Not At All (1) to Very Much So (4) with 10 statements being reverse coded. The STAI-T is comprised of 20 statements that assess how the participant *generally* feels. Each statement is rated on a 4-point scale, from Almost Never (1) to Almost Always (4) with 9 statements being reverse coded. For both the STAI-T and the STAI-S, the ratings are summed providing a total score between 20 and 80 for each measure, with higher scores indicating higher levels of anxiety. Norms, stratified by age and gender, exist for both forms of the STAI (See Spielberger, 1983) and these standardized T-scores were used in the current analyses.

In a study of the reliability generalization for the STAI, based on 117 reliability coefficients obtained from 45 articles, Barnes, Harp, and Jung (2002) found that for the STAI-T internal consistency ranged from 0.72 to 0.96 with an average of 0.89 (median = 0.90), and for the STAI-S internal consistency ranged from 0.65 to 0.96 with an average of 0.91 (median = 0.92). The test-retest reliability across these 45 articles for the STAI-T tended to be higher than that for the STAI-S – ranging from 0.82 to 0.94 versus 0.34 to 0.96, respectively. This outcome is not surprising: given that the STAI-S is a measure of *state* anxiety, it would be expected to have lower stability over time as it should reflect the influence of unique situational factors that exist at the time of testing (Spielberger, 1983). Overall, the STAI seems to have quite stable internal consistency and test-retest reliability across various studies and, although somewhat variable, the reliability of the scores on the STAI-T and STAI-S are generally satisfactory.

In the current study, the STAI-S was administered twice: immediately before and after a very cognitively demanding task. The goal of this procedure was to assess the participants' susceptibility to anxiety in the context of a cognitively demanding situation (i.e., *anxiety susceptibility*). The STAI-S was chosen for this purpose due to its focus on one's experience, or state, in-the-moment. Scores on the STAI-S generally increase in response to psychological stress, and the scale has been used extensively to assess the level of in-the-moment anxiety induced by stressful experimental procedures, as well as unavoidable real-life stressors (e.g., imminent surgery, job interviews, or important school tests) (Spielberger, 1983).

Neuropsychological Assessment Tools.

Auditory Consonant Trigrams (CCC). The CCC (a.k.a. The Brown-Peterson task; Brown, 1958; Peterson & Peterson, 1959) is considered a measure of working memory ability. The examinee is presented with trigrams consisting of three consonants (e.g. QWR), read aloud by the examiner, which the examinee must then try to recall after a delay of varying length; however, during the delay, the examinee is required to count backwards – aloud – by intervals of three from a number that is given by the examiner immediately after the third letter of the trigram has been presented. At the end of the delay interval, the examinee is asked to recall the trigram. Five trials are given for each delay interval. The Total score represents the number of letters correctly recalled across all 20 items (ranging from 0 to 60). In the current study, the administration procedures described by Stuss, Stethem, Hugenholtz and Richard (1989) were used. Delay intervals of zero, 9, 18, and 36 seconds were used as these lengths are intended to minimize ceiling effects (Strauss, Sherman, & Spreen, 2006).

Construct validity has been assessed, and the CCC has moderate positive correlations with Digit Span Backwards, a recognized measure of working memory (see Strauss et al., 2006). Further, the CCC has demonstrated adequate sensitivity to distinguish patients with “mild concussion” from a normal control group whereby individuals with mild TBI performed more poorly, across all three delay intervals, than did the control group (Stuss et al., 1989).

Controlled Oral Word Association Test (COWAT). The COWAT is a test of verbal fluency. It includes two parts: the first is a phonemic fluency task (i.e., FAS), which includes three trials, each 60 seconds long, during which the examinee is asked to say as many different words as they can that start with a given letter – a different letter is given at the start of each trial. The second part is a semantic fluency task, during which the examinee is asked to name as many different things belonging to a particular category (e.g., animals) as they can in 60 seconds. Norms, stratified by age and education (available in Strauss, Sherman, & Spreen, 2006), were used to calculate *z*-scores for each participant based on the raw number of correct words that were provided in the time limit for each task (i.e., phonemic fluency, or FAS, and semantic fluency, or Animals).

These tasks are generally viewed as assessing aspects of executive function such that they provide a measure of the efficiency of selecting and retrieving information, and require efficient task initiation, planning, organization, monitoring, and flexibility (Birn et al., 2010), as well as attentional control and working memory (see Strauss, Sherman, & Spreen, 2006). Functional MRI studies have corroborated the role of frontal systems in the performance of these tasks (Birn et al., 2010).

Meta-analysis has shown both phonemic and semantic fluency to be sensitive to the effects of TBI, and phonemic fluency to be more sensitive than other commonly used measures of executive functioning (e.g., the Wisconsin Card Sorting Test; Henry & Crawford, 2004). The COWAT has also been shown to be sensitive to the *severity* of acute TBI such that individuals with uncomplicated mild TBI performed significantly better than individuals with complicated mild, moderate, and severe TBI (Iverson, Franzen, & Lovell, 1999).

North American Adult Reading Test (NAART). The NAART (Blair & Spreen, 1989) is used to provide an estimate of premorbid intellectual ability, or level of ability before the onset of known or suspected disease or injury (Schoenberg & Scott, 2011). The NAART is comprised of 61 irregular words that the examinee is asked to read aloud as best they can; they are asked to attempt all words, even if they are unfamiliar. Because the words are irregular (e.g., island, subpoena), phonological decoding or intelligent guesswork will not provide the correct pronunciation. Therefore, it has been argued that performance depends more on previous knowledge than on current cognitive capacity (see Strauss, et al., 2006). Further, the ability to pronounce irregular words is generally resistant to cognitive decline – more so than regular words (Franzen, Burgess, & Smith-Seemiller, 1997). These characteristics make the NAART (and other, similar measures that rely on word reading or vocabularly) a good measure of premorbid functioning. Raw scores are based on the number of words correctly pronounced (between 0 to 61), and z-scores were then calculated using normative data, stratified by age and education (available in Strauss, Sherman, & Spreen, 2006).

In previous research, scores on the NAART were found to have good internal consistency (Cronbach's $\alpha = 0.94$) and scores on the NAART were moderately correlated with full scale IQ on the Wechsler Adult Intelligence Scale – Revised (WAIS-R; $r = 0.75$) (Blair & Spreen, 1989).

Paced Auditory Serial Addition Test (PASAT). The PASAT (Gronwall & Sampson, 1974) is a serial-addition task used to assess working memory, divided attention, and information processing speed. The administration of the PASAT involves presenting a series of single digit numbers and requires the examinee to sum pairs of digits, such that each number is added to the one immediately preceding it on the list, before the presentation of the next digit. It includes four trials of the same series of digits, presented at different interval rates (2.4 seconds, 2.0 seconds, 1.6 seconds, and 1.2 seconds). (Strauss et al., 2006). In the current study, only the 2.4 second trial was administered. Analyses in the current study used *z*-scores calculated based on the number of correct responses given using norms stratified by age and education.

A review of the literature investigating the sensitivity of the PASAT after mild TBI suggests that the PASAT is, in fact, sensitive to the acute cognitive effects of concussion (e.g., within the first week to month post-injury), especially when the injury is associated with significant acceleration/deceleration forces such as occurs in motor vehicle accidents (Tombaugh, 2006). Further, the PASAT has also been shown to have a relatively high degree of sensitivity in patients reporting post-concussion symptoms months to years post-injury – even when care is taken to eliminate the scores of individuals for whom there was an indication of suboptimal effort (see Tombaugh, 2006).

In the current study, the 2.4 second trial of the PASAT was used primarily as an anxiogenic stimulus in order to assess anxiety susceptibility, or the change in anxiety experienced from before to after a cognitively demanding situation. The PASAT was chosen to serve this purpose because it is highly demanding, both cognitively and emotionally. For example, Tombaugh (2006) cites that, even within a sample of young-to-middle aged cognitively intact individuals, the percent of correct responses is relatively low – especially at the faster presentation rates. Further, the PASAT is sensitive to speed of information processing and likely taps into cognitive processes including sustained and divided attention, and working memory (see Tombaugh, 2006), which are cognitive domains commonly affected by TBI. The emotional impact of the PASAT is directly due to how difficult it is and, regardless of one's cognitive status, many researchers and clinicians have indicated that examinees frequently report the task to be frustrating, aversive, and highly anxiety provoking (see Tombaugh, 2006). It has been effectively used in a number of other studies to induce stress (Strauss, Sherman, & Spreen, 2006; Tombaugh, 2006).

Ruff 2 & 7 Selective Attention Test (Ruff). The Ruff 2 & 7 Test is a theoretically based measure of sustained and selective attention, as well as processing speed (Ruff & Allen, 1996; Ruff, Niemann, Allen, Farrow, & Wylie, 1992). The task consists of twenty, 15-second trials of a visual search and cancellation task. In all trials, the examinee is to cross out all of the instances of the digits “2” and “7” that they can before they are signaled to move on to the next trial. There are 10 “Automatic Detection” trials, in which the target digits are embedded among letters, and 10 “Controlled Search” trials, in which the target digits are embedded among other digits. Correct hits and errors (omissions and commissions) are tabulated for each trial, and serve as the basis for computation of the scores (Ruff & Allen, 1996).

In the current study, the *Total Accuracy* score, which is based on the number of targets correctly identified in relation to the number of possible targets, calculated as the number of hits divided by the number of hits plus errors, was used as a measure of sustained attention. The *Total Speed* score, which reflects the total number of correct targets identified, was used to assess processing speed. Both of these scores were converted to standardized T-scores, based on the normative data stratified by age and education, before being used in the current analyses.

The Ruff 2 & 7 Test has demonstrated sensitivity to injury severity in adults with TBI (Strauss et al., 2006). The Total Accuracy score has been shown to have high specificity when individuals with PCS were compared to normal controls (Cicerone & Azulay, 2002) and good construct validity when compared to other tests of attention (e.g., the Test of Everyday Attention) (Strauss et al., 2006). The Total Speed score has been found to have good specificity and a reliable, positive association with PCS. Individuals with PCS are almost twice as likely to have an impaired speed score as controls (Cicerone & Azulay, 2002). There is also evidence for good construct validity when compared to other measures of processing speed (e.g., Digit Symbol) (Strauss et al., 2006).

Victoria Symptom Validity Test (VSVT). The VSVT (Slick, Hopp, Strauss, & Thompson, 2005) is based on a two-alternative forced-choice paradigm, and designed to help confirm (or disconfirm) the validity of an examinee's reported cognitive impairments. In other words, it was designed to assess test-taking effort in order to help determine if an examinee is putting forth poor/sub-optimal effort on cognitive tests which, in the presence of other factors (e.g., intentional feigning in the context of possible secondary gain) may be suggestive of malingering. As discussed earlier, sub-optimal effort, symptom exaggeration, and malingering can be issues following mTBI – most often in the context of litigation or other compensation (Bender & Matusewicz, 2013; Carroll et al., 2004; Iverson & Lange, 2011b). As such, it has become standard practice to include some formal means of assessing effort and symptom validity in clinical studies investigating recovery after mTB in order to aid the interpretation of research results (see Iverson & Lange, 2011b). Therefore, the VSVT was used for this purpose in the current study.

The VSVT is a computer-based task that includes a total of 48 items across three blocks (16 items per block). Each item consists of a five-digit number presented for 5 seconds, followed by a *retention interval* during which the examinee views a blank screen. The retention interval is 5 seconds in the first block, 10 seconds in the second block, and 15 seconds in the third block. Following the retention interval, the examinee is presented with two five-digit numbers, one on the left-hand side of the screen and one on the right-hand side; one will always match the preceding five-digit number (i.e., the target) and the other is a foil. Within each block, half of the items are "easy" and the other half are "difficult": for easy items, the target and the foil share no common digits; for difficult items, the foil is identical to the target except for a transposition of the second and third, or the third and fourth digits. As a part of the standard instructions for the task, the examinee is told that they are "taking a test of memory that requires concentration" and that "people with memory problems often find this test to be difficult" (Slick et al., 2005).

In previous research, scores on the easy and difficult items of the VSVT showed large correlations with a well-validated effort/symptom validity measure (the Rey 15-item test; *r*s = 0.77 and 0.69, respectively), indicating convergent validity (Slick et al., 2005). Divergent validity, demonstrated by small correlations between scores on tests designed to measure dissimilar constructs, was also investigated. Both easy and difficult items showed excellent divergent validity, and no memory test shared more than 5% of its variance with either VSVT scores (Slick et al., 2005). Slick and colleagues (2005) state that divergent validity is particularly important for measures of symptom validity as they should be relatively insensitive to one's *actual* level of cognitive functioning.

Non-standardized measures. In addition to the standardized measures reviewed above, a number of unstandardized measures were included in order to obtain additional information about the participants (see Appendix).

Background Demographics Questionnaire. The background demographics questionnaire included questions such as the participant's level of education, history of attention or learning difficulties, as well as their current and premorbid work history and living situation.

Telephone Screening Interview. The telephone screening interview was developed in order to determine eligibility for the study. In addition to including the IES-R (reviewed above) to assess PTSD symptomatology, the telephone screening interview also included basic demographic questions (age, gender, and level of English proficiency); questions about the participant's history of TBI, including questions assessing the severity and number of previous TBIs; their general physical and psychological health history; any history of illnesses or disorders that can, or have, affected their brain function; and their substance use (current, as well as any history of substance abuse).

In addition to the interview completed with the potential participants, for individuals who were deemed eligible to participate in the study, a collateral interview was conducted with someone who knew the participant well (e.g., a friend, family member, or spouse). The purpose of this interview was to corroborate the information provided by the participant regarding the circumstances surrounding their injury. The collateral interview did not provide any data used in analyses.

TBI Social Identity Questionnaire (TBI-SIQ). The TBI-SIQ was developed for the current study in order to more fully assess the participant's TBI-related social identity – specifically, aspects of affiliation with the brain injury community, feelings of belonging within said community, and self-categorization as an individual with a brain injury were investigated. Development of the 16 TBI-SIQ questions was informed by literature on social identity, as well as questions found within previous studies investigating social identity in other populations. For example, Finney, Noyes, Coutts, and Moos (1998) investigated changes in attitudes, beliefs, and behaviours over the course of inpatient substance abuse treatment in a large number ($N = 3228$) of veterans with alcohol and/or drug abuse/dependence diagnoses. Questions such as "How many AA/NA/CA meetings have you attended since you started treatment?" and "How many of your friends are active in AA, NA, or CA?" were used to asses engagement in 12-step behaviours and activities (p. 373 – 374, Finney, Noyes, Coutts, & Moos, 1998). Walsh, Muldoon, Gallagher, and Fortune (2015) investigated the role of various aspects of identity on emotional status after brain injury using questions such as "Which group of people you belong to is most important to who you are?" (p. 561).

Most of the TBI-SIQ items were rated on a five-point scale, although the response options varied depending on the type of question: some responses were based on the length of time or frequency of involvement in a group or program for individuals with TBI (e.g., from "Did not attend/Less than 1 month" to "More than 12 months" of involvement, or from "None" to "30 or more" sessions attended) with higher ratings indicating a longer length of time or more frequent involvement in the program or group in question; other responses used a Likert-like scale, ranging from "Not at all" to "Extremely" (e.g., "Do you feel that you belong to the larger community of 'brain injury survivors'?”). Analyses were based on a total score (TBI-SIQ), which represents the sum of all[2] ratings, with higher scores indicative of a stronger TBI-related social identity.

Un-Paced Auditory Serial Addition Test (U-PASAT). The U-PASAT is, as its name suggests, an un-paced version of the PASAT. Like the PASAT, it requires the examinee to add together pairs of single digit numbers. The numbers are auditorily presented by the computer (without any additional visual stimulus or cues) and the examinee must then provide their answers aloud. However, unlike the PASAT, the examinee is only presented with two digits at a time and is only provided with the next pair of digits once he/she has provided their answer. The examinee is provided with as much time needed, but is asked to respond as quickly as possible. Immediately after providing a verbal response, the examinee is instructed to press the 'Space Bar’ to indicate that they are ready for the next pair of numbers. The task includes five practice questions to ensure that the examinee fully understands the task, followed by 25 test items. The score is based on the number of items correct out of 25.

This task was developed to help rule out difficulties with basic arithmetic as a major factor in the examinee's performance on the PASAT.

Procedure

Tables 1 and 2 outline the screening procedure and the research protocol, respectively. As mentioned, all potential participants made initial contact with the researcher via email or telephone after learning about the study through one of the recruitment methods described above. During the initial conversation, the potential participant was provided with additional information about the purpose of the study and the procedure, had any questions about the study answered and, if still interested in

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[2] The TBI-SIQ total score does not include the item *“How supported have you felt by these groups/programs or the people involved in them?”* due to a large number of missing data points (10 of the 15 participants did not respond).

Table 1

Screening phases to determine eligibility for study participation.

Phase	*N*	Tests Administered	Exclusion Criteria
Phase 1: Telephone screen	56	IES-R	• Unable to speak/read English • Less than 1 year post-injury • No history of TBI (e.g., stroke or tumor) • Any history of severe TBI • More than 65 years of age at the time of participation • Diagnosis of major psychiatric illness (e.g., schizophrenia) • Current alcohol/substance dependence • Current significant symptoms of PTSD based on total mean score >1.60 on IES-R • History of other neurological illness (e.g., Multiple Sclerosis, Parkinson's Disease) • Major medical conditions/treatments associated with possible cognitive dysfunction (e.g., Type II Diabetes, Lupus, Radiation treatment, Coronary Bypass Surgery, etc.) • Current involvement in litigation related to the injury
Phase 2: Collateral Interview	21	--	• No known history of mTBI • Known history of severe TBI

Table 2

Assessment protocol for the face-to-face session, completed by participants enrolled in the full study.

Neuropsychological Tests	Self-report Questionnaires
NAART	Demographics Questionnaire[†]
VSVT	NSI
PASAT	CES-D
Ruff	NPRS
COWAT (FAS & Animals)	STAI
CCC	HISDS (Past & Present)
U-PASAT[†]	CSE-TBII
	TBI-SIQ[†]

[†] Non-standardized measures.

NAART – North American Adult Reading Test (Blair & Spreen, 1989); VSVT – Victoria Symptom Validity Test (Slick, Hopp, Strauss, & Thompson, 2005); PASAT – Paced Auditory Serial Addition Test (Gronwall & Sampson, 1974); Ruff – Ruff 2 & 7 Selective Attention Test (Ruff & Allen, 1996); COWAT – Controlled Oral Word Association Test; CCC – Auditory Consonant Trigrams (Brown, 1958; Peterson & Peterson, 1959); U-PASAT – Un-Paced Auditory Serial Addition Test; NSI – Neurobehavioral Symptom Inventory (Cicerone & Kalmar, 1995); CES-D – Centre for Epidemiological Studies Depression Scale (Radloff, 1977); NPRS – Numerical Pain Rating Scale; STAI – State-Trait Anxiety Inventory (Spielberger, 1983); HISDS – Head Injury Semantic Differential Scale (Tyerman & Humphrey, 1984); CES-TBII – Collective Self-Esteem Scale for TBI – Identity Subscale (based on Luhtanen & Crocker, 1992); TBI-SIQ – TBI-Social Identity Questionnaire.

participating, were scheduled for a 30 minute telephone-based screening interview. All screening interviews were conducted by the author.

As part of the screening interview, the potential participants completed the IES-R to determine their current level of PTSD-like symptomatology. When the questionnaire was introduced, individuals were not asked to specify the nature of any traumatic event that they may have experienced and were told that it was not necessary that the trauma be related to the TBI – individuals were explicitly told that they could respond to the IES items based on the situation that led to the TBI or any other traumatic event that they may have experienced in their life and that they still think about.

Also during the screening interview, the name and contact information for a collateral informant (described to the participant as "...someone who knows you well and who could answer some questions about your injury") was obtained; the collateral interview was conducted at a later date. Based on the information obtained during these two interviews, eligibility for the next part of the study was determined. All participants were informed of their eligibility or ineligibility.

Eligible participants were invited to complete a face-to-face data collection session. They were informed that this session could take place at the University of Victoria campus or, if they were not able or it was not convenient to travel to campus, at another appropriate location that was more convenient for them. All but one participant came to campus for the face-to-face session. All subjects were tested individually, generally in a single 2-hour session (although one participant required the protocol to be broken up into two sessions). Regular rest breaks were given as necessary to avoid subject fatigue. At the start of the face-to-face session, the informed consent form was reviewed with each participant and time was given for them to then read over the form and ask any questions that they might have had. Once informed consent was provided, participants completed the protocol in a standard order[3] (see Table 3), and all but two participants completed the full protocol. Once data collection was complete, each participant was debriefed about the purpose of the study and given the opportunity to ask the examiner any questions that they may have had about the study.

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[3] The TBI-SIQ was added to the protocol after data collection had begun. Therefore, if the participant had already completed their face-to-face session, they were invited to complete this questionnaire over the phone, via email, or in person.

Table 3

Standard protocol for order of measure completion during face-to-face session.

Measures in order of completion
Background Demographics Questionnaire
Neurobehavioural Symptom Inventory (NSI)
State-Trait Anxiety Inventory – Trait (STAI-T)
North American Adult Reading Test (NAART)
Center for Epidemiological Studies Depression Scale (CES-D)
Victoria Symptom Validity Test (VSVT)
Numerical Pain Rating Scale (NPRS)
State-Trait Anxiety Inventory – State, Pre-PASAT (STAI-S-Pre)
Paced Auditory Serial Addition Test (PASAT)
State-Trait Anxiety Inventory – State, Post-PASAT (STAI-S-Post)
Ruff 2 & 7 Test of Selective Attention (Ruff)
Controlled Oral Word Association Test (COWAT)
- FAS
- Animals
Auditory Consonant Trigrams (CCC)
Head Injury Semantic Differential Scale – III (HISDS)
- Pre-injury (HISDS-Pre)
- Current / post-injury (HISDS-Post)
Un-Paced Auditory Serial Addition Test (U-PASAT)
Collective Self-Esteem Scale for TBI – Identity subscale (CSE-TBI-I)
TBI Social Identity Questionnaire (TBI-SIQ)[†]

[†] The TBI-SIQ was added to the protocol part way through data collection and it was not possible to obtain this data from all 21 participants. It was administered during the face-to-face session for 10 of the 15 participants who completed the measure; the 5 remaining participants completed the TBI-SIQ 1 to 4 months following their face-to-face session, and either in person, over the telephone, or via email.

Quality control. Data from the face-to-face sessions were collected by three undergraduate Psychology research assistants (RAs) from the University of Victoria in addition to the author. Therefore, a number of precautions and steps were taken in an attempt to ensure the quality and accuracy of the data through minimizing examiner-based variability. First, all of the chosen RAs were senior undergraduate students within the Department of Psychology who had work and/or volunteer experience with individuals with brain injuries or other neurological conditions. Second, all of the RAs underwent training – including didactics, demonstration, and hands-on practice – in the standardized administration of the measures included in the protocol. As part of the training procedure, the RAs observed the author complete the full protocol with a colleague who was familiar with the administration of the measures included in the protocol and who also had clinical experience working with individuals with mTBI, thus allowing for demonstration of potential circumstances that could arise and how to deal with them. Further, each RA was required to run through the full protocol with a "practice participant"; three undergraduate psychology students volunteered as the practice participants. Third, a script book was developed by the author, which included all of the tasks' instructions in order to ensure that the same information was provided to each participant. Finally, in order to ensure consistency in the scoring methods, the author scored all of the measures for all participants.

Results

Participant Characteristics

The total sample included 21 participants (female, *N* = 12; male, *N* = 9), between 20 and 65 years of age (*M* = 43.00, *SD* = 15.65), living in or around Victoria, BC. All participants reported that they had sustained a TBI, determined to be mild, with various underlying etiologies reported. Participants were, on average, just over five years post their most recent injury. More information regarding the sample's injury-related demographics can be found in Table 4, and information regarding injury etiology in Table 5. All participants were able to speak and read fluently in English and 85.71% of the sample reported that English was their first language. All participants denied any history of diagnosed learning disability (or significant learning difficulties), although two of the participants reported that they had been diagnosed with attention deficit/hyperactivity disorder (ADHD) – one as a child (pre injury) and one as an adult (post injury); it is not known who made these diagnoses or upon what information the diagnoses were based. The sample was relatively well-educated: all participants had graduated from high school and approximately 67% had completed two or more years of post-secondary education (*M* = 14.95 years of education, *SD* = 2.65). Performance on the NAART, a measure of premorbid functioning, was consistent with this: based on published normative data stratified by age and education (Strauss, Sherman, & Spreen, 2006), the mean *z*-score for the group was well within the average range (see Table 6).

Regarding employment history, one participant reported that they had been unemployed at the time of their injury; all other participants reported that they were working full-time or attending school at the time of their injury. However, post-injury (at the time of study participation), eight of the participants were either unemployed, on

Table 4

Injury-related demographic characteristics for the sample, including PCS severity as reported on the Neurobehavioural Symptom Inventory (N = 21).

	Mean	SD	Range	% of *N* per response
Number of mTBIs	2.81	2.20	1 – 9	38.10% - One 19.05% - Two 14.29% - Three 9.52% - Four 9.52% - Five 4.76% - Seven 4.76% - Nine
Time post mTBI (months)	64.86	73.40	13 – 336	23.81% - 12-18 mo 19.05% - 19-24 mo 14.29% - 25-48 mo 42.86% - > 48 mo
Estimated length of PTA[†] (minutes)	318.79	595.41	0 – 1620	14.29% - 0 min 19.05% - 1 min 23.81% - 5-10 min 19.05% - 11-60 min 23.81% - > 60 min
Neurobehavioural Symptom Inventory	Mean	SD	Range	
Total Symptoms (22 items)	21.76	15.43	2 – 61	
Physical Symptoms (6 items)	4.52	4.79	0 – 18	
Cognitive Symptoms (5 items)	6.93	4.59	0 – 19	
Affective Symptoms (4 items)	4.10	3.60	0 – 13	
Sensory Symptoms (2 items)	2.50	2.02	0 – 8	
Headache (1 item)	1.14	0.79	0 – 3	
Sleep (1 item)	1.29	1.31	0 – 4	

[†] Estimated length of PTA based on sum total of length of self-reported LoC, anterograde amnesia, and retrograde amnesia.

Table 5

Frequency of mTBI etiology reported by participants (most recent injury; N = 21).

Etiology	Frequency	(% of sample)
Sports-related	12	(57.14%)
Motor vehicle accident (MVA)	4	(19.05%)
Falls	3	(14.29%)
Work-related	1	(4.76%)
Other	1	(4.76%)

disability, or retired (see Table 7). Regarding the participants' living situation, as Table 8 illustrates, the participants' living status pre- and post-injury were relatively stable (with the maturation of a number of individuals who had, for example, lived with their parents prior to their injury but live alone or with a spouse at the time of participation in the study).

None of the participants had a history of severe psychiatric difficulties (e.g., no history in-patient psychiatric care); however, 10 individuals (47.62%) reported that they had been diagnosed with a mental health condition (e.g., depression or anxiety) at some point in their lives and 6 participants (28.57%) were taking medication for mood and/or anxiety at the time of their involvement in the study. At the time of data collection, the participants' self-reported levels of depression (CES-D) and anxiety (STAI-T) were not suggestive of clinically significant symptomatology, and symptoms of PTSD were all below the cut-off of 1.60 on the IES-R as per inclusion/exclusion criteria (see Table 6). Ten of the participants (47.62%) reported no current use of alcohol and only two participants (9.52%) reported consuming more than 7 drinks per week (*M* = 2.90 drinks

Table 6

Descriptive statistics for all questionnaires and cognitive measures.

	N	Mean	(SD)	Skew	Actual Range	*(Possible Range)*
Questionnaires						
NSI Total	21	21.76	(15.43)	1.29	2.00 – 61.00	*(0 – 88)*
IES-R Total	21	0.84	(0.36)	0.18	0.18 – 1.55	*(0 – 4)*
CES-D	21	13.93	(9.07)	1.39	3.00 – 37.00	*(0 – 60)*
STAI-Trait [†]	21	56.67	(14.12)	1.44	36.00 – 98.00	
STAI-State-Pre [†]	21	49.43	(12.17)	1.80	37.00 – 89.00	
STAI-State-Post [†]	21	55.95	(12.94)	0.98	38.00 – 92.00	
STAI-S-Diff[††]	21	6.52	(8.04)	-0.45	-8.00 – 19.00	
HISDS-Pre	21	5.30	(0.92)	-0.40	3.25 – 6.70	*(1 – 7)*
HISDS-Post	21	4.98	(0.80)	0.55	3.65 – 6.75	*(1 – 7)*
HISDS-Diff	21	-0.33	(1.25)	0.36	-2.45 – 2.10	*(-6 – 6)*
CSE-TBI-I	21	14.05	(6.39)	-0.03	4.00 – 24.00	*(4 – 28)*
TBI-SIQ	15	12.60	(8.03)	2.11	6.00 – 34.00	*(0 - 40)*
Cognitive Measures						
NAART [‡]	21	0.15	(0.70)	-0.30	-1.34 – 1.26	
PASAT [‡]	20	-0.60	(0.95)	-0.03	-2.61 – 0.84	
Ruff – Speed [†]	20	49.75	(7.66)	0.58	37.00 – 68.00	
Ruff – Accuracy [†]	20	47.05	(9.95)	-0.86	24.00 – 59.00	
COWAT – FAS [‡]	21	0.20	(1.21)	0.76	-1.36 – 2.97	
COWAT – Animals [‡]	21	0.18	(0.88)	-0.53	-1.83 – 1.50	
CCC Total	20	45.50	(8.18)	-0.21	30.00 – 58.00	*(0 – 60)*
CCC – 9" [‡]	20	-0.07	(1.18)	-1.01	-3.14 – 1.19	
CCC – 18" [‡]	20	-0.26	(1.18)	-0.52	-2.94 – 1.53	
CCC – 36" [‡]	20	-0.15	(1.30)	0.02	-2.00 – 1.87	
U-PASAT	19	24.63	(0.96)	-3.41	21.00 – 25.00	*(0 – 25)*

[†] T-scores: mean = 50, standard deviation = 10; [††] (STAI-State-Post) – (STAI-State-Pre), T-scores of both used in calculation; [‡] *z*-scores: mean = 0, standard deviation = 1.00.

Questionnaires: NSI - Neurobehavioural Symptom Inventory; IES-R - Impact of Event Scale – Revised; CES-D - Centre for Epidemological Studies – Depression Scale; STAI - State Trait Anxiety Inventory; HISDS-III - Head Injury Semantic Differential Scale – III; CSE-TBI-I - Collective Self-Esteem for TBI – Identity Subscale; NPRS - Numerical Pain Rating Scale.

Cognitive Measures: NAART - North American Adult Reading Test; PASAT - Paced Auditory Serial Addition Test; Ruff - Ruff 2 & 7 Selective Attention Test; COWAT - Controlled Oral Word Association Test; CCC - Auditory Consonant Trigrams; U-PASAT - Un-Paced Auditory Serial Addition Test.

Table 7

Frequency (%) for levels of employment status at the time of the injury (i.e., pre-injury) and currently (post-injury) (N = 21).

	Full-time work	Part-time work	Student	Unemployed	Disability
Pre-injury	14 (66.67%)	0 (0.00%)	6 (28.57%)	1 (4.76%)	0 (0.00%)
Post-injury	9 (42.86%)	0 (0.00%)	4 (19.05%)	4 (19.05%)	3 (14.29%)

Table 8

Frequency (%) of participants' reported living arrangements at the time of injury (i.e., pre-injury) and currently (i.e., post-injury) (N = 21).

	Alone	Spouse	Parent	Other family	Friend/ Roommate	Other
Pre-injury	3 (14.29%)	9 (42.86%)	3 (14.29%)	2 (9.52%)	3 (14.29%)	1 (4.76%)
Post-injury	3 (14.29%)	13 (61.90%)	1 (4.76%)	2 (9.52%)	2 (9.52%)	0 (0%)

per week, *SD* = 4.89); three of the participants indicated that that they had had a problem with alcohol use in the past, but all three indicated that they no longer had a problem (and did not report significant alcohol use currently). Sixteen of the participants (76.19%) denied current use of illicit drugs, with the other five participants reporting some current use of marijuana.

An additional 35 individuals (15 female, 20 male) completed some or all of the telephone screening interview but were found to be ineligible for participation. Reasons for exclusion[4] included: current age older than 65 years (*n* = 3); current involvement in litigation related to their injury (*n* = 11); history of a severe TBI, based on a hospital admission of greater than 24 hours (*n* = 8) and/or loss of consciousness lasting longer than 30 minutes (*n* = 7); history of a non-traumatic, acquired brain injury (e.g., stroke) (*n* = 4); a significant health condition that could have an impact on the health and functioning of their brain (e.g., Multiple Sclerosis, Type II Diabetes) (*n* = 9); and clinically notable symptoms of post-traumatic stress disorder, based on a mean score greater than the cut-off of 1.60 on the IES-R (*n* = 14). All individuals who participated in the full study scored within the expected range on the Victoria Symptom Validity Test with no indication of insufficient effort. Therefore, no participants had to be excluded from analyses due to issues related to effort or validity.

Post-Concussion Symptoms (PCS)

In this study, PCS was measured using the Neurobehavioural Symptom Inventory (NSI). Table 6 (above) reports the samples' mean NSI score. The frequency with which the various items on the NSI were reported to be a problem (i.e., the number of participants who rated each item as at least a 1, or a Mild Disturbance, on the 0 to 5 scale), as well as the mean, median, and mode of the ratings are available in Table 9.

Regarding the influence of demographic characteristics on the report of PCS, there was no significant difference in NSI scores reported by males (*n* = 9, *M* = 17.22, *SD* = 9.31) and females (*n* = 12, *M* = 25.17, *SD* = 18.44), *SE* = 6.16, *t*(19) = 1.18, *p* = .25, and no significant correlations between NSI scores and the participants' current age (*r* = .27) ,

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[4] Reasons for exclusion were not mutually exclusive; some individuals met more than one of the exclusion criteria.

Table 9

Frequency and severity (range, mean, median, and mode) of post-concussion symptoms, as reported on NSI items (N = 21).

Item	Problem Freq.	Range	Mean (SD)	Mdn	Mode
Feeling Dizzy.	12	0 – 2	0.71 (0.72)	1	0, 1
Loss of balance.	10	0 – 4	0.81 (1.12)	0	0
Poor coordination, clumsy.	11	0 – 3	0.90 (1.04)	1	0
Headaches.	17	0 – 3	1.14 (0.79)	1	1
Nausea.	6	0 – 3	0.48 (0.87)	0	0
Vision problems, blurring, trouble seeing.	10	0 – 4	0.90 (1.18)	0	0
Sensitivity to light.	13	0 – 4	1.36 (1.33)	1	0
Hearing difficulty.	10	0 – 2	0.52 (0.60)	0	0
Sensitivity to noise.	16	0 – 4	1.14 (1.06)	1	1
Numbness or tingling on parts of my body.	9	0 – 4	0.62 (0.97)	0	0
Change in taste and/or smell.	3	0 – 1	0.14 (0.36)	0	0
Loss of appetite or increased appetite.	8	0 – 3	0.71 (1.06)	0	0
Poor concentration, can't pay attention, easily distracted.	17	0 – 4	1.62 (1.20)	2	2
Forgetfulness, can't remember things.	18	0 – 4	1.48 (0.98)	1	1, 2
Difficulty making decisions.	13	0 – 4	1.07 (1.23)	1	0, 1
Slowed thinking, difficulty getting organized, can't finish things.	17	0 – 4	1.43 (1.12)	1	1
Fatigue, loss of energy, getting tired easily.	15	0 – 4	1.33 (1.20)	1	1
Difficulty falling or staying asleep.	14	0 – 4	1.29 (1.31)	1	0, 1
Feeling anxious or tense.	13	0 – 4	0.90 (1.00)	1	1
Feeling depressed or sad.	12	0 – 4	0.95 (1.12)	1	0
Irritability, easily annoyed.	12	0 – 4	1.00 (1.14)	1	0
Poor frustration tolerance, feeling easily overwhelmed by things.	14	0 – 4	1.45 (1.22)	1	0, 1

Problem Frequency calculated as the number of participants who reported experiencing the symptom (rated as 1 or greater); all items rated from 0 (not a problem) to 4 (very severe problem).

years of education (*r* < .01), time post-injury (*r* = .18), or self-reported PTA (*r* = -.21; all *p*s > .05). Also, no there was no significant difference in symptom severity for individuals who sustained one mTBI (*n* = 8; *M* = 20.88, *SD* = 18.36) compared to those who had sustained two or more mTBIs (*n* = 13; *M* = 22.31, *SD* = 14.11), *SE* = 7.58, *t*(19) = -0.20, *p* = .84.

Because of the small sample size, it was not possible to investigate NSI scores as a factor of injury etiology as it was initially defined (i.e., MVA, fall, sports-related, work-related, or other/assault); however, it was possible to create near-even dichotomous groups by comparing those with sports-related injuries (*n* = 12) to the group of those who sustained their injuries through all other etiologies (i.e., non-sport related, *n* = 9). It was found that those with sports-related injury reported significantly fewer symptoms (*M* = 14.42, *SD* = 10.08) than those with non-sports related injury (*M* = 31.56, SD = 16.33), *SE* = 6.17, *t*(19) = -2.97, *p* = .01, *d* = 1.26.

Cognitive Functioning

The specific cognitive domains assessed were: *Processing Speed*, measured with the Ruff 2 & 7 Total Speed composite (Ruff-Speed); *Attention*, measured with the Ruff 2 & 7 Total Accuracy composite (Ruff-Acc); *Working Memory,* measured with the Auditory Consonant Trigrams (CCC) Total score; *Phonemic Fluency*, measured by the “FAS” component of the Controlled Oral Word Association Test (COWAT); and *Semantic Fluency*, measured by the “Animals” component of the COWAT (See Table 6 above for descriptive statistics for the groups' performances on these measures). With a Bonferroni adjustment for multiple analyses, none of the measures of cognitive functioning were significantly correlated with any demographic variables, although there were trends in the relations between Ruff-Accuracy (i.e., attention) and education, CCC-Total (i.e., working memory) and education, COWAT-FAS (i.e., phonemic fluency) and length of PTA reported, and COWAT-Animals (i.e., semantic fluency) and age (See Table 10).

Table 11 reports the results of *t*-tests comparing cognitive performance across the domains assessed based on gender, number of mTBIs reported, and injury etiology. The only significant difference was in semantic verbal fluency (i.e., COWAT-Animals), where females scored significantly higher than males.

In addition to the individual scores, the degree of *cognitive impairment* within the sample was investigated based on the standardized scores (either *z*-scores or T-scores) obtained across all of the completed cognitive measures[5]. Because of a high number of missing data points (i.e., five of the nine scores missing), cognitive impairment could not be investigated in this way for one participant. Therefore, of the 20 included participants, 14 (70%) had at least one score that was one standard deviation or more below the normative mean (i.e., a *z*-score of -1.00 or less and/or a T-score of 40 or less) but, as Table 12 illustrates, the sample performed within the normal range on most of the cognitive measures.

A *weighted cognitive impairment score* was then created for each of the 20 participants with sufficient data such that lower scores (i.e., scores that were more impaired or more standard deviations below the normative mean) were weighted more heavily. The number of standard scores within the specific ranges were tallied and the number of scores between 1.0 and 1.49 standard deviations below the mean were given a weight of one; the number of scores between 1.50 and 1.99 standard deviations were given a weight of two; between 2.00 and 2.99 standard deviations a weight of three; and the number of scores more than 3.00 standard deviations below the mean were given a

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[5] This investigation included scores/measures not included in the main analyses. Scores from the NAART, PASAT, Ruff-Speed, Ruff-Accuracy, COWAT-FAS, COWAT-Category, CCC-9sec, CCC-18sec, and CCC-36sc were included.

Table 10

Pearson correlations between cognitive functioning measures and demographic variables (N = 21).

	Processing Speed Ruff-Speed†	*Attention* Ruff-Accuracy†	*Working Memory* CCC-Total†	*Phonemic Fluency* FAS	*Semantic Fluency* Animals
Age	*r* = .05 (*p* = .84)	*r* = .19 (*p* = .42)	*r* = .03 (*p* = .91)	*r* = -.05 (*p* = .83)	***r* = -.45** **(*p* = .04)**
Education	*r* = -.22 (*p* = .36)	***r* = .44** **(*p* = .06)**	***r* = .46** **(*p* = .04)**	*r* < .01 (*p* = .99)	*r* = .01 (*p* = .95)
Time post injury	*r* = .13 (*p* = .59)	*r* = -.17 (*p* = .47)	*r* = .18 (*p* = .44)	*r* = -.16 (*p* = .50)	*r* = -.10 (*p* = .65)
PTA	*r* = -.14 (*p* = .55)	*r* = -.12 (*p* = .61)	*r* = -.35 (*p* = .13)	***r* = -.47** **(*p* = .03)**	*r* = -.04 (*p* = .86)

† *N* = 20 for the measures indicated.
Bold = Trend suggested in relation between variables (based on Bonferroni correction).

weight of four. Weighted cognitive impairment scores ranged from 0 to 16, with an observed mean score of 2.95 (*SD* = 3.75) and a median score of 3.00.

Pearson correlations calculated between the weighted impairment scores and demographic variables indicated no significant associations with age (*r* = -0.32, *p* = .16), education (*r* = -0.33, *p* = .16), and self-reported PTA (*r* = 0.39, *p* = .09). There was also no significant difference in weighted cognitive impairment scores based on gender (*SE* = 1.61, *t*(18) = 0.24, *p* = .82), number of mTBIs (*SE* = 2.15, *t*(18) = 1.72, *p* = .10), or etiology (*SE* = 2.00, *t*(18) = -0.36, *p* = .72).

Hypothesis #1: cognitive functioning and PCS. It was predicted that participants' cognitive functioning would predict PCS severity as measured by the NSI. First, in order to investigate this hypothesis, a Multiple Regression with Processing Speed (Ruff-Speed), Attention (Ruff-Accuracy), Working Memory (CCC – Total score),

Table 11

Results of t-tests investigating cognitive performance based on gender, number of mTBIs, and injury etiology (N = 21).

	Gender		*Number of mTBIs*		*Etiology*	
	Male	Female	One	Two+	Sports	Non-sports
Processing Speed: Ruff-Speed†	49.89 (6.97)	49.64 (8.51)	44.43 (7.21)	52.62 (6.44)	50.58 (9.02)	48.50 (5.32)
	t(18) = -0.07, *p* = .94		*t*(18) = -2.61, *p* = .02		*t*(18) = 0.59, *p* = .57	
Attention: Ruff-Acc.†	45.44 (9.11)	48.36 (10.84)	43.43 (13.00)	49.00 (7.78)	46.25 (9.37)	48.25 (11.31)
	t(18) = 0.64, *p* = .53		*t*(8.38)‡ = -1.04, *p* = .33		*t*(18) = -0.43, *p* = .67	
Working Memory: CCC-Total	46.33 (8.32)	44.82 (8.32)	44.71 (10.55)	45.92 (7.05)	46.50 (7.89)	44.00 (8.91)
	t(18) = -0.40, *p* = .69		*t*(18) = -0.31, *p* = .76		*t*(18) = 0.66, *p* = .52	
Phonemic Fluency: FAS	-0.06 (1.25)	0.40 (1.19)	-0.06 (0.98)	0.37 (1.34)	0.36 (1.23)	-0.01 (1.22)
	t(19) = 0.87, *p* = .40		*t*(19) = -0.78, *p* = .45		*t*(19) = 0.68, *p* = .51	
Semantic Fluency: Animals	**-0.34 (0.92)**	**0.58 (0.64)**	0.16 (1.12)	0.20 (0.76)	0.11 (1.07)	0.28 (0.59)
	***t*(19) = 2.72, *p* = .01**		*t*(19) = -0.09, *p* = .93		*t*(17.74)‡ = -0.48, *p* = .64	

† *N* = 20 for the measures indicated

‡ Levene's test for equality of variances not met; adjusted *df* reported

Bolded values are significant.

Table 12

Frequency (%) of participants with scores on the cognitive measures within different levels of impairment (N = 20).

	Number of scores within range of impairment			
Level of impairment	Zero	One	Two	Three
1.00 – 1.49 SD below mean	6 (30%)	7 (35%)	6 (30%)	1 (5%)
1.50 – 1.99 SD below mean	13 (65%)	5 (25%)	2 (10%)	--
2.00 – 2.99 SD below mean	16 (80%)	3 (15%)	1 (5%)	--
> 3.00 SD below mean	19 (95%)	1 (5%)	--	--

Phonemic Fluency (FAS), and Semantic Fluency (Animals) regressed on NSI scores was completed. These five variables only accounted for 3% of the total variability ($R^2 = 0.03$, $F_{5,\,19} = 0.09$, $p = .99$). None of the cognitive measures were significant predictors of PCS severity (all ps > .50). Second, NSI scores were regressed on the weighted cognitive impairment scores. Similarly, weighted cognitive impairment was not a significant predictor of PCS severity and it accounted for almost none of the variability in NSI scores ($R^2 < .01$, $F_{1,\,18} < 0.01$, $p = .99$).

These findings, indicating that cognitive functioning does not predict PCS severity, may be due, in part, to the breadth of symptoms assessed by the NSI. Therefore, in order to see if cognitive functioning predicted only the report of *cognitive* symptoms, first the scores from each of the cognitive measures were regressed on the Cognitive subscale of the NSI. Interestingly, the five variables together still accounted for only 4% of the total variability in the NSI Cognitive subscale ($R^2 = 0.04$, $F_{5,\,14} = 0.13$, $p = .98$) and none of the cognitive variables were significant predictors of self-reported cognitive symptoms (all *p*s > .50). The scores on the Cognitive subscale of the NSI were then regressed on the weighted cognitive impairment scores. Again, weighted cognitive impairment did not predict the severity of reported cognitive symptoms ($R^2 < 0.01$ $F_{1,18} = 0.03$, $MS_e = 17.50$, $p = .86$).

Anxiety Susceptibility

Anxiety susceptibility, or one's experience of increased anxiety in the context of a cognitively demanding situation, was assessed using the State Trait Anxiety Inventory – State version (STAI-S) administered immediately before (STAI-S-Pre) and after (STAI-S-Post) the completion of the Paced Auditory Serial Addition Test (PASAT)[6]. Anxiety susceptibility was operationally defined as the difference between the standard scores for the STAI-S-Post and STAI-S-Pre (i.e., STAI-S-Diff), such that higher difference scores are indicative of higher levels of anxiety susceptibility (i.e., higher self-reported symptoms of anxiety *after* the PASAT). The average pre-post difference (STAI-S-Diff) was 6.52 (*SD* = 8.04) and ranged from -8 to 19. There were no significant correlations between anxiety sensitivity and education ($r = -.02$), time post injury ($r = .31$), or length of PTA ($r = .12$, all *p*s > .10). Also, *t*-tests indicated no significant differences in anxiety susceptibility reported by males ($M = 6.67$, $SD = 7.89$) versus females ($M = 6.42$, $SD = 8.50$), $SE = 3.60$, $t(19) = -0.07$, $p = .95$; those with one mTBI ($M = 8.50$, $SD = 7.54$) versus those with two or more mTBIs ($M = 5.31$, $SD = 8.39$), $SE = 3.54$, $t(19) = 0.88$, $p = .39$; nor sports related injuries ($M = 6.83$, $SD = 9.02$) versus non-sports related injuries ($M = 6.11$, $SD = 7.03$), $SE = 3.50$, $t(19) = 0.20$, $p = .84$. Further, there was also no relation between participants' performance on the PASAT and their reported change in anxiety from before to after ($r = -.22$, $p = .34$).

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[6] One participant was unable to complete the PASAT as they were not able to comprehend the task or adequately complete the practice items; therefore, this individual completed the STAI-S before and after multiple attempts at the PASAT practice items.

Hypothesis #2: anxiety susceptibility and PCS. It was hypothesized that one's experience of increased anxiety in the context of a cognitively demanding situation, or anxiety susceptibility, would predict PCS severity. In order to investigate this hypothesis, the STAI-S-Diff scores were regressed on NSI total scores. The STAI-S difference scores only accounted for 1% of the total variability and did not predict NSI scores ($R^2 = 0.01$, $F_{1, 20} = 0.22$, $p = .66$). Again, due to the symptom breadth of the NSI, the same analysis was carried out using the Cognitive subscale of the NSI. The STAI-S difference scores accounted for none of the total variability and also did not predict scores on the NSI Cognitive subscale ($R^2 < 0.01$, $F_{1, 20} = 0.06$, $p = .82$). Therefore, anxiety susceptibility in a cognitively demanding situation was not a significant predictor of overall PCS severity, nor of the severity of self-reported cognitive PCS.

It is possible that there may be a differential effect of education on the relationship between anxiety sensitivity and PCS severity. For example, individuals with higher levels of educational attainment may experience more anxiety in the context of perceived cognitive difficulties. In order to investigate the role of education as a covariate, a sequential multiple regression analysis was conducted with education being entered into the model with NSI scores first, and then the STAI-S-Difference scores added to the model second. Including education as a covariate did not significantly improve the ability of anxiety susceptibility to predict PCS ($\Delta R^2 = .01$, $F_{1, 18} = 0.19$, $p = .67$).

Given that anxiety susceptibility did not have an effect on the severity of PCS within this sample, an exploratory comparison of *trait* anxiety (measured using the STAI-T) and anxiety susceptibility (STAI-S-Diff) in the prediction of PCS severity was conducted using multiple regression analyses. Together, these variables explained 57% of the total variability ($R^2 = 0.57$, $F_{2,18} = 11.98$, $p < .01$); however, only trait anxiety was a significant predictor of PCS severity ($\beta = 0.81$, $p < .01$; see Figure 2). In order to determine if the large association between PCS and trait anxiety was due to the fact that affective symptoms (e.g., depression and anxiety) can, themselves, be considered post-concussion symptoms and so are included in the NSI, two additional exploratory analyses were conducted. First, STAI-T scores were regressed on the scores from the Cognitive subscale of the NSI. The STAI-T was a significant predictor of NSI Cognitive subscale scores, accounting for 49% of the explained variability ($R^2 = 0.49$, $F_{1,19} = 18.42$, $p < .01$). Second, a non-affective NSI score was created by subtracting NSI Affective subscale scores from the NSI Total score; the STAI-T was then regressed on these non-affective NSI scores. The results indicated that the STAI-T was also a significant predictor of non-affective NSI scores and that it accounted accounted for 44% of the total variability ($R^2 = 0.44$, $F_{1,19} = 15.00$, $p < .01$).

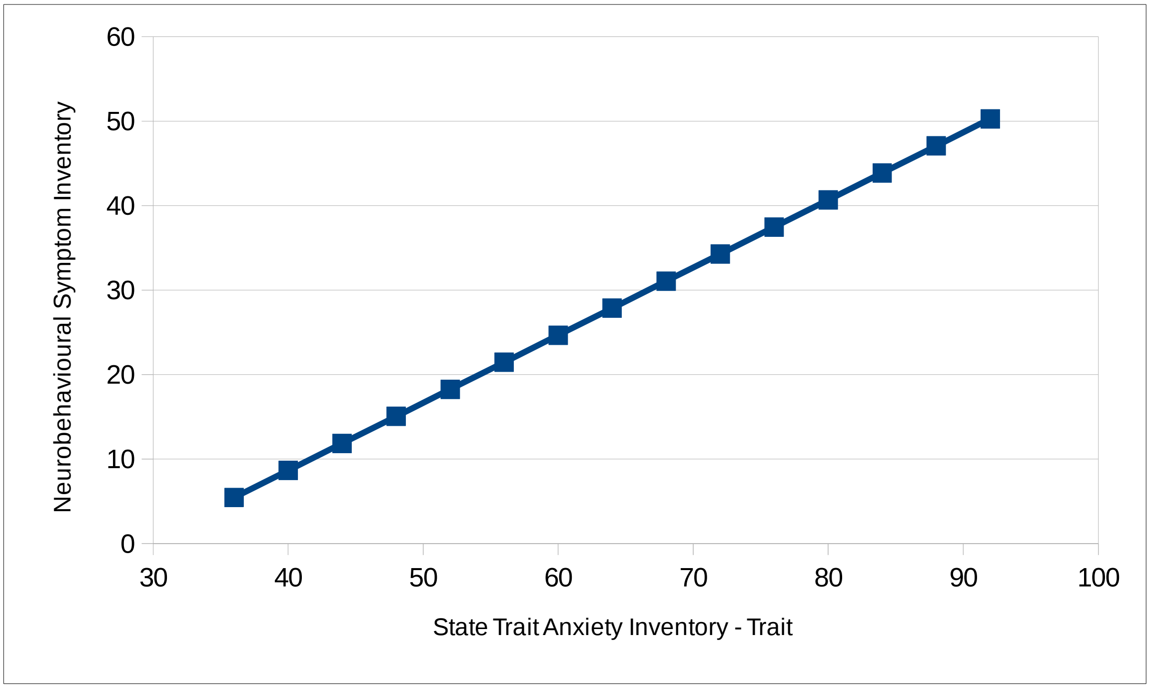

Figure 2. Regression line for prediction of post-concussion symptoms (Neurobehavioural Symptom Inventory, NSI) based on trait anxiety (State-Trait Anxiety Inventory-Trait, STAI-T). Pearson $r = .73$

Hypothesis #3: anxiety susceptibility, cognitive functioning, and PCS. It was further proposed that anxiety susceptibility would moderate the relationship between cognitive functioning and PCS severity. In order to investigate this, an interaction term was created between STAI-S-Diff scores and the weighted cognitive impairment scores (with both variables centered prior to creating the interaction term). Weighted cognitive impairment, STAI-S-Difference scores, and the interaction were then regressed on NSI scores. The model was not significant ($F_{3, 15} = 0.25$, $p = 0.86$, $MS_e = 181.96$, $R^2 = 0.05$).

Identity

Informed by the social identity approach, multiple components of identity were investigated in the current study: self-perception was measured using the Head Injury Semantic Differential Scale (HISDS), TBI-related self-concept was measured using the Collective Self-Esteem Scale for TBI – Identity subscale (CSE-TBI-I), and TBI-related social identity was measured using the TBI – Social Identity Questionnaire[7] (TBI-SIQ).

On the HISDS, participants rated their self-perception based on various attributes. Ratings were provided for both their pre-injury (HISDS-pre) and current, post-injury, (HISDS-post) self-perception. Ratings were converted to a 1 to 7 scale, with the final score representing the average of the ratings. The difference in the participants' self-perception from pre- to post-injury was also calculated (HISDS-diff = HISDS-post – HISDS-pre) with higher scores representing a more positive current self-perception (see Table 6, above, for descriptive statistics). A paired samples *t*-test indicated that there was no significant difference between the pre-injury and post-injury self-perception ratings ($SE = 0.27$, $t(20) = 1.19$, $p = .25$) nor a significant correlation between the ratings of pre- and post-injury self-perception ($r = -.06$, $p = .81$).

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[7] The TBI-SIQ was added to the protocol part way through data collection. Therefore, despite numerous requests, six participants did not complete this measure. The other measures (HISDS and CSE-TBI-I) were completed by the full sample.

Bivariate correlations between the each of the measures of self-perception (HISDS-pre, -post, and -diff) with age, years of education, time post-injury, and PTA were not significant (see Table 13). Multiple *t*-tests also revealed no significant differences between self-perception based on gender, number of mTBIs, and etiology (see Tables 14, 15, and 16, respectively).

Next, participants rated their agreement with statements regarding the importance of the TBI to their self-concept on the CSE-TBI-I (see Table 6, above, for mean and SD). There were no significant correlations between scores on the CSE-TBI-I and age (*r* = .20), years of education (*r* = .17), time post-injury (*r* = .39), or length of PTA (*r* = .20). Additionally, multiple *t*-tests revealed no differences in CSE-TBI-I scores for males (*M* = 14.56, *SD* = 6.73) versus females (*M* = 13.67, *SD* = 6.40), *SE* = 2.91, *t*(19) = -0.31, *p* = .76; single mTBI (*M* = 13.13, *SD* = 6.96) versus multiple mTBIs (*M* = 14.62, *SD* = 6.24), *SE* = 3.01, *t*(19) = -0.51, *p* = .62; nor sports related injury (*M* = 12.92, *SD* = 6.60) versus non-sports related injury (*M* = 15.56, *SD* = 6.15), *SE* = 2.80, *t*(19) = -0.93, *p* = .36.

Table 13

Pearson correlations between reported self-perception (pre-injury, post-injury, and pre-post injury difference) and demographic variables.

	Demographics			
	Age	Education (Y)	Time post (mo)	PTA
HISDS-pre	*r* = .21 (*p* = .35)	*r* = .15 (*p* = .51)	*r* = -.09 (*p* = .69)	r < .01 (*p* > .99)
HISDS-post	*r* = -.33 (*p* = .15)	*r* = .18 (*p* = .43)	*r* = -.23 (*p* = .31)	*r* = .02 (*p* = .94)
HISDS-diff	*r* = -.37 (*p* = .10)	*r* < .01 (*p* = .99)	*r* = -.08 (*p* = .73)	*r* = .01 (*p* = .96)

HISDS – Head Injury Semantic Differential Scale

Table 14

Results of t-tests for HISDS scores by gender (N = 21).

	Gender		
	Males *M* (SD)	Females *M* (SD)	*t*-test output
HISDS-Pre	5.54 (0.58)	5.13 (1.11)	*SE* = .37, *t*(17.28)‡ = -1.10, *p* = .29
HISDS-Post	5.14 (0.86)	4.86 (0.77)	*SE* = .36, *t*(19) = -0.79, *p* = .44
HISDS-Difference	-0.40 (0.77)	-0.27 (1.56)	*SE* = .52, *t*(16.83)‡ = 0.25, *p* = .81

‡ Levene's test for equality of variances not met (*p* < .05); adjusted *df* reported.

Table 15

Results of t-tests for HISDS scores by number of mTBIs reported (N = 21).

	Number of mTBIs		
	1 mTBI *M* (SD)	2+ mTBIs *M* (SD)	*t*-test output
HISDS-Pre	5.10 (1.02)	5.43 (0.88)	*SE* = .44, *t*(19) = -0.79, *p* = .44
HISDS-Post	4.77 (0.61)	5.10 (0.89)	*SE* = .33, *t*(19) = -0.91, *p* = .37
HISDS-Difference	-0.33 (1.54)	-0.33 (1.11)	*SE* = .63, *t*(19) < 0.01, *p* > .99

Table 16

Results of t-tests for HISHS scores by mTBI etiology (N = 21).

	Etiology		
	Sports related *M* (SD)	Non-sports related *M* (SD)	*t*-test output
HISDS-Pre	5.33 (0.71)	5.28 (1.20)	*SE* = .45, *t*(12.15)‡ = 0.11, *p* = .92
HISDS-Post	5.20 (0.86)	4.68 (0.63)	*SE* = .33, *t*(19) = 1.55, *p* = .14
HISDS-Difference	-0.12 (1.12)	-0.60 (1.44)	*SE* = .58, *t*(19) = 0.86, *p* = .40

‡ Levene's test for equality of variances not met (p < .05); adjusted df reported

Finally, on the TBI-SIQ, participants (*n* =15) rated aspects of their TBI-related social identity based on their affiliation with brain injury related groups (Table 17), their sense of belonging to the wider group of TBI survivors (Table 18), and their self-categorization as a person with a brain injury (Table 19). All but four of the 15 participants reported some degree of affiliation with brain injury services or other individuals with brain injury, 10 of the participants reported at least a little sense of belonging within the community of brain injury survivors, and all 15 participants reported some degree of self-categorization as a person with a brain injury (although, notably, mTBI was not reported as one of their current groups/roles regarded as most important to who they are). See Table 6 for the samples' mean and standard deviation for the TBI-SIQ total score.

No significant correlations were found between the TBI-SIQ and any of the demographic variables, including: age (r = -.07), education (r = -.17), time post injury (r = -.25), and PTA (r = .07). Further, there was no significant difference in the TBI-SIQ scores for males (n = 6, M = 9.17, SD = 2.64) versus females (n = 9, M = 14.89, SD = 9.69), SE = 3.40, $t(13)$ = 1.40, p = .19; those who sustained sports related injuries (n = 8, M = 9.50, SD = 2.56) compared to those with non-sports related injuries (n = 7, M = 16.14, SD = 10.75), SE = 4.16, $t(6.60)$ = -1.60, p = .16; nor those with a single (n = 5, M = 8.80, SD = 2.28) versus multiple mTBIs (n = 10, M = 14.50, SD = 9.28), SE = 3.11, $t(13)$ = -1.33, p = .21.

In order to determine if these measures (namely, the HISDS, CSE-TBI-I, and TBI-SIQ) were actually assessing divergent aspects of identity, the Pearson correlations between the scores were investigated. The correlation matrix can be found in Table 20. None of the relevant correlations (i.e., ignoring the correlations between HISDS scores)

Table 17

Frequencies of participants' responses to TBI-SIQ items regarding affiliation with the brain injury community (N = 15).

Type of affiliation	Frequency	% per Duration of involvement
Have you ever been involved in a *group* for individuals with brain injury?	Yes = 2	50% - 6-12 mos. 50% - > 12 mos.
	No = 13	
Have you ever attended a *formal rehabilitation program* related to your brain injury?	Yes = 6	17% - 1-3 mos. 50% - 3-6 mos. 17% - 6-12 mos. 17% - >12 mos.
	No = 9	
Have you ever been involved in any *other* type of group/program for people with brain injury?	Yes = 3	100% - 6-12 mos.
	No = 12	
Have you attended a brain injury focused group/program in the past *3 months*?	Yes = 2	50% - 1 – 9 times 50% - 30+ times
	No = 13	
How many of your friends and/or family members have had a brain injury?	None = 5 1 person = 5 2-3 people = 2 6+ people = 3	

Table 18

Frequencies of participants' responses to TBI-SIQ items regarding sense of belonging in the brain injury community (N = 15).

Question	Response	Freq. (%)
Do you feel that you belong to the larger community of "brain injury survivors"?	Not at all	7 (47%)
	A little	6 (40%)
	Somewhat	--
	Quite a bit	2 (13%)
	Extremely	--
How important is this feeling of "belonging" to you currently?	Not at all	7 (47%)
	A little	4 (27%)
	Somewhat	1 (7%)
	Quite a bit	--
	Extremely	2 (13%)
	Missing	*1 (7%)*

Table 19

Frequencies of participants' responses to TBI-SIQ brain injury related identity items (N = 15).

Question	Response	Freq. (%)
How comfortable are you sharing with others that you have had a *concussion*?	Not at all	--
	A little	--
	Somewhat	2 (13%)
	Quite a bit	7 (47%)
	Extremely	6 (40%)
How comfortable are you sharing with others that you have had a *brain injury*?	Not at all	1 (7%)
	A little	--
	Somewhat	2 (13%)
	Quite a bit	7 (47%)
	Extremely	5 (33%)
Do you think that others categorize – or “see” - you as a person with a brain injury?	Not at all	11 (73%)
	A little	1 (7%)
	Somewhat	2 (13%)
	Quite a bit	--
	Extremely	--
	(Missing)	*1 (7%)*
Do you find it difficult to socialize with people who have never had a brain injury?	Not at all	11 (73%)
	A little	1 (7%)
	Somewhat	2 (13%)
	Quite a bit	1 (7%)
	Extremely	--
Which of your current groups/roles is the most important to who you are currently?	TBI-related	--
	Not TBI-related	11 (73%)
	(Missing)	*4 (27%)*

Table 20

Correlation matrix for Pearson correlations between all identity measures (N = 21)[†].

	HISDS-pre	HISDS-post	HISDS-diff	CSE-TBI-I	TBI-SIQ[†]
HISDS-pre	$r = 1.00$ --	$r = -.06$ $p = .81$	$r = .77$ $p < .01$	$r = -.05$ $p = .83$	$r = -.35$ $p = .20$
HISDS-post		$r = 1.00$ --	$r = -.68$ $p < .01$	$r = -.30$ $p = .18$	$r = -.38$ $p = .17$
HISDS-diff			$r = 1.00$ --	$r = -.16$ $p = .50$	$r = .04$ $p = .88$
CSE-TBI-I				$r = 1.00$ --	$r = .04$ $p = .89$
TBI-SIQ					$r = 1.00$ --

[†] All correlations with TBI-SIQ are based on $N = 15$

were significant. As such, the measures seem to be assessing different aspects of the participants' identity: namely, self-perception, TBI-related self-concept, and TBI-related social identity.

Hypothesis #4: identity and PCS. It was hypothesized that identity would predict self-reported PCS severity. The role of self-perception was investigated first. Both the HISDS-pre and HISDS-post were regressed on NSI scores. Together, these variables accounted for 40% of the total variability in NSI scores ($R^2 = 0.40$, $F_{2, 18} = 5.92$, $p = .01$). Interestingly, only *post*-injury self-perception was a significant predictor of PCS severity ($\beta = 1.08$, $p < .01$; see Figure 3). Next, the role of TBI-related self-concept was investigated, with scores for the CSE-TBI-I regressed on NSI scores. The CSE-TBI-I accounted for 7% of the total variability in NSI scores, and was not a significant predictor of PCS severity ($R^2 = 0.07$, $F_{1, 19} = 1.35$, $\beta = 0.62$, $p = .26$). Finally, TBI-related social

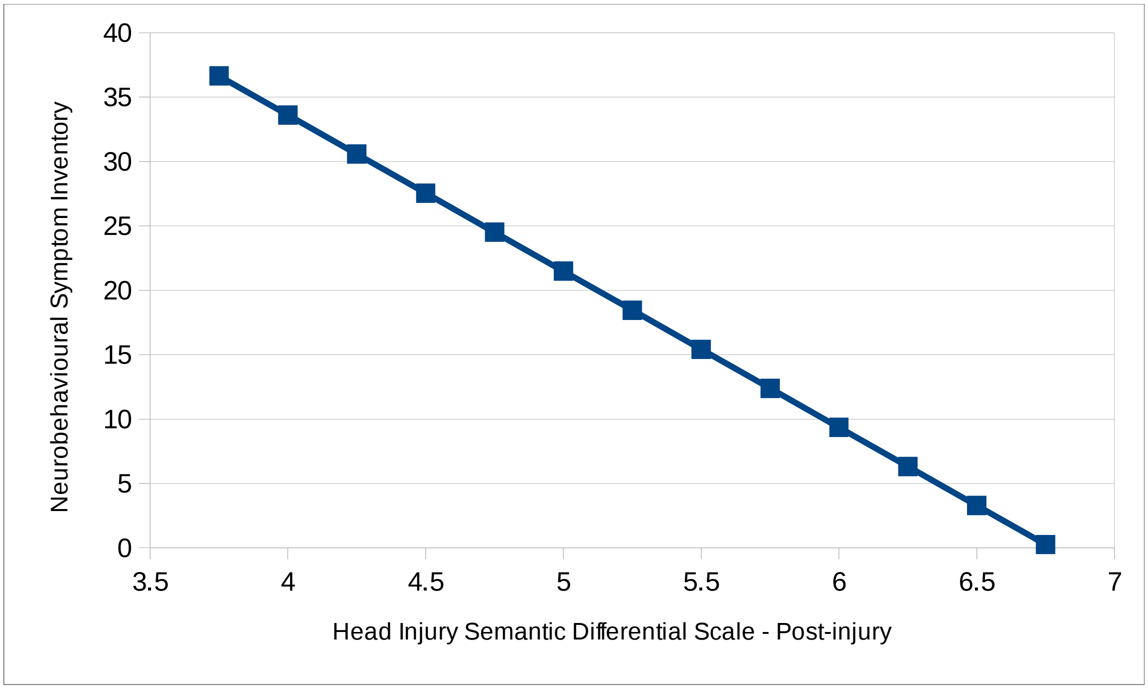

Figure 3. Regression line for prediction of post-concussion symptoms (Neurobehavioural Symptom Inventory, NSI) based on current self-perception (Head Injury Semantic Differential Scale – Post-injury, HISDS-Post). Pearson $r = -.63$

identity was investigated was a predictor of PCS severity: TBI-SIQ scores were regressed on scores for the NSI. The TBI-SIQ accounted for 47% of the total variability in NSI scores and was a significant predictor of NSI scores ($R^2 = 0.47$, $F_{1,\,13} = 11.52$, $\beta = 1.12$, $p < .01$; see Figure 4).

Hypothesis #5: identity, cognitive functioning, and PCS. It was further hypothesized that post-injury aspects of identity would moderate the relationship between cognitive impairment and PCS severity. Therefore, the role of current self-perception (HISDS-post), the change in self-perception from pre- to post-injury (HISDS-Difference), TBI-related self-concept (CSE-TBI-I), and TBI-related social identity (TBI-SIQ) were each investigated in turn. First, an interaction term was created between each of these identity scores and the weighted cognitive impairment scores (all scores were centered first). Subsequently, sequential multiple regressions were used to analyze the data such

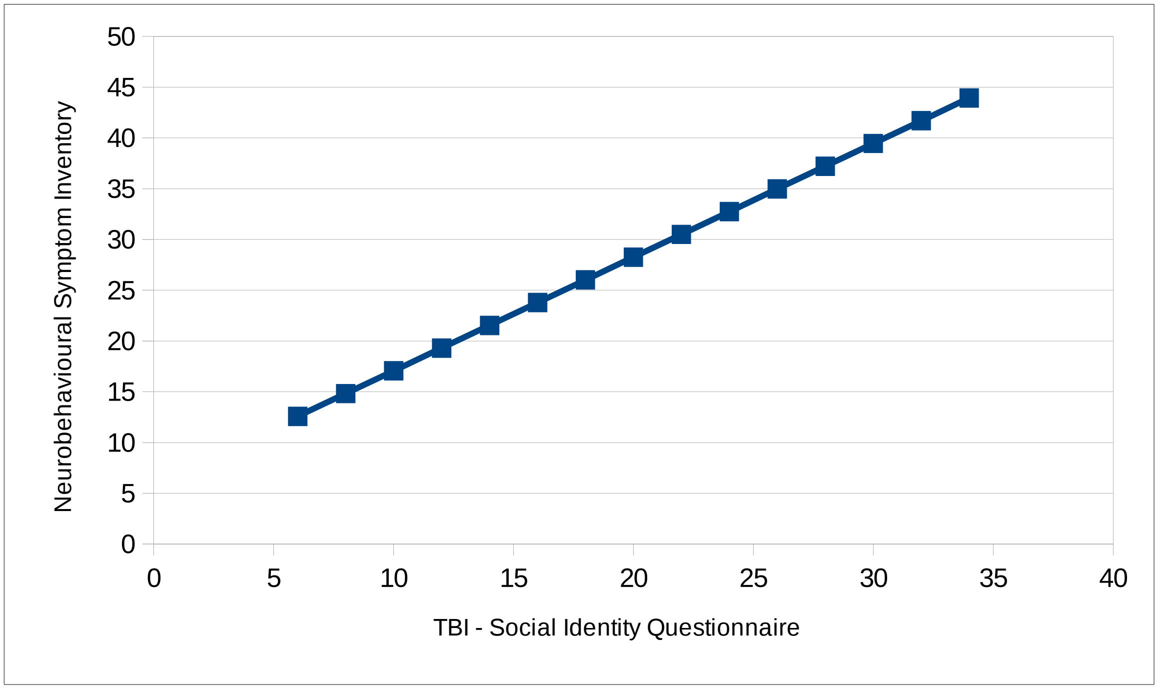

Figure 4. Regression line for prediction of post-concussion symptoms (Neurobehavioural Symptom Inventory, NSI) based on TBI-related social identity (TBI-Social Identity Questionnaire, TBI-SIQ). Pearson $r = .69$

that weighted cognitive impairment and the identity measure were first regressed on NSI scores, with the interaction term then added to the model separately. The change in the amount of variability accounted for (i.e., ΔR^2) was then investigated for each model including the interaction variable.

The model including current self-perception (HISDS-post), cognitive impairment, and their interaction was significant ($F_{3,\,16} = 4.77$, $p = .01$, $MS_e = 103.67$, $R^2 = .47$); however, inclusion of the interaction term did not significantly improve prediction ($\Delta R^2 = .01$, $p = .54$) and only HISDS-post was a significant predictor of NSI scores ($\beta = -13.28$, $p < .01$). The remaining models were not significant: difference in self-perception (HISDS-diff; $F_{3,\,16} = 1.00$, $p = .42$, $MS_e = 165.35$, $R^2 = .16$) and TBI-related self-concept (CSE-TBI-I; $F_{3,\,16} = 0.89$, $p = .47$, $MS_e = 168.49$, $R^2 = .14$).

The model including TBI-related social identity (TBI-SIQ), cognitive impairment, and the interaction significantly predicted NSI ($F_{3, 11} = 9.85$, $p < .01$, $MS_e = 59.74$, $R^2 = .73$). Further, inclusion of the interaction term significantly improved prediction over the model including weighted cognitive impairment and TBI-SIQ scores only ($\Delta R^2 = .26$, $p < .01$). TBI-SIQ was also a significant predictor in both models (*without interaction:* $\beta = 1.12$, $p = .01$; *with interaction:* $\beta = 1.56$, $p < .001$). Weighted cognitive impairment was only a significant predictor of NSI scores when the interaction term was added to the model ($\beta = -3.33$, $p = .01$). In order to further understand the interaction between these variables, predicted NSI scores were calculated based on high and low values for weighted cognitive impairment and TBI-SIQ, where low scores were those at the first quartile and high scores were those at the third quartile, as described in Tabachnick and Fidell (2007). The interaction (as depicted in Figure 5) suggests that, for individuals with lower cognitive impairment scores, higher TBI-SIQ scores are associated with slightly higher NSI scores whereas, for those with higher cognitive impairment scores, higher TBI-SIQ scores are actually associated with lower NSI scores.

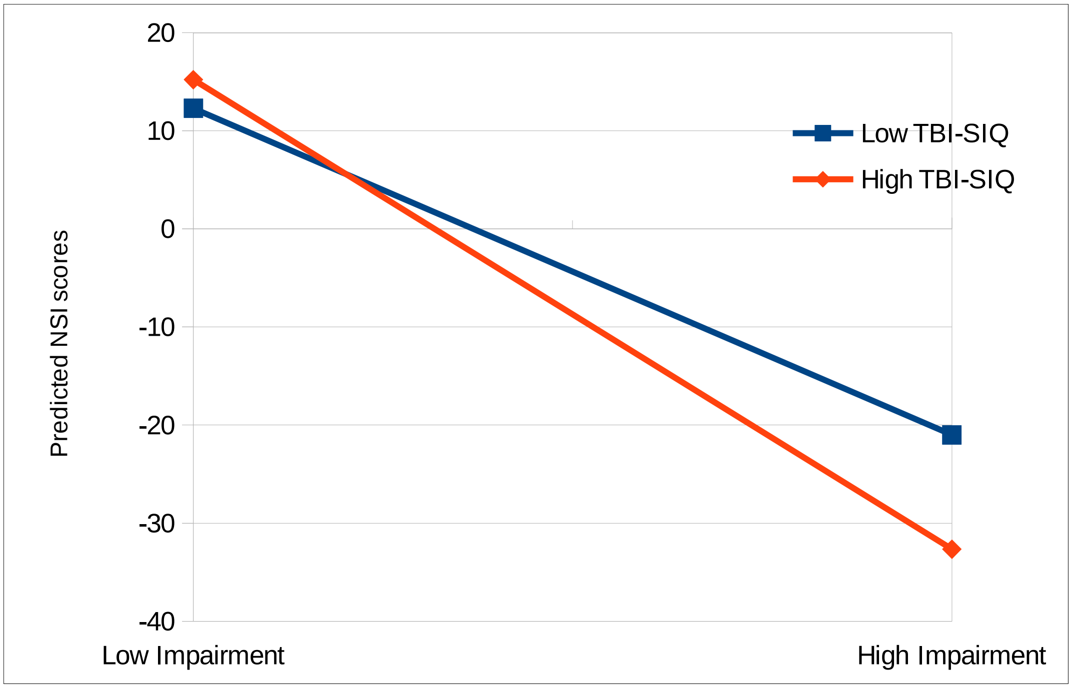

Figure 5. Predicted Neurobehavioural Symptom Inventory (NSI) scores based on low and high weighted cognitive impairment and TBI-Social Identity Questionnaire (TBI-SIQ) scores. Low Impairment and TBI-SIQ represent scores at the 25th percentile and High Impairment and TBI-SIQ represent scores at the 75th percentile.

Discussion

The goal of the current study was to undertake an investigation of persistent post-concussion symptoms from a broad, neuropsychosocial perspective. The main underlying assertion to this research was that there are multiple factors that underpin persistent PCS, and that a purely neuropathological or psychogenic perspective is not sufficient to understand this concept or the experience of those who suffer ongoing difficulties after having sustained an mTBI. Instead, it was argued that a neuropsychosocial approach is best suited to understanding the process of recovery after mTBI and the experience of persistent PCS.

Informed by findings within the relevant literature as well as clinical experience working with individuals experiencing persistent symptoms following mTBI, the factors investigated were cognitive functioning, anxiety susceptibility, and identity. *Cognitive functioning* was measured via standardized neuropsychological tools with demonstrated sensitivity within the mTBI population and validity assessing the domains of processing speed, attention, verbal fluency, and working memory. Cognitive functioning was also represented through a weighted cognitive impairment measure, which was calculated using the participants' performances, compared to age- and education-based normative data, across all of the neuropsychological measures completed. *Anxiety susceptibility* was assessed in the context of a cognitively demanding situation, and based on the difference in participants' reported levels of in-the-moment anxiety immediately before and immediately after completing a very challenging cognitive task (i.e., the PASAT). Lastly, multiple components of *identity* were investigated including self-perception (both before and after the injury), TBI-related self-concept, and TBI-related social identity. The current study investigated the role of these variables in predicting PCS severity.

Specifically, it was hypothesized that cognitive functioning, anxiety susceptibility, and identity would each predict the severity of PCS; further, it was hypothesized that anxiety susceptibility and identity would each modify the relationship between cognitive functioning and PCS.

In order to investigate these hypotheses, 21 adults at least one year post-mTBI participated in the study. Importantly, none of these individuals were involved in litigation at the time of their participation and there was no evidence of malingering or suboptimal effort based on the results of validity testing (i.e., the VSVT). Further, there was no history of severe psychiatric conditions and no significant symptoms of post-traumatic stress disorder at the time of involvement in the study. The reported levels of depression and anxiety were not suggestive of clinically significant symptomatology, on average.

One of the aims of the study was to obtain a sample that would reflect a range of mTBI-related outcomes, including individuals with ongoing difficulties as well as individuals with a complete recovery (i.e., no ongoing difficulties). The participants indeed reported a wide range of PCS severity on the NSI: based on the normative data provided by Soble and colleagues (2014), the sample's scores ranged from above the 51st percentile to below the 1st percentile and the sample's mean symptom severity was within the average range (25th - 50th percentile). Of note, these norms were derived from 108 military personnel who had sustained an mTBI during the course of deployment (Soble et al., 2014) and, while the current sample was quite etiologically diverse, no military personnel participated in the study (despite attempts to recruit from this population). This may be relevant given systematic differences in the experience and/or reporting of symptoms following mTBI. For example, Rigg and Mooney (2011) report that it is "common" to for military personnel to minimize their report of symptoms due to qualities such as stoicism and endurance, which are “admired within military culture” (p. S381). As such, miliary personnel may tend to report fewer/less severe PCS than civilians, on average. Given that the current sample's reported symptom severity was similar to that reported by Soble and colleagues' (2014) sample of military personnel (i.e., the current sample's mean was within the average range for the normative group), it is possible that the current sample may report fewer/less severe PCS than would typically be found within the general (non-military) mTBI population. However, given that there are no civilian based normative data available, it is unknown if this would truly be the case.

Beyond the severity of symptoms, the current sample also seems to represent a rather wide range of injury severity within the category of mild TBI. For example, estimated PTA and number of mTBIs varied quite widely across the sample, suggesting a breadth of severity in the participants' most recent injury (i.e., PTA) as well as a range of “cumulative” injury severity for those who reported having sustained multiple mTBIs. In addition, there were some individuals who reported that they now received disability or had to retire early due to difficulties following their mTBI, which may suggest that these individuals sustained more severe or debilitating injuries. The participants also represented a wide range of injury chronicity (i.e., time post injury).

Importantly, almost none of the injury-related variables (i.e., length of PTA, number of mTBIs, time post-injury) nor any of the demographic variables (i.e., gender, age, education) were found to be related to PCS severity. This is consistent with previous literature that has demonstrated that injury severity is almost never a significant predictor of recovery and outcome (e.g., Carroll et al., 2004). Etiology (i.e., sports related injury vs. non-sports related injury) was the only injury-related demographic variable relevant to the severity of PCS in the current study: individuals who had sustained their injuries while involved in a sport (including cycling) reported significantly less severe PCS than did those who sustained non-sports related injuries (e.g., motor vehicle accidents, falls). Again, this finding is consistent with previous research which indicates that athletes often recover more quickly and demonstrate less severe long-term concussive effects[8] (e.g., Ruff & Jamora, 2009; Iverson, 2011; Karr, Areshenkoff, & Garcia-Barrera, 2014). It has been proposed that this relatively consistent pattern in the literature may be due the fact that athletes are frequently healthier, are less likely to have pre-existing complicating factors (e.g., premorbid depression), and that the injuries themselves are typically less severe (Ruff & Jamora, 2009). A variety of psychosocial benefits gained through involvement in the sport (e.g., social support, identity, well-being) may also play a role in the better recovery commonly seen following sports-related injuries. Regardless, the pattern of findings within the current study and the literature suggest that it is inappropriate to ignore injury etiology (i.e., sports related vs. non-sports related injuries) in studies investigating recovery after mTBI. A more precise picture of patterns of, and expectations for, recovery may be garnered by investigating sports related and non-sports related injuries separately.

Cognitive Functioning

The participant's premorbid functioning (based on the NAART) was estimated to be, in general, within the average range for their age and education. Further, on all of the neuropsychological measures, the sample's mean standard scores (i.e., *z*- or T-scores) were within the average range compared to normative data. This suggests that the group's performances on all of the neuropsychological measures were consistent with what would be expected given a) their estimated level of functioning prior to the injury and b) their

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[8] Of course, this does not include the neurodegenerative effects (or chronic traumatic encephalopathy; CTE) thought to be due to a high number of concussive (and sub-concussive) head injuries sustained by professional athletes in contact sports, such as American football (see Iverson et al., 2015 for a review).

age and education. In other words, the sample did not show specific deficits in any of the cognitive domains assessed. This is consistent with previous research that has measured functioning within multiple cognitive domains at the chronic post-injury stage (e.g., Barker-Collo et al., 2015; Karr, Areshenkoff, & Garcia-Barrera, 2014; McCrea et al., 2009). Of course, there was much individual variability within performances on the cognitive measures: 14 participants had at least one neuropsychological test score between 1 and 1.5 standard deviations below the normative mean; seven participants had at least one score between 1.5 and 2 standard deviations below the mean; four participants had at least one score between 2 and 3 standard deviations below the mean; and one participant had at least one score more than 3 standard deviations below the normative mean. The weighted cognitive impairment score (based on the number of scores at least one standard deviation below the mean, with those scores further below the mean – or more impaired – being weighted more heavily) also illustrated a sizable range in participants' overall level of cognitive functioning. None of the personal demographics (i.e., age, education, and gender) nor injury-related demographics (i.e., etiology, length of PTA, and number of mTBIs) were associated with any of the measures of cognitive functioning/impairment.

Despite previous evidence demonstrating a relationship between PCS and various domains/measures of cognitive functioning (e.g., King & Kirkwilliam, 2011; Sterr et al., 2006) the data from the current study were not consistent with these findings and did not support the related hypothesis. In fact, cognitive functioning (based on performance in specific cognitive domains as well as overall level of impairment across domains) accounted for almost none of the variability in PCS severity in the current study. Further, the participants' objective cognitive functioning (again, both within specific domains and across domains based on the weighted cognitive impairment score) was not associated with the severity of self-reported cognitive symptoms (i.e., scores from the Cognitive subscale of the NSI). In other words, the sample's objective performance did not appear to be consistent with their subjective experience/report of even closely related symptoms. This is consistent with previous research where individuals with mTBI have been found to have poor self-assessment of their cognitive functioning (e.g., Spencer, Drag, Walker, & Bieliauskas, 2010). The discrepancy between subjective report and objective performance may be due to a number of factors. For example, Spencer and colleagues (2010) found that self-reported cognitive symptoms were, instead, associated with depression and anxiety such that those with more psychiatric symptomatology reported more cognitive deficits. In fact, this is consistent with the current finding that trait anxiety was a significant predictor of self-reported cognitive difficulties. Other possible factors include: participants with poor insight into their functioning; a tendency (intentionally or not) to under- or over-report symptom severity; the use of a questionnaire that did not accurately assess the specific cognitive difficulties experienced; the use of neuropsychological measures that were not sufficiently sensitive to detect the deficits that exist; or, relatedly, objectively “minor” deficits such that most participants perform within the average range despite a true (and subjectively perceivable) decline in cognitive functioning.

Measuring intra-individual variability (IIV) in cognitive functioning may be a means of assessing the presence of subtle changes in cognitive functioning, and may be more sensitive to the types of difficulties associated with more chronic mTBI. IIV reflects transient (i.e., trial-by-trial or day-to-day), within-person fluctuations in behavioural/cognitive performance as opposed to mean differences between groups

(MacDonald, Nyberg, & Bäckman, 2006). Importantly, calculating average performance from a single measurement occasion (as was done in the current study) can be a significant oversimplification of behaviour patterns and can lead to flawed estimates, and erroneous inferences, of mean group differences – especially as IIV increases and represents systematic (as opposed to random) error (MacDonald, Nyberg, & Bäckman, 2006). In other words, average performance can appear 'normal' despite possible fluctuations in underlying cognitive processes. Standard neuropsychological tests are generally not able to measure such fluctuations in performance. Research demonstrating increased levels of IIV in a variety of populations – including older adults, and individuals with dementia, ADHD, multiple sclerosis, and brain injury – suggests that IIV is a behavioural indicator of neurological integrity (Hill, Rohling, Boettcher, & Meyers, 2013; MacDonald, Nyberg, & Bäckman, 2006; Mazerolle, Wojtowicz, Omisade, & Fisk, 2013). Specifically, increased IIV has been linked to frontal lobe lobe dysfunction and white matter damage (MacDonald, Nyberg, & Bäckman, 2006; Mazerolle, Wojtowicz, Omisade, & Fisk, 2013). The association of IIV with these brain structures suggests that it may be of particular relevance following TBI given that these injuries are frequently associated with similar neuropathological features (i.e., DAI and/or coup-contrecoup injury often involving white matter tracts and the frontal lobes, respectively). That being said, limited research has been conducted investigating IIV following TBI, with only a small number of studies looking at individuals with mild injuries. For example, Hill and colleagues (2013) included individuals with TBI of varying severity and found that IIV increased with injury severity; however, a control group was not included thereby precluding any comparison of the mTBI group to those without a history of TBI. A study by Karr and colleagues (2014) investigated IIV on three executive-related cognitive tasks in a group of university athletes with mTBI. These authors found no difference in IIV between the mTBI group and a matched control. However, their use of a very specific sample (i.e., varsity athletes), known to typically experience full and rapid recovery (e.g., Ruff & Jamora, 2009), may not be representative of the wider mTBI population. As such, IIV in cognitive functioning is an area in need of further research.

Anxiety Susceptibility

The second possible predictor of PCS investigated in the current study was anxiety susceptibility, which was defined as the anxiety experienced in the context of a cognitively demanding situation. Anxiety susceptibility was measured using the STAI-S administered immediately before and after completion of the PASAT. When individual scores were examined, most of the participants reported higher levels of anxiety after the PASAT than they had immediately prior; however, the amount of change in the overall sample's in-the-moment anxiety symptomatology was not statistically significant. In fact, some of the participants reported *lower* levels of anxiety following the PASAT than they had reported immediately prior. It is unclear if this represents a true decline in anxiety for these individuals or, for example, issues related to reliable completion of the questionnaire (e.g., failure to consistently attend to and adjust direction of responses on reverse-coded items). From a clinical perspective, in-the-moment anxiety was within the average range (based on age- and gender-based norms) both before and after the PASAT, and the level of anxiety changed by just over half of a standard deviation. As such, it is not likely that the change in anxiety that the sample experienced in the context of a cognitively demanding situation (i.e., their anxiety susceptibility) would be considered clinically significant or meaningful.

It is somewhat surprising that the sample reported minimal stress in response to the PASAT as it is known to be a highly frustrating and anxiety-proving task. This has been perported to be largely due to how cognitively demanding the task is is – even for non-clinical, healthy individuals – relying on divided and sustained attention, working memory, and processing speed (Strauss, Sherman, & Spreen, 2006). For example, Wills and Leathem (2004) had to discard PASAT data from three of their 45 healthy, adult participants because "they had become so upset by the test that they refused to or could not go on" (p. 181). Further, significant increases in anxiety (based on self-report and physiological indicators) both during and/or following completion of the PASAT have been demonstrated in a number of previous studies (e.g., Willis & Leathem, 2004; or see Tombaugh, 2006 for a review). Importantly, it has also been shown that the STAI-S is a valid measure of the anxiety experienced during/after completion of the PASAT. For example, STAI-S scores have been shown to be strongly associated with both subjective ratings of overall anxiety severity (e.g., How anxious did you feel?) as well as biomarkers of stress (e.g., heart rate and salivary levels of α -amylase) measured in response to completion of the PASAT (Willis & Leathem, 2004; Noto, Sato, Kudo, Kurata, & Hirota, 2005). As such, despite employing a context that should have been sufficiently anxiety provoking (i.e., the PASAT) and a tool suitable to measure the anxiety experienced (i.e., the STAI-S), the current sample did not report marked anxiety susceptibility.

Furthermore, anxiety susceptibility was not related to objective performance on the PASAT (i.e., the standard scores obtained). In other words, the participants' experience of the task as stressful was not systematically associated with their actual ability or performance on the task. This suggests that one's susceptibility to anxiety in a cognitive demanding situation is, instead, a reaction to their believed performance (i.e., if they *feel* that they are doing well, and performing as they would expect themselves to, or if they feel that they are not). This theory is supported by previous research demonstrating no relationship between reported anxiety and actual performance on the PASAT, rather, anxiety being associated with *believed* performance on the PASAT (Willis & Leathem, 2004). While believed performance was not assessed in the current study, it was anecdotally observed that some of the participants expressed the belief they had done poorly on the task; however, it is unclear if these participants actually did report higher levels of anxiety susceptibility.

In the current study, none of the hypotheses regarding anxiety susceptibility and PCS were supported. Specifically, anxiety susceptibility did not directly influence the severity of PCS nor the severity of only cognitive symptoms. In addition, anxiety susceptibility did not moderate the relationship between cognitive functioning and PCS severity in any way. This is the first study to investigate the role of this type of context-dependent anxiety (i.e., anxiety susceptibility) in persistent PCS. The results suggest that anxiety experienced in a cognitively demanding situation is not a factor in mTBI symptom severity more than one year post-injury, despite clinical observations that may indicate the contrary. For example, Goldstein (1952) referred to emotional 'catastrophic reactions' seen in the context of rehabilitation, whereby individuals become overwhelmed when confronted by their disabilities following TBI. While anxiety may well play a role in such reactions, other authors have purported that catastrophic reactions are characteristic of TBI survivors who fail to develop a cohesive sense of self and for whom injury-related failures result in a threat to their self-esteem and self-concept (see Belchev et al., 2017). Therefore, it may be that negative reactions to post-TBI (cognitive) challenges may instead reflect problems related to identity, and anxiety may not be an appropriate measure of what is experienced in the moment. On the other hand, it may be that individuals with mTBI are less likely than individuals with more severe injuries to experience such reactions. For example, Belchev and colleagues (2017) cites research demonstrating that individuals who underestimate their post-injury functioning have fewer behavioural disturbances (e.g., catastrophic reactions) than individuals who overestimate their post-injury functioning (i.e., those who lack awareness of their deficits). The tendency towards the 'good old days' bias, whereby individuals underestimate their current functioning while overestimating their past functioning, could suggest that individuals with mTBI are less prone to these catastrophic reactions. In other words, because they already underestimate their current abilities, they are less likely to experience “failures” as threats to their self-esteem or self-concept. This theory could be consistent with the low levels of anxiety susceptibility reported by the participants in the current study. Additional research into the factors that underlie these negative reactions to post-TBI difficulties, and how these may relate to recovery and persistent PCS, is needed.

Despite there being no role for anxiety susceptibility in PCS severity, *trait* anxiety (measured using the STAI-T) was found to be significantly associated with symptom severity in the current study. Even when affect-related PCS items were removed from the analysis (in order to account for the circular logic of investigating the impact of anxiety on a group of symptoms that include both anxiety and depression), trait anxiety was still associated with the remaining PCS. Furthermore, trait anxiety was also significant related to self-reported cognitive difficulties (i.e., NSI Cognitive subscale). Therefore, trait anxiety seems to be a robust statistical predictor of PCS severity. Trait anxiety is defined as a stable tendency to experience feelings of tension and worrisome thoughts, whereas state anxiety (which would underlie anxiety susceptibility) is an in-the-moment response to external contingencies in the environment (or the interpretation of them) and should vary depending on these contingencies (Rossi & Pourtois, 2012). Consistent with the current study, there is much literature demonstrating that one's general level of anxiety (i.e., trait anxiety) is associated with outcome or PCS severity (e.g., see Moore, Terryberry-Spohr, & Hope, 2006; Broshek, De Marco, & Freeman, 2015 for reviews). Furthermore, research with a prospective cohort design has demonstrated that level of anxiety at two weeks post-injury is a significant predictor of post-concussion syndrome diagnosis at three and six months post injury (Hou et al., 2012). Of note, other aspects of anxiety have also been found to be related to outcome after mTBI. For example, Wood and colleagues (2014) demonstrated that anxiety sensitivity – which represents one's fear of the physiological indicators of anxiety (e.g., increased heart rate, sweating) and their social implications (e.g., loss of control, embarrassment) – was related to the severity of PCS. These authors note that individuals who have high levels of anxiety sensitivity are more prone to catastrophically misinterpret bodily sensations and exacerbate the impact of somatic sensations by narrowing the focus of their attention to their internal physiological state (Wood et al., 2014). This pattern may play a role in the issue of misattribution of symptoms that can occur after mTBI (i.e., where previously existing but unnoticed, or new but unrelated, sensations or "symptoms" come to be incorrectly attributed to the mTBI – see Iverson & Lange, 2011b).

Taken together, these finding suggest that PCS is influenced by one's overall, general level of anxiety and/or their (mis)interpretation of the symptoms experienced due to anxiety, as opposed to levels of in-the-moment anxiety experienced in the context of a specific (cognitively demanding) situation. These results may also indicate that individuals with preexisting difficulties with anxiety could be at increased risk of experiencing persistent symptoms following an mTBI. This may be due to a reduction in the symptom-perception threshold associated with the experience of anxiety or because of the exacerbation of symptoms due to increased levels of psychological distress (Fink, Rosendal, & Toft, 2002). Regardless, the presence of a history of anxiety should be inquired about by medical professionals during the acute post-injury phase, and levels of anxiety monitored over the course of recovery so that appropriate intervention can be provided if needed. The evidence suggests that the severity of PCS may actually be reduced by providing treatment that is aimed at reducing levels of anxiety. For example, equipping individuals with skills to help them better cope with anxiogenic thoughts and situations may be of significant benefit and help to reduce the severity of other persistent symptoms following mTBI.

Identity

The role of psychosocial factors in recovery after brain injury is a relatively newer area of study, yet essential if we are to garner a more holistic understanding of the processes involved. For instance, factors such as perceived social support have been shown to influence recovery and the experience of symptoms after mTBI (e.g., McCauley, Boake, Levin, Contant, & Song, 2001). Identity – or one’s experience of who one is – is known to be commonly affected by brain injury (e.g., Carroll & Coetzer, 2011; Iverson, Lange, Brooks, & Rennison, 2010; Muenchberger et al., 2008) however the potential impact of identity on PCS specifically had not been previously investigated. Thus, the current study aimed to begin to understand how identity may impact recovery. Further, this question was investigated from a social identity approach such that multiple components of identity were measured, including personal/individual aspects (i.e., self-perception) as well as more group-based aspects (i.e., TBI-based self-concept and social identity). None of the measures of identity included in the current study (i.e., the HISDS, CSE-TBI-I, and TBI-SIQ) had significant intercorrelations, so it can be assumed that divergent aspects of identity were, in fact, being assessed. Furthermore, none of these variables were significantly influenced by any of the demographic factors.

While the participants' ratings of their pre-injury self-perception did not significantly differ from their post-injury (current) self-perception, on average, pre-injury self-perception was slightly more positive than post-injury self-perception ratings. This may be seen as generally consistent with previous research, which has demonstrated poorer, or more negative, post-injury self-perception (e.g., Beadle, Ownsworth, Fleming, & Shum, 2016; Levack, Kayes, & Fadyl, 2010; Doering, Conrad, Rief, & Exner, 2011). This finding may also represent the "good old days" bias commonly believed to be an issue following more mild injuries (Iverson, et al., 2010), whereby individuals tend to underestimate past problems and view their past, non-injured, self as more healthy than their current self. On the other hand, a number of the participants in the current study reported their current self-perception as more positive when compared to their pre-injury self-perception. This suggests that at least some of the current participants experienced what may be termed post-traumatic growth, or the experience of positive changes that occur, often, as a result of attempts at coping with the traumatic event itself and/or its sequelae (Tedeschi & Calhoun, 2004). Some of the changes that are associated with post-traumatic growth include a greater sense of personal strength, as well as improved relationships, a sense of new life possibilities, and a greater appreciation of life (Tedeschi & Calhoun, 2004). Anecdotally, this was also evident in comments made by some of the participants indicating that their injury had, for example, helped them to rethink their priorities or appreciate their lives and the people in it more than they had in the past.

Clinically, post-traumatic growth may be the antithesis of the good old days bias, and this spectrum appears to have been represented within the current sample.

The hypothesis that identity would predict PCS was largely supported in the current study: both personal and social aspects of identity seemed to influence the experience of persistent PCS after mTBI. Regarding personal/individual aspects of identiy, the results suggest that current (post-injury) self-perception is significantly associated with PCS severity such that individuals with more negative current self-perception report more severe PCS. While the association between self-perception and PCS has not been previously investigated, existing research does demonstrate that self-perception is related to other measures of outcome such as quality of life (Doering et al., 2011; Vickery, Gontkovsky, & Caroselli, 2005). In the current study, neither pre-injury self-perception nor the amount of change in one's self-perception from before to after the injury were significant predictors of the severity of PCS.

The current study also indicates that TBI-related social identity (i.e., one's affiliation with brain injury groups, sense of belonging to the wider community of TBI survivors, and self-categorization as a person with a brain injury) is significantly associated with PCS severity. In other words, those with stronger ties to the TBI community report more severe PCS. This may be seen as generally consistent with what would be predicted based on social identity theory whereby an individual's self-categorization regarding a given illness group can lead symptom perception to converge towards those associated with that illness, and can also increase the reported severity of relevant symptoms (St. Claire & Clucas, 2012; see Haslam, Jetten, Postmes, & Haslam, 2009 for a review). Of course, because of the present research design, the true direction of these relations cannot be known. For example, one cannot rule out the possibility that, instead, those participants who experience more severe PCS following their injury are more likely to search out, be referred to, and/or engage in groups and services – or the TBI-community more generally – and then develop stronger ties to this community as a means of coping with their post-injury difficulties.

The final hypothesis investigated in the current study predicted that identity would moderate the relation between cognitive functioning and PCS severity. It was found that there was a significant interaction between TBI-related social identity and weighted cognitive impairment scores as they relate to PCS severity. Interestingly, the interaction suggests that social identity had little impact on the severity of PCS reported by those with lower levels of cognitive impairment (i.e., better cognitive functioning); however, for those with higher levels of cognitive impairment, stronger social identity was associated with less PCS. While the relation and interaction between these variables is deserving of further investigation, it is possible that this seemingly unexpected result may be due to the role that social identity and self-categorization play in the dynamics of effective social support. Haslam and colleagues (2009) describe a growing body of work illustrating that social support is more likely to be given, received, and interpreted in the spirit in which it was intended when those who are giving and receiving that support perceive themselves to share a social identity. Similarly, research has shown that a sense of shared identity underpins the capacity for members of disadvantaged groups to work together to buffer themselves from negative consequences that may arise as a result of their circumstances (see Haslam et al., 2009 for review). It is possible that objective cognitive difficulties in particular make support from non-TBI-related sources less beneficial due to a potential loss of previously perceived shared social identities (e.g., those acquired through roles within an employment setting, or certain hobbies);

subsequently, those with ongoing cognitive difficulties may be in a position to make better use of the social capital (including social support) that is offered through social interactions with those of the TBI-related in-group. What is clear from these results is that social identity in and of itself, as well as its association with other factors relevant to individuals following TBI, is deserving of further investigation in order to clarify their impact on recovery and outcome.

Implications

Taken together, the results of the current study suggest that the severity of persistent PCS, at least one year post injury, is clearly influenced by psychosocial factors – namely general propensity to experience symptoms of anxiety, current self-perception, and TBI-related social identity. Neuropsychological factors (i.e., level of cognitive impairment) were not relevant to the experience of PCS as assessed in the current study. Given the current argument that mTBI is best understood using a whole-person, neuropsychosocial perspective, what do these findings suggest regarding the application of this perspective to further research into recovery after mTBI and the experience of persistent PCS? Perhaps the psychogenic perspective (which purports that PCS is solely of psychological origin) is the appropriate framework from which to consider the experience of PCS. Consistent with the current results, many studies have demonstrated the association between psychological difficulties (i.e., anxiety and depression) and PCS (see Broshek, De Marco, & Freeman, 2015 for a review). That being said, the role of neuropathological factors in recovery and outcome after mTBI should not be discounted; instead, it may be that cognitive functioning (as measured in the current study) is not a good indicator of underlying neuropathology in mTBI – especially at more than one year post injury and in a sample that represents the spectrum from good to poor recovery. For example, previous research demonstrating a relation between neuropsychological functioning and PCS severity more than one year post injury included only those with significant PCS, or a 'diagnosis' of post-concussion syndrome (e.g., King & Kirkwilliam, 2011) which could indicate that the association between cognitive functioning and PCS is only relevant for this specific group of individuals (i.e., those with a high number of symptoms or who meet diagnostic criteria for the syndrome). It is also possible that other measures of cognitive functioning – such as IIV, a measure of underlying cognitive fluctuations and neurological integrity – may be more relevant in chronic mTBI and associated with ongoing difficulties post-injury; however, this is an area that has yet to be adequately investigated. That being said, research has demonstrated that other factors such as cognitive reserve can protect against the development of PCS, where cognitive reserve refers to the idea that individuals have different amounts of 'reserve' that act as a buffer when the brain is injured – perhaps through protective factors that moderate the brain's ability to withstand the injury, or through factors that promote/facilitate repair and recovery of the damage caused (Oldenburg, Lundin, Edman, Nygren-de Boussard, & Bartfai, 2015). Further, there is evidence that, even for individuals who experience a very quick and full recovery, differences in the structure and/or function of the brain can remain. For example, when high school athletes with mTBI were tested 13 hours post injury, they demonstrated objective cognitive deficits (in reaction time and working memory), reported significant concussion-related symptoms, and had decreased activation of right hemisphere attentional networks relative to controls. When tested again at seven weeks post injury, a full recovery was indicated (i.e., no statistically, or clinically, significant differences between the mTBI group and controls in cognitive functioning or self-reported concussion-related symtpoms); however, the mTBI group now demonstrated *increased* activation in the same brain region relative to the control group. The authors suggest that this improvement in symptom severity and cognitive functioning may be mediated by compensatory increases in activation (Hammeke et al., 2013) suggesting decreased efficiency, or the brain having to “work harder,” to attain the same level of functional outcome as their non-injured peers. In fact, this process may underlie the cognitive fatigue so commonly reported after brain injury. As such, it may be that cognitive functioning as assessed in the current study was not a sensitive enough measure of underlying neuropathology. As the literature suggests, intact cognitive functioning does not necessarily imply the absence of underlying neuropathological changes.

It may be that neuroscientific methods, such as neuroimaging, would represent more sensitive (and direct) measures of neuropathology and thus be better able to elucidate the role of the 'neuro' in the neuropsychosocial approach. Until recently, neuroimaging lacked the sensitivity (and methodology) to fully investigate the possible changes following mild injuries. More recently, methods such as magnetic resonance imaging (MRI), and specifically diffusion tensor imaging (DTI), have provided a means of gaining a better understanding of even mTBIs that would previously be considered 'non-complicated' (i.e., those injuries with no overt evidence of damage, such as bleeding or contusion, on more traditional imaging such as CT scan). For example, a number of MRI studies have demonstrated structural differences in both grey and white matter areas of the brain – particularly (although not exclusively) in the frontal lobes (Churchill et al., 2017) – even at a year or more post-injury (Dean, Sato, Vieira, McNamara, & Sterr, 2015; Ljungqvist et al., 2017; Zhou et al., 2013). Other studies utilizing functional MRI or DTI have demonstrated functional/connectivity changes (e.g., Churchill, Hutchinson, Leung, Graham, & Schweizer, 2016; Jang & Kwon, 2015; Jang & Lee, 2017) and even degenerative changes in white matter microstructure over the first few months of recovery (Næss-Schmidt et al., 2017). Even through the use of electroencephalagram (EEG), differences in brain function (i.e., the latency of the visually-evoked potential) have been found up to 10 years post injury (Fimreite, Ciuffreda, Yadav, 2015). Genetic factors relevant to brain function have also begun to be investigated for the role that they may play in the brain's ability to recover after injury. For example, McDevitt and colleagues (2015) found that differences in the genes for specific NMDA receptors in the brain were associated with time to recover from mTBI such that, those with specific alleles were more likely to experience a prolonged recovery (defined in this study as taking longer than 60 days to recover). Other research has demonstrated the likelihood that the apolipoprotein E gene (APOE) ε4 allele may adversely influence recovery following TBI (Lawrence, Comper, Hutchison, & Sharma, 2015). That being said, the association between these fundamental neurophysiological/biological factors and recovery, or persistent PCS is unclear; a possible association has only been investigated in a few studies and the results are inconsistent, with some studies supporting a neurophysiological basis for PCS (e.g., Dean, Sato, Vieira, McNamara, & Sterr, 2015) and others finding no correlation between brain changes and PCS severity (e.g., Næss-Schmidt et al., 2017). Still, these means of assessing neuropathology will soon be much more sensitive than the indirect means of assessment that is neuropsychological/cognitive functioning and, as such, they may be more appropriate to furthering the investigation of mTBI and persistent PCS in particular.

Further, the relevance of (psycho)social factors in recovery after mTBI and persistent PCS is ignored in both the psychogenic and the neuropathological perspectives. As the current study has demonstrated, both personal and socially-mediated aspects of identity are important following mTBI and have important and complex relations with ongoing PCS. This may not be surprising given that previous research has demonstrated that identity is commonly affected by brain injury (e.g., Beadle, Ownsworth, Fleming, & Shum, 2016). Still, many questions remain regarding the roles of various individual and social factors in post-injury identity development and, while quantitative methods are able to demonstrate changes in identity, qualitative methods are likely best suited to investigate the processes underlying these changes in identity. For example, Roger, Wetzel, Hutchinson, Packer, and Versnel (2014) utilized a multiple perspectives case study sample and interpretive description analysis in order to investigate how individuals with various neurological conditions maintain a sense of self. Their research demonstrated that participants utilized a variety of cognitive and behavioural strategies to maintain a strong (although possibly changing) sense of self, and social contextual factors played a role in the process for many of their participants. For example, making use of both old and new communities of support was identified as a way that participants managed their self-perception and sense of well-being. The authors purport that these communities offer individuals with neurological disorders a sense of belonging to a community where they are not the anomaly. Participants also described making use of social comparison in order to inform their identity and to put their sense of self into perspective, whereby they were able to reflect on the obstacles in their own lives while also acknowledging that they were lucky relative to many other individuals. The authors suggest that this is part of the participants' process of developing acceptance of their condition (Roger et al., 2014). While further investigation is needed to clarify the roles that identity and other psychosocial factors have in recovery after mTBI, it is evident that these are highly relevant aspects of post-injury life.

When the lines of research discussed above are considered together with the results of the current study, the argument for approaching mTBI research from a neuropsychosocial perspective remains strong. Neuropathological changes, which in certain individuals may be long lasting, underlie mTBI in at least some (if not all) cases – even when indirect measures such as neuropsychological functioning are unable to demonstrate their existence. Psychological factors, including one’s own reaction to and interpretation of their experiences (i.e., their tendency to experience anxiety), are highly relevant to outcome after mTBI and may, in some cases, be (at least in part) the direct result of some of the neuropathological changes caused by the injury (e.g., Moreno-López, Sahakian, Manktelow, Menon, & Stamatakis, 2016). Finally, psychosocial factors, including one's beliefs about themselves and their affiliation with TBI-based social groups, seem to be important to the experience of persistent PCS and likely play a significant role in the ongoing process of recovery. As such, it seems important for researchers and clinicians to consider the whole person and the possible interactions of these neuropsychosocial factors when developing hypotheses or conceptual understandings of mTBI.

Treatment implications. The results of the current study also provide insight into treatment options and areas of focus that may be particularly helpful at the chronic post-injury phase. Specifically, the results imply that psychological treatments aimed at decreasing anxiety and increasing acceptance of the current self may be particularly beneficial for those experiencing persistent PCS. Unfortunately, there has been a paucity of research investigating the effects of treatment following TBI in general, and research is especially limited regarding psychological or emotion-focused treatments – particularly for individuals who sustained mild injuries (Allen, 2007; Potter & Brown, 2012).

Most of the existing literature discusses the efficacy of cognitive-behavioural therapy (CBT) following brain injury. This may be because a number of difficulties commonly targeted during the course of CBT interventions have been identified as highly relevant following TBI, including locus of control (e.g., blaming others for the injury), reduced active coping strategies (with strategies such as avoidance, worry, and wishful thinking associated with worse outcome following TBI), as well as poor self-esteem and reduced perceived ability to cope with the injury (see Potter & Brown, 2012 for a review). The fundamental cognitive techniques of CBT may also be particularly suited to help individuals begin to think about and challenge previously held attributions and causal beliefs about the injury (e.g., the belief that symptoms are solely the direct consequence of the original injury) as well as unhelpful, pre-existing patterns of thinking (e.g. jumping to conclusions, catastrophising, all-or-nothing thinking) and personality traits (e.g., perfectionism) (Potter & Brown, 2012). Behavioural activation, another fundamental technique of CBT, can also help individuals begin to re-engage in rewarding activities that may now be avoided due to issues related to the injury (Potter & Brown, 2012). While only a few studies have investigated the actual effectiveness of CBT for individuals who have sustained mTBI, the results have been promising and suggest that brain injury survivors do benefit from CBT-based interventions (e.g., Backhaus, Ibarra, Klyce, Trexler, & Malec, 2010). One randomized controlled trial of treatment consisting of both CBT and cognitive remediation for individuals more than one year post mTBI has been conducted and the results illustrated a notable benefit to both emotional and cognitive functioning (Tiersky et al., 2005). There is also research demonstrating that mindfulness-based stress reduction (MBSR) training – which involves learning attentional control and non-judgmental awareness of thoughts, feelings, and bodily sensations – is associated with significant improvements in self-efficacy, cognitive functioning (namely, attentional control and working memory), as well as decreases in PCS severity in individuals at least seven months post-injury (Azulay, Smart, Mott, & Cicerone, 2013).

These treatment approaches (i.e., CBT and MBSR) may be particularly appropriate for individuals whose profile of difficulties is strongly suggestive of emotional distress and/or a functional (as opposed to neurological) disorder. In fact, for these individuals, other interventions (such as those focusing purely on cognitive rehabilitation) may actually be contraindicated (Mateer, Sira, & O'Connell, 2005). That being said, it is important for clinicians to consider that modifications to treatment may be required in order to accommodate the needs of individuals with brain injury. The presence of cognitive difficulties (such as poor attention regulation, slowed speed of information processing, reduced recall, disorganization, etc.) may necessitate some or all of the following modifications: an increase in the number and/or frequency of sessions; additional time in order to explain concepts and to repeat procedures; modeling (particularly of more complex techniques) used more frequently; and utilization of written notes and reminders regarding topics covered and/or techniques discussed, as well as logs to track homework assigned/completed (e.g., Azulay et al., 2013; Khan-Bourne & Brown, 2003). Further, the use of a fixed, session-by-session treatment protocol may be difficult to establish (or utilize) due to the heterogeneity of mTBI and PCS presentations (Potter & Brown, 2012). As such, it is important for clinicians to be flexible in their approach to treatment when working with individuals with PCS following mTBI.

Beyond the impact of individual factors, the current results also provide new insights into socially-mediated factors of identity following mTBI and suggest that affiliation with the TBI community is associated with poorer outcome (i.e., more severe

PCS) more than one year post-mTBI. While (as previously discussed) this finding seems consistent with social identity theory, it is surprising given that community involvement is generally believed to be beneficial. Variables related to one’s social network, such as network size and density, are reported to have a direct effect on health outcomes as well as an indirect effect through social support, which can buffer the detrimental effects of stress on well-being (Rauch & Ferry, 2001; Walsh, Muldoon, Gallagher, & Fortune, 2015). For example, at three years post mTBI, community integration was negatively associated with PCS severity, while social integration (i.e., frequency of socialization with others and level of companionship experienced) and productivity (i.e., employment- and school-related activities engaged in outside of the home) were positively associated with life satisfaction (Stålnacke, 2007). That being said, the current study did not assess the participants' social network more generally, or overall levels of social integration, and instead focused on affiliation within the TBI community. This focus on TBI-related social identity may have resulted in aspects of identity more important to the participants being overlooked. This is exemplified by the fact that, when asked the open-ended question, “Which of your current groups or roles do you feel is most important to who you are?”, none of the participants provided a response related to their status as a TBI survivor. Further, while the current study asked about feelings of belonging to the TBI community, perceived social support was not investigated. The importance of this distinction is highlighted in research conducted by Walsh and colleagues (2015) which demonstrates that 'belonging' to a group may not be sufficient, particularly if one does not perceive themselves to be supported by other members of the in-group. In keeping with this, research investigating a TBI peer-mentoring program found that mentees actually reported increased levels of depressive symptomatology after participating in the program

(Struchen et al., 2011). While the sample for this study was small (i.e., 12 individuals matched with mentors, and 18 in a wait-list control; Struchen et al., 2011), the results are evocative and highlight an area in much need of further investigation. That being said, it may well be that mere affiliation with the TBI community can result in an exacerbation of symptoms (particularly for those with mTBI), whereas the amount of perceived support received from the community is what can help to ameliorate the negative impacts of the injury.

Pursuant to this line of thought, the current results also suggest that affiliation with the TBI community may actually have iatrogenic effects in terms of recovery and symptoms severity for individuals with mTBI. Iatrogenesis is a state of ill health or adverse outcome that is essentially caused (or worsened) by some type of intervention or medical treatment (Iverson & Lange, 2001b) and iatrogenic symptoms can form when an individual believes what they are told, regardless of the veracity of the information (Bender & Matusewicz, 2013). It is well known that, in general, an individual's knowledge and beliefs about an illness will affect how they interpret bodily sensations: a number of studies have shown that early intervention in the form of (appropriate) education and reassurance can have beneficial effects on outcome following mTBI (Allen, 2007). In fact, even in the post-acute period, intervention designed specifically for individuals with mTBI and including group-based education and support, was found to have beneficial effects on long-term outcome and PCS (Vikane et al., 2017). Furthermore, much of the research demonstrating beneficial outcomes following neurorehabilitation are focused on individuals who have sustained severe brain injuries (e.g., Malec, Smigielski, Depompolo, & Thompson, 1993). Unfortunately, specialized services for individuals with mTBI are lacking in Victoria, BC. This means that any services obtained – either formal (such as outpatient neurorehabilitation) or informal (such as those offered at community-based brain injury societies) – will most probably include individuals across the spectrum of TBI severity. Therefore, it is likely that the participants who reported higher levels of affiliation with the TBI community had spent comparatively more time with individuals who had sustained more severe injuries and, thus, would be experiencing more significant post-injury difficulties. It is possible that these interactions may convey unrealistic (and negative) expectations for recovery. The effectiveness of currently-available brain injury related services for individuals of varying levels of injury severity is an area in need of further investigation as it should not be assumed that individuals with mild injuries will experience the same benefits from these interventions.

The final major implication arising from the current study is the significant amount of individuality inherent within the mTBI population. For example, as Iverson and Lange (2011a) state, “It is incorrect to assume that mTBIs *cannot* cause permanent brain damage and it is incorrect to assume that mTBIs *typically* cause permanent brain damage” (p. 697). This quote encapsulates the difficulty of conducting research on recovery after mTBI. First, as previously discussed, the diagnostic criteria used to determine injury severity result in individuals with a momentary alteration of consciousness (e.g., feeling dazed or confused) falling into the same category as individuals with up to 30 minutes of unconsciousness. Based on this factor alone, one could expect these individuals to have very different post-injury experiences and outcomes. Differences in the type, extent, and location of damage to the brain can have vastly different outcomes (e.g., focal damage versus diffuse axonal injury). Then we must consider the pre-existing personalities, stressors, coping strategies, and abilities that individuals possess, and how these may interact with the biological, psychological, and social effects of the injury. For example, certain personality characteristics (such as perfectionism or 'over-achievers') are believed to exacerbate the impact of relatively minor changes in functioning (Iverson & Lange, 2011b). On the other hand, for individuals working in certain settings (e.g., lawyers, physicians, air traffic controller, etc.) even relatively minor cognitive changes (which may not appear to be “deficits” on testing, yet represent a decline from an individual's previous level of functioning) can result in them being unable to return to their previous employment. The complexity of recovery after brain injury cannot be overstated and it is this complexity that can make mTBI so difficult to study using typical, quantitative research methods: the results of the group do not necessarily reflect the experience of the individual. Therefore, while well-designed studies can provide insight into typical patterns of recovery or interactions between variables that may make a good outcome more likely, it is very difficult to know how well the knowledge obtained will generalize to a different group of survivors or a specific individual who has sustained an mTBI. It is essential for clinicians and health care providers to work with the “individual in the room”, so to speak, and to take into consideration the role diverse factors such as medical and mental health history, past experiences, current situation (e.g., socioeconomic status, living situation, availability of social support, etc.), as well as expectations for recovery and long-term prognosis can have on an individual's presentation in the moment. The variety of aspects that should be considered in any one patient's care following a TBI highlights the importance of collaborative, interdisciplinary treatment teams comprised of professionals with varying areas of expertise who communicate with each other and work together in the patient's best interest.

Limitations and Future Directions

There are a number of limitations of the current study that should be acknowledged and taken into consideration when conducting further research in this area. First, although the sample size was deemed to be sufficient based on *a priori* power analyses, because many of the variables of interest had not been previously investigated (or not investigated as they relate to persistent PCS) it was exceedingly difficult to find appropriate estimates of effect size in the existing literature. Thus, it is possible that some of the null findings were due to inadequate power to detect significant effects which may account for some of the statistically non-significant findings. That said, the current study could serve as an exploratory investigation of these variables and inform future research that may be interested in the neuropsychosocial factors of recovery. Second, injury severity was based solely on self-report (i.e., length of LoC, PTA, and hospitalization) and so may not be accurate. While it would be ideal to obtain hospital records (including GCS and results of any neuroimaging completed) this was not feasible in the current study. More broadly, one must also then consider that a large number of individuals who do not seek out immediate medical attention following an mTBI would not be included in the research as they would not have objective corroboration of injury severity markers. Third, it is unclear how well the sample in the current study actually reflects the population of mTBI survivors. Specifically, in order to avoid significant confounding variables, individuals currently involved in litigation related to the injury and individuals with significant symptoms of PTSD were excluded from participation. Also, it is important to highlight that this self-selected sample was quite well educated, included more females than males, and tended to report levels of depression and anxiety that are not considered clinically significant. It is unknown how the results of the current study would generalize to individuals who differ from this sample in some of these facets thought to be relevant to recovery. Fourth, as previously mentioned, because of the research design, the true direction of the associations found is unclear. For example, it may well be that individuals with more symptoms develop a stronger TBI-based social identity because they seek out or are offered services to help with their symptoms (rather than the other way around). Finally, the applicability of these results to the "real world" is unclear. For example, while seemingly low levels of anxiety sensitivity were found in the current study, this may be due to the relatively low-stakes setting of the lab and may not be a true reflection of the participants' experiences when encountering a cognitively demanding situation in daily life, where the stakes are much higher (e.g., at work, where "failing" to perform in a demanding situation could have a multitude of possible consequences).

The results of the current study suggest many directions for future research. Given inconsistent results in the literature regarding cognitive functioning in chronic mTBI and failure to find an association between cognitive functioning and PCS severity in the current study, further investigation into cognitive IIV in the context of mTBI may be beneficial. Ongoing complaints of cognitive difficulties as well as symptoms such as cognitive fatigue, even among individuals who have experienced a relatively good recovery, suggest the possible presence of a functional change that the standard means of investigating cognition (namely, neuropsychological test scores) may not be sufficiently sensitive to measure. The ideal domains to assess would likely be those known to be affected in the acute post-injury stage (e.g., processing speed, attention, working memory). Further, IIV may be a more appropriate measure of cognitive functioning (and possibly neurological integrity, as well) and it would be worthwhile to clarify how such variability in functioning may relate to recovery and persistent PCS. Additionally, in order to gain a better understanding of persistent PCS after mTBI, it would be beneficial to look into possible patterns or profiles of symptom-reporting. Awareness of PCS profiles may help to then clarify any differences in the roles that the neuropsychosocial factors may play in the process of recovery as well as further inform specific treatment targets and appropriate modalities. That being said, more research is needed on the benefits (and possible costs) of various types of interventions following mTBI. This is an area that remains relatively unexplored; as the current study suggests, applying the results of studies on severe TBI to these more mild injuries may not be appropriate and may, in fact, be detrimental to recovery. Similarly, longitudinal studies of mTBI that attempt to further understand recovery from a neuropsychosocial perspective would likely be of significant benefit to our understanding of the process of recovery and may further elucidate who is at greatest risk of developing persistent PCS, as well as when and how to intervene. Finally, when taken in the context of the existing literature, the current results highlight the need to thoroughly assess both subjective experience and personal beliefs across a broad range of variables. For example, assessing beliefs about the effects of, and recovery from, TBI could then allow investigation into how much convergence there is between these beliefs and the reported experience. Similarly, beliefs about cognitive performance may be more relevant than objective performance, or perceived group belonging may be more relevant than mere affiliation. Further, investigating these questions using qualitative methodology could provide a great deal of insight into the rich diversity of experiences and perspectives following mTBI, as well as commonalities between groups of survivors, which could help to guide future research. Addressing these research questions would be of great benefit to individuals following mTBI and provide much needed guidance to those who provide care during the process of recovery.

Conclusion

The current study has addressed a need within the literature by investigating mTBI and persistent post-concussion symptoms from a broad, 'whole-person,' neuropsychosocial perspective. It was argued that this perspective is the most appropriate from which to view PCS, and that a purely neuropathological or psychopathological perspective is insufficient to understand the multitude of factors that are relevant to the process of recovery after mTBI. In particular, the results of the current study highlight the importance of psychosocial factors after brain injury, such as anxiety and identity, and speaks to the complex interaction of individual and social elements that should be considered by clinicians working with individuals following mTBI.

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Appendix

Telephone Screening Interview (includes the IES-R)

Center for Epidemiologic Studies Depression Scale

Collective Self-Esteem Scale for TBI – Identity Subscale

Head Injury Semantic Differential Scale – III

Neurobehavioural Symptom Inventory

Numerical Pain Rating Scale

State-Trait Anxiety Inventory (sample questions)

Background Demographics Questionnaire

TBI Social Identity Questionnaire

Persistent PCS Telephone Screen
(S. Ross & R. Skelton)

Introduction
Hello. You had recently called our lab at the University of Victoria because you were interested in participating in our study looking at recovery after brain injury. An appointment for a telephone interview was scheduled with you, and this is the time that was arranged. Is this still a good time to talk? The interview will take about 30 minutes.

Yes *No* *(If no, rescheduled date & time:_______________)*

If yes: Great. First, thank you for your interest in our study! The purpose of this phone call is to complete a screening interview, which will help us learn a bit more about you and the injury that you experienced. We are looking for people with specific characteristics to participate in this study; your answers to today's questions will help us determine whether or not you are a good fit for this study. However, it is important for you to know that I'll be asking you some questions about how you got your brain injury, how you felt immediately after the injury, a bit about your personal and medical history, as well as some questions about current thoughts or feelings that you may be having in reaction to the accident that caused your injury. Some of these questions may make you sad, angry, anxious, frustrated or maybe even embarrassed. Please remember that you can skip any questions that you don't want to answer or even discontinue the interview at any time if you choose – no explanation required.

Before we begin, I would like to tell you how we will handle the information that you share during this phone call. The information that you share will be part of your research file which will be kept confidential You will be assigned a participant number which will be used instead of your name and your name and other identifying information will be kept separate from your actual responses. If you don't end up being a good fit for the study for one reason or another, we would still like to keep the information that you provide today in order to create statistics about how many people are interested in the study as compared to how many people enrol. But we will immediately remove anything that could be used to identify you and we will make the data completely anonymous.

Before we go on, do you have any questions about any of this?

If you have any questions during our phone call today, please don't hesitate to ask.

If you are comfortable, do I have your permission to proceed? *Yes* *No*

You can decline to answer any question or stop at any time if you decide you are not comfortable.

Demographic Information

Date of Birth: _______/_______ Age: ________ Gender: M F
(month) (year)

Is English your first language? Y N
If "no," at what age did you learn English? _________
What is the primary language spoken in your home? ______________________

Brief Study Description
This study is being conducted by myself, Ms. Stacey Ross, a graduate student in clinical neuropsychology at the University of Victoria. I am supervised by Dr. Colette Smart, a professor in the Department of Psychology at the University of Victoria.

If you participate in the full study, after this phone interview we will schedule a face-to-face meeting during which you will be asked to complete a few different tasks. These tasks will all be completed during one appointment in our lab at the University of Victoria or, if you can't make it to the university, at an appropriate location that is near you, like a library. During this visit, after you consent to participate in the study, you will complete some paper and pencil measures of your mood, current situation, and thinking abilities. These measures include questionnaires and standardized neuropsychological assessment tools. Your participation should take about 90 minutes (plus breaks, which will be provided as needed).

Do you have any questions about the study?

Screen Continued
If you think that you may like to participate in this study, I'd like to ask you some more questions. Do you want to continue with the phone screen?

When we first spoke, I asked you if you had a person who knows you well who could answer some questions about your brain injury. Did they agree to be contacted?
Y N *(if yes, ask the following questions)*

What is their name? _______________________________
What is your relationship with them? ____________________________
How long have you known them? __________________
Phone number or email address: __________________________________
Are you ok with someone from our lab contacting *(Informant)* to ask them a few questions about your brain injury? Y N

Brain Injury Related Demographics
(if more than 1 brain injury, record info for all; note most recent)

For this study, we are looking for people who have had a mild brain injury, which is sometimes called a concussion – they are basically the same thing. A mild brain injury can occur when someone has some type of accident where either their head gets hit or their head gets shaken around a lot (like in a car accident or during contact sports). Right after the accident, the person may feel confused or disoriented (not know where they are, what time it is, what they're doing) and have a headache, blurry or double vision, trouble speaking or understanding what others are saying, balance problems or feel dizzy, feel sick or even throw-up. The person may also lose consciousness, or pass out, for a few minutes and, later, have trouble remembering what happened right before or right after the accident. A mild brain injury is thought to have occurred if a person experiences any of these symptoms after hitting their head. If you experienced any of these symptoms, AND you were unconscious for more than a few minutes, or were in a coma, then this would be considered a moderate or severe brain injury.

Have you ever experienced something like this that would be considered a mild brain injury or a concussion? Y N

If no: Unfortunately, to take part in this study you have to have had a mild brain injury or concussion. Would you be willing to have us keep your name and information on file in case another study comes up that you could participate in? Y N
Thank-you for your interest in our study. Have a nice day.

If yes: Have you ever had a more severe brain jury? That is, have you ever been knocked unconscious for more than just a few minutes? Y N

If yes: How long were you out (or unconscious) for? __________
How do you know? ____________________________________

How long were you hospitalized for the brain injury? ____________
(clarify that hospitalization was due to the brain injury and not solely due to other injuries, e.g., orthopaedic, internal organ, etc.)

*If **unconscious >30 minutes**, or **hospitalized >1 night:***
Unfortunately, to take part in this study you have to have had a mild brain injury or concussion and it sounds like your injury was more than just mild. Would you be willing to have us keep your name and information on file in case another study comes up that you could participate in? Y N

Thank-you for your interest in our study. Have a nice day.

*If **no history of moderate/severe TBI**:*
How many mild brain injuries/concussions have you had? __________________

When did your brain injury/injuries occur? ______________________________
____

How did you get your brain injury/injuries? ______________________________
____

Did you go to the hospital/emergency room after your injury? Y N
If yes, were you admitted to the hospital? Y N
If yes, how long were you in the hospital? ____________________

Do you have any loss of memory for events before the injury? Y N D/K
(For example, do you remember what you were doing a few hours before the incident? A few minutes or seconds before?)
What is the last thing you remember before the event? ____________________
How long before the incident did this happen (approx)? ____________________

Do you have any loss of memory for events after your injury? Y N D/K
(For example, do you remember what happened immediately after the incident? What happened a few hours after the incident?)

What is the first thing you remember after the incident? ____________________
How long after the incident did this happen (approx)? _____________________

Did you lose consciousness at the time of the injury? Y N D/K
If yes, how long were you out (or unconscious) for? __________________

How do you know? ____
____

Were you intoxicated or under the influence of a substance at the time of the incident?
Y N

Are you currently involved in litigation related to your injury? Y N

Now I'd like to ask you some more general questions about your health, functioning and background:

Overall, how would you describe your health? ____________________________

Have you had any major medical problems or surgeries? Y N
If yes: What and when? ____

Do you have any illnesses or disorders that can or have affected your brain (e.g., stroke, pre-existing epilepsy, Parkinsons, Multiple Sclerosis, Lupus, Diabetes)? Y
N

If yes: What is it? ____

How much alcohol do you drink per week? _______________________________

Have you ever had a problem with alcohol? Y N

Do you use any illicit substances (i.e., drugs) or do you take more than prescribed of any prescription medications? Y N

If yes: Can you tell me a little more about this? What substances do you take and how often? ____

Have you ever been diagnosed with a psychiatric disorder? Y N
(For example: depression, anxiety, PTSD, schizophrenia, a personality disorder)
If yes, what disorder? ____
Have you ever taken medication to treat a psychiatric disorder? Y N

Impact of Events Scale – Revised (IES-R) (Weiss & Marmar, 1997)

Now I will read you a list of difficulties people sometimes have after stressful life events. After I read each item, please indicate how distressing each difficulty has been for you during the last seven (7) days with respect to the events that led to your brain injury/concussion, or in fact any other very stressful or traumatic event that you still think about. How much were you distressed or bothered by these difficulties? Please remember, if you find any of these questions distressing, we can stop right away.

	Not at all	A little bit	Moder -ately	Quite a bit	Extrem -ely
Any reminder brought back feelings about it.	0	1	2	3	4
I had trouble staying asleep.	0	1	2	3	4
Other things kept making me think about it.	0	1	2	3	4
I felt irritable and angry.	0	1	2	3	4
I avoided letting myself get upset when I thought about it or was reminded of it.	0	1	2	3	4
I thought about it when I didn’t mean to.	0	1	2	3	4
I felt as if it hadn’t happened or wasn’t real.	0	1	2	3	4
I stayed away from reminders of it.	0	1	2	3	4
Images of it popped into my mind.	0	1	2	3	4
I was jumpy and easily startled.	0	1	2	3	4
I tried not to think about it.	0	1	2	3	4
I was aware that I still had a lot of feelings about it, but I didn’t deal with them.	0	1	2	3	4
My feelings about it were kind of numb.	0	1	2	3	4
I found myself acting or feeling as though I was back at that time.	0	1	2	3	4
I had trouble falling asleep.	0	1	2	3	4
I had waves of strong feelings about it.	0	1	2	3	4
I tried to remove it from my memory.	0	1	2	3	4
I had trouble concentrating.	0	1	2	3	4
Reminders of the event caused physical reactions like sweating, difficulty breathing, nausea, or palpitations.	0	1	2	3	4
I had dreams about it.	0	1	2	3	4
I felt watchful or on-guard.	0	1	2	3	4
I tried not to think about it.	0	1	2	3	4

Final Statement

Thank you so much for answering all of my questions. As I explained, we are looking for specific individuals to participate in this study. The information that you provided during this phone call will be used to determine if you will be eligible to participate in this study. We will review your information and get back to you as soon as we can regarding participation. Please know that we will keep everything you have told us confidential, regardless of whether you are able to participate or not. Do you have any questions for us at this time?

____

Significant Other/Corraborating Interview

I am calling from the University of Victoria. Your phone number was provided to us by _(Participant's name)_ who may participate in a study that we are conducting. Our study is investigating different factors that may be important to recovery after mild brain injuries. We would like to talk to you to get a bit of information about (*Participant's)* brain injury. You will not be considered a participant in this study, and are instead an informant. We won't be collecting any additional personal information from or about you, and the information that we do have (your name and phone number) will remain confidential and not be shared with anyone. Also, because no identifying information about *(Participant)* will ever be disclosed, you will also be unidentifiable. Finally, we will not share your responses with (*participant)* or anyone else,

Do you have any questions about this?

Are you comfortable proceeding? *Y* *N*

If yes: Would you be willing to answer some questions about *(Participant)* ? *Y* *N*

> *If no:* Ok. Well thank-you very much for your time. If you change your mind, please give us a call at 250-721-7552 or email tbistudy@uvic.ca
>
> *If yes:* Do you have a few minutes to answer these questions now? *Y* *N*
>
> > *If no:* When would be a better time to call? __________________
> >
> > *If yes:* Great! *(Proceed to questions below)*

If yes, continue with interview:
What is the nature of your relationship with *(Participant)*? ____________________

How long have you known *(Participant)*? _________________________________

I am interested in what you remember about (*Participant's*) brain injury or concussion.

As I mentioned, in this study we are looking for people who have had a mild brain injury, which is sometimes called a concussion – they are basically the same thing. A mild brain injury can occur when someone has an accident that may cause some damage to their brain either through their head getting hit or significant shaking of their head (like in a car accident or during contact sports). Right after the accident, the person may appear confused or disoriented (not know where they are, what time it is, what they're doing); they may complain of a headache, blurry or double vision; they may have trouble speaking or seem to have trouble understanding what others are saying; they may have balance problems or say that they feel dizzy, feel sick or they may even throw-up. The person may also lose consciousness, or pass out, for a few minutes and, later, they have trouble remembering what happened right before or right after the accident. A mild brain injury or concussion is thought to have occurred if a person experiences any of these symptoms. If (*participant*) experienced any of these symptoms, AND they were unconscious for more than a few minutes, or were in a coma, then this would be considered a moderate or severe brain injury.

Do you know if (*participant*) ever experienced symptoms consistent with a mild brain injury or concussion after an accident? Y N

> *If no:* Ok. We won't need any additional information from you at this time. Thank-you very much for your time though.
>
> *If yes:* Has (*participant*) ever had a more severe brain jury? That is have they ever been knocked unconscious for more than just a few minutes? *Y N*
>
> > *If yes:* How long were they out (unconscious) for? ____________
> > How long were they hospitalized for the brain injury? ___________
> > *(clarify that hospitalization was due to the brain injury and not solely due to other injuries, e.g., orthopaedic, internal organ, etc.)*
> >
> > > *If* ***unconscious >30 minutes****, or* ***hospitalized >1 night****, skip to "Thank You" at the end of the script.*

If no history of moderate/severe TBI:
Did you know [*participant*] when he/she got their concussion? *Y N*

Do you remember how [*participant*] was around the time of their concussion?
Y N

> *If no:* Ok. We won't need any additional information from you at this time. Thank-you very much for your time though.

What were the circumstances around *(Participant)*'s injury? In other words, how did they sustain their injury? ____

Did *(Participant)* go to the hospital/emergency room after their injury? *Y N D/K*
If yes: Was *(Participant)* admitted to the hospital? *Y N D/K*
If yes: How long was *(Participant)* in the hospital? _______________

Did *(Participant)* have any memory loss for events before the injury? Y N D/K
(For example, what s/he was doing a few hours before the incident? A few minutes or seconds before?)

Did *(Participant)* have any memory loss for events after the injury? Y N D/K
(For example, does s/he remember what happened immediately after the incident? What happened a few hours after the incident?)

Did *(Participant)* lose consciousness at the time of the injury? Y N D/K
Did *(Participant)* report any symptoms in the minutes or hours after his/her injury?
Please list:
____
____
____

Thank you so much for answering all of my questions. The information that you provided during this phone call will be helpful in determining if *(Participant)* **will be eligible to participate in this study. Please know that we will keep everything you have told us confidential, regardless of whether (*participant*) is able to participate or not. Do you have any questions for us at this time?**

Center for Epidemiologic Studies – Depression Scale (CES-D)

(Radloff, 1977; Eaton, Muntaner, Smith, Tien, & Ybarra, 2004)

The following questions are about ways that you might have felt or behaved recently. Please indicate how often you have felt or behaved this way during the past week:

	Rarely / None of the time (<1 day)	Some / A little of the time (1-2 days)	Occasionally / Moderate amount of the time (3-4 days)	Most / All of the time (5-7 days)
I was bothered by things that usually don't bother me.	☐	☐	☐	☐
I did not feel like eating; my appetite was poor.	☐	☐	☐	☐
I felt that I could not shake off the blues even with help from my family or friends.	☐	☐	☐	☐
I felt that I was just as good as other people.	☐	☐	☐	☐
I had trouble keeping my mind on what I was doing.	☐	☐	☐	☐
I felt depressed.	☐	☐	☐	☐
I felt that everything I did was an effort.	☐	☐	☐	☐
I felt hopeful about the future.	☐	☐	☐	☐
I thought my life had been a failure.	☐	☐	☐	☐
I felt fearful.	☐	☐	☐	☐
My sleep was restless.	☐	☐	☐	☐
I was happy.	☐	☐	☐	☐
I talked less than usual.	☐	☐	☐	☐
I felt lonely.	☐	☐	☐	☐
People were unfriendly.	☐	☐	☐	☐
I enjoyed life.	☐	☐	☐	☐
I had crying spells.	☐	☐	☐	☐
I felt sad.	☐	☐	☐	☐
I felt that people dislike me.	☐	☐	☐	☐
I could not get “going”.	☐	☐	☐	☐

CSE – Identity (CSE-TBI-I)

Please read each of the statements below and circle the number that best represents how much you personally **agree** with each one.

1	**2**	**3**	**4**	**5**	**6**	**7**
Strongly Disagree	**Disagree**	**Mildly Disagree**	**Neither / Neutral**	**Mildly Agree**	**Agree**	**Strongly Agree**

Having a brain injury is an important part of my self-image.

1 2 3 4 5 6 7

Having a brain injury is unimportant to my sense of what kind of person I am.

1 2 3 4 5 6 7

Having a brain injury is an important reflection of who I am.

1 2 3 4 5 6 7

Having a brain injury has very little to do with how I feel about myself.

1 2 3 4 5 6 7

Semantic Differential Scale (HISDS)

Below is a list of word pairs. Each pair forms a continuum (or range) of personal attributes or characteristics.

Please read each word in the pair and indicate where, in general, *you* believe you fell on the following dimensions **in the past** (before your brain injury).

	Extremely	Quite	Slightly	Neither	Slightly	Quite	Extremely	
Bored	____	____	____	____	____	____	____	Interested
Unhappy	____	____	____	____	____	____	____	Happy
Helpless	____	____	____	____	____	____	____	In Control
Worried	____	____	____	____	____	____	____	Relaxed
Dissatisfied	____	____	____	____	____	____	____	Satisfied
Unattractive	____	____	____	____	____	____	____	Attractive
Unhopeful	____	____	____	____	____	____	____	Hopeful
Lack Confidence	____	____	____	____	____	____	____	Self-Confident
Emotional	____	____	____	____	____	____	____	Stable
Worthless	____	____	____	____	____	____	____	Valuable
Forgetful	____	____	____	____	____	____	____	Mindful
Irritable	____	____	____	____	____	____	____	Calm
Unfeeling	____	____	____	____	____	____	____	Caring
Clumsy	____	____	____	____	____	____	____	Agile
Dependent	____	____	____	____	____	____	____	Independent
Inactive	____	____	____	____	____	____	____	Active
Difficult	____	____	____	____	____	____	____	Cooperative
Withdrawn	____	____	____	____	____	____	____	Talkative
Unfriendly	____	____	____	____	____	____	____	Friendly
Stupid	____	____	____	____	____	____	____	Clever

Below in the same list of word pairs. This time, please read each word in the pair and indicate where, in general, ***you*** believe you fall on the following dimensions **now** (since your brain injury).

	Extremely	Quite	Slightly	Neither	Slightly	Quite	Extremely	
Bored	____	____	____	____	____	____	____	Interested
Unhappy	____	____	____	____	____	____	____	Happy
Helpless	____	____	____	____	____	____	____	In Control
Worried	____	____	____	____	____	____	____	Relaxed
Dissatisfied	____	____	____	____	____	____	____	Satisfied
Unattractive	____	____	____	____	____	____	____	Attractive
Unhopeful	____	____	____	____	____	____	____	Hopeful
Lack Confidence	____	____	____	____	____	____	____	Self-Confident
Emotional	____	____	____	____	____	____	____	Stable
Worthless	____	____	____	____	____	____	____	Valuable
Forgetful	____	____	____	____	____	____	____	Mindful
Irritable	____	____	____	____	____	____	____	Calm
Unfeeling	____	____	____	____	____	____	____	Caring
Clumsy	____	____	____	____	____	____	____	Agile
Dependent	____	____	____	____	____	____	____	Independent
Inactive	____	____	____	____	____	____	____	Active
Difficult	____	____	____	____	____	____	____	Cooperative
Withdrawn	____	____	____	____	____	____	____	Talkative
Unfriendly	____	____	____	____	____	____	____	Friendly
Stupid	____	____	____	____	____	____	____	Clever

Neurobehavioural Symptom Inventory (NSI)

(Cicerone, 1995)

Please rate how much the following symptoms have disturbed or bothered you in the **LAST *2 weeks*** and circle the number that best corresponds to your answer:

0 = **None** – Rarely if ever present; not a problem at all.

1 = **Mild** – Occasionally present, but it does not disrupt my activities; I can usually continue what I'm doing; doesn't really concern me.

2 = **Moderate** – Often present, occasionally disrupts me activities; I can usually continue what I'm doing with some effort; I feel somewhat concerned.

3 = **Severe** – Frequently present and disrupts activities; I can only do things that are fairly simple or take little effort; I feel I need help.

4 = **Very Severe** – Almost always present and I have been unable to perform at work, school or home due to this problem; I probably cannot function without help.

Feeling dizzy	0	1	2	3	4
Loss of balance	0	1	2	3	4
Poor coordination, clumsy	0	1	2	3	4
Headaches	0	1	2	3	4
Nausea	0	1	2	3	4
Vision problems, blurring, trouble seeing	0	1	2	3	4
Sensitivity to light	0	1	2	3	4
Hearing difficulty	0	1	2	3	4
Sensitivity to noise	0	1	2	3	4
Numbness or tingling on parts of my body	0	1	2	3	4
Change in taste and/or smell	0	1	2	3	4
Loss of appetite or increased appetite	0	1	2	3	4
Poor concentration, can't pay attention, easily distracted	0	1	2	3	4
Forgetfulness, can't remember things	0	1	2	3	4
Difficulty making decisions	0	1	2	3	4
Slowed thinking, difficulty getting organized, can't finish things	0	1	2	3	4
Fatige, loss of energy, getting tired easily	0	1	2	3	4
Difficulty falling or staying asleep	0	1	2	3	4
Feeling anxious or tense	0	1	2	3	4
Feeling depressed or sad	0	1	2	3	4
Irritability, easily annoyed	0	1	2	3	4
Poor frustration tolerance, feeling easily overwhelmed by things	0	1	2	3	4

Numerical Pain Rating Scale

Rate how intense your pain is right now, in this moment, on a scale from 0 to 10 – where '0' equals no pain at all, and '10' equals the most severe pain you have ever experienced in your life. Where would you say that your pain is on that scale right now?

0	1	2	3	4	5	6	7	8	9	10
No Pain										*Extreme Pain*

State-Trait Anxiety Inventory (STAI)
SAMPLE ITEMS

State (STAI-S):

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel *right* now, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	Not at all	Somewhat	Moderately So	Very Much So
I feel calm	1	2	3	4
I feel upset	1	2	3	4
I feel comfortable	1	2	3	4
I feel confused	1	2	3	4

Trait (STAI-T):

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you *generally* feel.

	Almost Never	Sometimes	Often	Almost Always
I feel nervous and restless	1	2	3	4
I feel rested	1	2	3	4
I make decisions easily	1	2	3	4
I feel inadequate	1	2	3	4

Demographic Information Form

Handedness: Right Left Both

Education (in years): ______________________________

Mother's ed: ______________________________

Father's ed: ______________________________

Problems in school or diagnosed with a learning disability? Yes No

____

____

Diagnosed with ADD/ADHD? Yes No

Working/student at the time of the injury? Yes: ________________________

No: ___ Retired? ___ Disability?

Currently working or a student? Yes: ________________________

No: ___ Retired? ___ Disability?

Living arrangement at time of injury?

___ Alone
___ Spouse/Partner
___ Parent
___ Family member (not parent)
___ Friend/Roommate
___ Other? (_________________)

Current living arrangement?

___ Alone
___ Spouse/Partner
___ Parent
___ Family member (not parent)
___ Friend/Roommate
___ Other? (_________________)

TBI – Social Identity Questionnaire (TBI-SIQ)

Please read each of the following questions and, for each one, circle the *ONE* response that best applies to you.

Have you ever been involved in a group for individuals with brain injury?
(e.g., support group, social group)

YES NO

How long did you regularly attend the group(s)?

Did not attend / Less than 1 month	1–3 months	3-6 months	6-12 months	More than 12 months

Have you ever attended a formal rehabilitation program related to your brain injury? (e.g., VGH Outpatient Neurorehab Program)

YES NO

How long did you regularly attend the program(s)?

Did not attend / Less than 1 month	1–3 months	3-6 months	6-12 months	More than 12 months

Have you ever been involved in any other type of group or program for people with brain injury?

YES NO

How long did you regularly attend?

Did not attend / Less than 1 month	1–3 months	3-6 months	6-12 months	More than 12 months

In the past 3 months, how many times have you attended any brain injury-centered group or program?

None	1 – 9	10 – 19	20 – 29	30 or more

How supported have you felt by these groups/programs or the other people involved in them?

Not at all	A little bit / Not much	Moderately / Somewhat	Quite a bit / Very much	Extremely

How many of your friends and/or family members have had a brain injury?

None	1	2 – 3	4 – 5	6 or more

Do you find it difficult to socialize with people who have never had a brain injury?

Not at all	A little bit / Not much	Moderately / Somewhat	Quite a bit / Very much	Extremely

Do you feel that you belong to the larger community of “brain injury survivors”?

Not at all	A little bit / Not much	Moderately / Somewhat	Quite a bit / Very much	Extremely

Overall, how important is this feeling of “belonging” to you currently?

Not at all	A little bit / Not much	Moderately / Somewhat	Quite a bit / Very much	Extremely

Some people readily share with others the fact that they have sustained an injury, while other people do not share this information. Additionally, some people prefer to use the term “brain injury”, others prefer the term “concussion”, while others will use there terms interchangeably or depending on the situation/context that they are in at the time. With that in mind:

How comfortable are you sharing with others that you have had a concussion?

Not at all	A little bit / Not much	Moderately / Somewhat	Quite a bit / Very much	Extremely

How comfortable are you sharing with others that you have had a brain injury?

Not at all	A little bit / Not much	Moderately / Somewhat	Quite a bit / Very much	Extremely

Do you think that other people categorize – or “see” - you as a person with a brain injury, or as a brain injury survivor?

Not at all	A little bit / Not much	Moderately / Somewhat	Quite a bit / Very much	Extremely

We all belong to a number of groups and have multiple roles at any point in time. This includes groups/roles that we are born into (e.g., sex or gender, race, culture and family groups; roles of child, sibling, etc.) and groups/roles that we “join” or “acquire” later (e.g., groups based on our beliefs, activities, hobbies; roles such as friend, spouse/partner, parent, patient, or based on our job/career, etc.).

These groups and roles contribute to our identity – how we see ourselves, how we act, and how others may see us. The importance of these groups and roles can change over time, and we can have roles that are very relevant to our identity at specific points in time but irrelevant, or not applicable, at others (e.g., student or patient). Also, the importance or salience of one's current groups/roles to their identity can change depending on what they are doing, who they are with, or what is happening around them.

Overall, which of your current groups or roles do you feel is the most important to who you are? ____