A literature review examining the unique and shared postconcussive symptoms in mild traumatic brain injuries and posttraumatic stress disorder

By

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A thesis submitted to the School of Community Services in partial fulfillment of the requirements for the degree of Bachelor of Applied Arts in Behavioural Psychology

St. Lawrence College
Kingston, Ontario
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March 10, 2014
Abstract

There has been a divide among professionals regarding the nature of postconcussive symptoms (PCS) and whether these symptoms are a product of psychological damage (PTSD) or organic damage (brain injury). The purpose of this thesis and literature review is to examine the etiology of PCS, specifically the role of mild traumatic brain injuries and posttraumatic stress disorder. Peer-reviewed journal articles were accessed from online databases open to St. Lawrence College, Queen’s University, and Providence Care. Relevant articles were read and summarized in a table format (see Appendix A). Current literature suggests that PCS is not specific to concussions, and are exhibited across many populations, including those with substance abuse issues, depression, and otherwise healthy university students without head injuries. It appears that psychiatric factors, such as stress and anxiety, accounted for most reported PCS. Further, the symptoms in PCS are not sufficient enough to make-up a diagnosis. This is reflected by the refusal of PCS as a diagnosis in the DSM 5. However, certain postconcussive symptoms such as headaches, dizziness, balance issues, and visual deficits are more attributable to mild traumatic brain injuries, while symptom clusters of avoidance and re-experiencing are more attributable to PTSD. It is important to note that these conclusions are mixed within the literature. It is recommended that, for future research, professionals concerned with brain injuries focus on reaching a consensus for a definition of brain injuries. Those interested in researching PCS should assess veterans for PCS before and after deployment to better understand the impact of traumatic events and brain injuries.
Acknowledgements

Yolanda
Martin
Jeff
Neil
Drew

I would like to thank my college supervisor, Dr. Yolanda Fernandez, for her insightful, and much needed feedback and guidance over the nearly 8 months of writing this thesis. It is an understatement to say that I am very happy you were my supervisor.

Thanks to Jeff O’Neill, my agency supervisor, for taking me on as a placement student and giving me such great opportunities to gain clinical experience.

Thanks to Dr. Martin Logan for setting me in the right direction and taking a genuine interest in the work I was doing.

Thanks to Neil Muchmore for always letting me drop in and bombard you with questions and advice, and including me for additional clinical experiences whenever you could.

Thanks to Dr. Andrew McNamara, my second reader, for giving me necessary feedback to improve my thesis for publication purposes.

Thanks to all the other professionals at Regional Community Brain Injury Services; we were strangers, but you welcomed me.

I consider myself extremely blessed that I was able to work with all of you.
# Table of Contents

Chapter 1: Introduction ............................................................................................................. 1  
Chapter 2: Literature Review .................................................................................................... 3  
  mTBI Epidemiology ................................................................................................................... 3  
  PTSD Epidemiology .................................................................................................................. 4  
  PCS as a Diagnosis ................................................................................................................... 5  
  Overlapping and Unique Postconcussive Symptoms ............................................................... 7  
  Developmental Risk Factors .................................................................................................... 11  
Chapter 3: Methodology ......................................................................................................... 16  
Chapter 4: Results .................................................................................................................. 17  
Chapter 5: Discussion ............................................................................................................. 19  
  Implications of Results ............................................................................................................ 19  
  Suggestions and Recommendations ......................................................................................... 20  
  Strengths and Limitations ......................................................................................................... 21  
References .................................................................................................................................. 22  
Appendices ............................................................................................................................... 26  
  Appendix A: Method – Summarized Articles ......................................................................... 26
Chapter 1: Introduction

Since the beginning of Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF) in 2001, almost 2 million U.S. troops have been deployed overseas (Polusny et al., 2011). The two military conflicts have regenerated an interest in how combat affects mental health, with two issues being at the forefront of concern: mild traumatic brain injury (mTBI), also referred to as concussion, and posttraumatic stress disorder (PTSD) (Tanielian & Jaycox, 2008; Vasterling, Brailey, Proctor, Kane, & Heeren, 2012). The literature surrounding both of these diagnoses has increased, with specific consideration of the overlap of PTSD symptoms and the residual and persistent symptoms of a concussion, or mild traumatic brain injury (mTBI) (Carlson et al., 2009). While most individuals with mild traumatic brain injuries, or concussions, recover to pre-injury functioning levels 1-3 months post injury (Carlson et al., 2009), a group of individuals continue to report cognitive, emotional, and physical complaints for upwards of a year (Benge, Pastorek, & Thornton, 2009). Collectively, these symptoms are called postconcussion syndrome (PCS), or Postconcussive symptoms, within the literature (Benge et al., 2009). Though the DSM-IV-TR Task Force (2000) refused the proposal to accept PCS as a diagnosis due to insufficient evidence, the American Psychiatric Association (APA, 2000) describes the main feature of PCS as “an acquired impairment in cognitive functioning, accompanied by specific neurobehavioral symptoms, that occurs as a consequence of closed head injury of sufficient severity to produce a significant cerebral concussion” (p. 760). Therefore, this paper will use the term PCS to refer to postconcussive symptoms and not a syndrome until evidence suggests otherwise. The diagnosis of PCS will be further discussed in the literature review, and subsequent sections.

Although postconcussive symptoms are associated with mild traumatic brain injuries, current research demonstrates that PCS are also closely associated with PTSD as well (McCrea, 2008). According to Stein and McAllister (2009), a professional divide exists as to whether these symptoms are psychological or biomechanical in nature; with mental health professionals considering trauma as an extreme emotional reaction to a threatening event, while neurologists and neurosurgeons consider trauma to signify destructive forces affecting the brain and other parts of the body. Essentially, the etiology of postconcussive symptoms is unknown, which is where the controversy begins. If PCS are better explained from biomechanical trauma, a traumatic brain injury, then it is possible that mTBI may play a significant role in the causation of PTSD. If PCS are better explained from psychological trauma, then PCS may be separate from mTBI altogether and simply co-occur. A clearer understanding of PCS may guide treatments from both mental health services and brain injury services, potentially resulting in more effective interventions. Additionally, understanding what PCS is should help guide research and literature.

According to Lung (2007), there are many mental health professionals in the civilian population that do not receive post-graduate training in mTBI and have difficulty distinguishing the presentation of mTBI from PTSD (as cited in Jones, Young, & Leppma, 2010). Misinterpreting these symptoms has significant treatment consequences, such as medication side effects from prescribing unwarranted medications, and failed interventions that could not address the underlying issue (Hoge, Goldberg, & Castro, 2009). Understanding the difference between individuals experiencing consequences associated with a psychiatric illness, such as PTSD, and individuals coping with symptoms directly associated with mTBI, is crucial for providing appropriate treatment (Arbisi, Polusny, Erbes, Thuras, & Reddy, 2011).
The purpose of this paper is to examine literature pertaining to the role of postconcussive symptoms in mTBI and PTSD, specifically, whether PCS are better explained by psychological or biomechanical trauma, and which symptoms under the label of PCS are unique and common to mTBI and PTSD. This paper seeks to clarify the issues related to these diagnoses through examining the commonalities and differences of epidemiology, symptom overlap, and developmental risk factors. The following thesis will have several different sections: a literature review, methodology, results, and discussion.

During the literature review, the purpose of the epidemiology review is to provide background information relevant to the diagnoses of traumatic brain injury and posttraumatic stress disorder, as well as definitions of relevant concepts. For example, while this literature review focuses on mTBI and PTSD, it is also important for practitioners to understand moderate and severe traumatic brain injuries (TBI) as well as acute stress disorder (ASD). Several articles, specifically in the traumatic brain injury literature, do not specify the severity of the brain injury. Secondly, empirical research regarding overlapping and distinct symptoms will be reviewed, with a focus on the nature of PCS. Again, this literature review focuses on mTBI and PTSD, but may include some literature regarding other severities of TBI and other psychiatric disorders, such as depression. Thirdly, developmental risks will be reviewed for both mTBI and PTSD, but with a focus on postconcussive symptoms. The method section will outline how the above research was gathered. The results section will provide a brief summary of the main findings from the literature review, and the discussion section will discuss implications of the results, highlight strengths and limitations, as well as provide recommendations and suggestions for future research on this subject.
Chapter 2: Literature Review

mTBI Epidemiology

TBI involves damage to the brain by external and mechanical forces that result in neural damage (Vasterling, Verfaellie, & Sullivan, 2009) versus head injury, which is injury to the head without brain damage. Brain injury is usually inferred by symptoms or signs immediately post-injury (Vasterling et al., 2009). These signs and symptoms are then used as a basis to categorize traumatic brain injury, ranging from mild, and moderate, to severe (Kennedy et al., 2007). Many definitions have been used to define mild traumatic brain injury without absolute consensus among professionals (Kennedy et al., 2007). However, the American Congress of Rehabilitation Medicine (Kay et al., 1993) has proposed a definition that has generally been accepted (Carroll et al., 2004; Kennedy et al., 2007). For an individual to have a mild traumatic brain injury, they must have experienced disruption of brain function from physical assault to the head, manifested by at least one of the following: 1) a period of loss of consciousness, (2) loss of memory of the event, either before or after, (3) an alteration in mental state after the accident, or (4) neurological deficits that last for any duration (Kay et al., 1993). According to Kay et al. (1993), a loss of consciousness (LOC) less than 30 minutes, posttraumatic amnesia (PTA) less than 24 hours, and a Glasgow Coma Scale (GCS) score of between 13 and 15 define the severity of mTBI. Moderate traumatic brain injuries are usually defined as a LOC from 30 minutes to 24 hours, PTA ranging from 1 to 7 days, and a GCS rating between 9 and 12 (Bryant, 2011). Severe traumatic brain injury is usually defined as LOC, GCS, and PTA greater than moderate traumatic brain injury on all items (Bryant, 2011). Physical assault to the head includes an object striking the head, the head striking an object, and/or extreme movements of the head, such as whiplash (Kay et al., 1993). Immediate postconcussive symptoms after injury include but are not limited to “headaches, dizziness, nausea, vomiting, drowsiness, blurred vision, diplopia, irritability, reduced tolerance for frustration, anxiety, moodiness or depression, poor concentration, forgetfulness, insomnia, fatigue, poor hearing, hypersensitivity to noise, appetite changes, decreased coordination, and difficulties thinking clearly” (Trahan, Ross, & Trahan, 2001, p. 436). Typically, the severity of symptoms increases with the severity of the brain injury. Likewise, neuroimaging (e.g., CT or MRI) may reveal neural insult of more severe TBIs but does not reveal brain damage of mild traumatic brain injuries (Vasterling et al., 2009).

Estimates suggest that 1.7 million Americans experience a traumatic brain injury (Faul, Xu, Wald, & Coronado, 2010). While this estimate is meant to reflect the incidence of TBI in the civilian population, it does not include the military population. Considering the deployed veterans in the Iraq and Afghanistan wars, the incidence of traumatic brain injuries is likely higher than this estimate (Morissette et al., 2011). Within the past 10 years of military intervention in the Middle East, researchers estimate that approximately 220,000 – 300,000 veterans have sustained a mild traumatic brain injury (Scherer, Weightman, Radomksi, Davidson, & McCulloch, 2013; Tanielian & Jaycox, 2008). However, according to Hoge et al. (2009), roughly 66% of all reported concussions or mTBI cases from the study above are based on a positive answer to one question: Whether the individual was dazed or confused at the time of the injury. They argue that, because an alteration in consciousness during combat could result from a variety of responses to injury (e.g., sleep deprivation, stress), the figure above is actually an overestimation.
PTSD Epidemiology

The DSM-IV-TR (2000) describes PTSD as an anxiety disorder with four significant criteria: 1) responding with intense fear or hopelessness to a life threatening event; (2) revisiting the trauma through intrusive thoughts and/or nightmares; (3) avoidant behaviours, such as avoiding thoughts, feelings, and other stimuli that may cue reminders of the event; and (4) an increase in arousal, which may be manifested in sleep disruptions, concentration, and irritability. Withdrawal and emotional numbing are also included with avoidance cluster and the inability to remember the event (APA, 2000). In order to receive a diagnosis of PTSD, an individual must have at least one re-experiencing symptom, three or more avoidant symptoms, and at least two increased arousal symptoms (APA, 2000). These symptoms must persist for at least a month post-trauma (APA, 2000). In order to fill the gap between the traumatic event and receiving the diagnosis of PTSD, the diagnosis of Acute Stress Disorder (ASD) was created (Bryant, 2011). ASD is conceptualized by similar symptoms of PTSD that manifest immediately after the traumatic event (APA, 2000), with the exception of ASD’s emphasis on dissociative symptoms. The key feature of PTSD is characterized by revisiting the trauma through intrusive thoughts and/or nightmares (Jones et al., 2010). Although prevalence estimates of PTSD vary widely, it is believed that roughly 10-17% of veterans are diagnosed with PTSD (Sundin, Fear, Iversen, Rona, & Wessely, 2010). While it is well established that PTSD is related to military settings, other common traumas include natural disasters, physical assault, rape, witnessing violence, and/or learning about a loved one who experienced any of the above (Breslau, 2002).

Based on the definitions, one of the characteristics of a traumatic brain injury appears to be a lack of memory about the event, while in contrast one of the key features of PTSD is the persistent and chronic reminder of the event. Because of these definitions, it was once believed that the two diagnoses could not co-occur in one individual (Warden et al., 1997), but recent research is now indicating the two diagnoses can co-occur (Hoffman, Dikmen, & Temkin, 2012).
PCS as a Diagnosis

As stated in the introduction, there has been a considerable amount of research regarding the relationship between TBI and PTSD. Some of this research speaks to the apparent overlap of symptoms between mTBI and PTSD (Carlson et al., 2009). Although individuals who have sustained a mild TBI typically recover from symptoms within days to weeks after the injury (Carroll et al., 2004), 10% - 15% of these individuals experience on-going symptoms upwards of a year (Stein & McAllister, 2009). These longer-term symptoms are collectively referred to as post-concussive symptoms. Comparing 29 individuals with mTBI to 30 individuals with orthopedic injuries, those with mTBI reported significantly more PCS at the initial assessment in the emergency departments and follow-up thirty days later (Sheedy, Geffen, Donnelly, & Faux, 2006). Average Rivermead Post-Concussion Symptoms Questionnaire (RPQ) scores were 17.29 (14.32) for the mTBI group and 5.76 (5.44) for the orthopedic group 30 days after the assessment. Individuals in the mTBI group also performed significantly worse on neurocognitive tests at initial assessment, which were correlated with PCS. Further, individuals with mTBI performed significantly worse on balance tests than a balance control group in the initial assessment. This study suggests that individuals sustaining mild traumatic brain injuries report significantly more postconcussive syndrome symptoms than other injuries. This study also lends support to the idea that the reported symptoms are mediated by the neurocognitive deficits associated with mTBIs. However, when taking psychiatric illnesses into account, specifically PTSD or depression, practitioners are finding results inconsistent with the findings above.

A study by Landre, Poppe, Davis, Schmaus, and Hobbs (2006) assessed 37 mTBI subjects and 39 trauma subjects in emergency departments for cognitive performance, PCS, and mental health and found contradictory results. While the mTBI group performed significantly worse on all cognitive measures, consistent with Sheedy et al. (2006), there were no significant differences between reports of PCS. Cognitive performance was not related to pain severity or emotional distress, nor did it appear to affect self-reporting of PCS (Landre et al., 2006). Postconcussive symptoms, on the other hand were consistently related to emotional distress. Those with higher intensity PCS scored lower on the Short Form-36 Mental Health ratings (Landre et al., 2006). In other words, those with poorer mental health ratings were more likely to report more PCS. There was no follow-up to this study. Ponsford et al. (2012) conducted a similar study with individuals admitted to emergency departments with traumatic brain injuries and other traumatic injuries not resulting in brain injury. During the acute phase, or a couple days just after injury and the 1-week follow-up, individuals with mTBI reported more than double the postconcussive symptoms than a trauma control group (32.5 vs. 13.5 during acute phase, and 16 vs. 7.5 at 1 week follow-up). However, at the 3-month follow-up, both groups reported similar PCS scores due to a substantial decrease in symptom reporting from the mTBI group. The strongest predictors of postconcussive symptoms at this time were psychiatric factors. These findings suggest that mTBI does play a significant role in the development of PCS, but particularly in the early stages of injury. During the acute phases of injury, those with mild traumatic brain injuries reported nearly 3 times more postconcussive symptoms than their orthopedic countergroups (Sheedy et al., 2006; Ponsford et al., 2012). However, these PCS scores are no longer mediated by mTBI but appear to be maintained by other psychiatric conditions. According to Stein and McAllister (2009), usage of the term postconcussive symptoms assumes that said symptoms are consistently caused by head injuries. The above literature demonstrates that PCS can indeed result from mTBIs, but also demonstrates that
reports of these PCS are similar to those with non-head injuries thirty days after injury. According to Vasterling (2009), these symptoms, once traditionally to be associated with mTBI, are quite non specific, and can be found across many different injuries (McCrea, 2008). For example, during the acute phases of recovery, Lange, Iverson, and Franzen (2008) compared 104 individuals with mTBIs to 104 individuals diagnosed with substance abuse using neuropsychological tests, and found that the cognitive deficits associated with mTBI were also present among individuals diagnosed with substance abuse. Further, there was no statistical difference in scores between the mTBI group and the substance abuse group on any of the eight cognitive measures (Trail Making Test part A and part B, and subtests from the Wechsler Memory Scale-Revised: digit span forward, digit span backward, logical memory 1, logical memory 2, visual reproduction 1, and visual reproduction 2).

Wang, Chan, and Deng (2006) also found that healthy university students reported symptoms of PCS. When looking at overall PCS scores indicated by the RPQ, it appears that PCS was not overall evident in the students. However, these students did report high frequencies of several specific symptoms: fatigue (76.9%), longer time to think (60.3%), poor concentration (58.7%), sleep disturbance (50.4%), and frustration and impatience (46.3%). Interestingly, there was a subgroup of university students with mild or more severe depression that reported the same symptoms at higher frequencies: fatigue (93.7%), longer time to think (81.2%), poor concentration (87.5%), sleep disturbance (81.2%), and frustration (93.7%). While this study demonstrates that postconcussive symptoms can be found in healthy individuals, it also suggests that the presence of psychiatric disorder, such as depression, may exacerbate the severity of reported symptoms. This notion is further supported by the findings of Garden and Sullivan (2010). Of 96 healthy participants, 74% reported more than 6 symptoms associated with PCS. Twenty-four individuals of the sample met criteria for depression, and their symptoms were assessed separately and compared with the non-depressed, healthy individuals. The results were similar to Wang et al. (2006) in that depressed individuals reported significantly more PCS than the non-depressed group. An earlier study by Chan (2001) also suggests that otherwise healthy adults may develop PCS. The most commonly reported symptoms were similar to the most common symptoms in the Wang et al. (2006) study. The two studies above suggest that certain postconcussive symptoms can be present in healthy individual but there does not appear to be an absolute reason why these symptoms are present. One possible explanation is that these symptoms are indicative of general stress experienced in everyday life. If PCS can exist in otherwise healthy populations due to stress, it raises the question whether the addition of a brain injury or psychiatric condition exacerbates already existing symptoms rather than causing symptoms. It is possible that the addition of a brain injury or psychiatric disorder causes new symptoms while exacerbating existing symptoms.

Iverson (2006) found that nearly 90% of 64 participants with diagnosed depression met liberal criteria for the diagnosis of PCS, and 50% met strict criteria for PCS. When looking at moderate to severe PCS, 72% of participants reported 3 or more symptoms, 27% reported 7 or more symptoms, and 2% reported more than 11 symptoms. These figures increase significantly when looking at mild PCS. Thirteen of the 64 participants reported past experiences with concussion accompanied by LOC, but interestingly, did not differ in frequency or intensity of reported PCS compared to the non-concussed individuals. In other words, the concussion did not affect reports of PCS. The studies above provide evidence that PCS symptoms are not specific to mTBI and are diagnosable in healthy populations, as well as individuals with psychiatric illnesses (e.g., depression, substance abuse). It is further supported in the literature that
symptoms of PCS are non-specific, which means these symptoms are not specific to one disorder, trauma, or cause. This calls into question the etiology and existence of PCS as a diagnosis, and that the development of PCS can be associated with other factors other than the neurological consequences of a brain injury. Lange et al. (2008) conclude that it has become common practice for practitioners and “clinicians to draw conclusions that an individual’s pattern of cognitive deficits is consistent with an established profile even though such a profile has not been identified” (p. 22). Therefore, in the present context, the term postconcussive symptoms will be used to describe a cluster of symptoms with an unknown etiology commonly found in many diagnostic groups, and not a syndrome consisting of symptoms strictly caused by concussion. The term postconcussive symptoms will not be used interchangeably with postconcussive syndrome as some clinicians use them in the brain injury literature. It was considered important to distinguish between PCS as a syndrome, which implies a state of being influenced by a cause, and PCS as a cluster of symptoms being mediated by unknown factors.

Overlapping and Unique Postconcussive Symptoms

While it may be that PCS is not a direct consequence of mTBI, there may be certain symptoms within that PCS cluster that are more attributable to brain damage than psyche damage. Morissette et al. (2011) used structural equation modeling (SEM) to examine competing hypotheses: PTSD and depression symptoms mediate the effects of TBI status on PCS or PCS fully mediates the effects of TBI on PTSD or depression. They found that the data were better supported by the hypothesis that PCS mediates the effects of TBI on PTSD. TBI and combat exposure accounted for 50% of the variance in PCS while PTSD and depression accounted for 40% of the variance in PCS. They conclude that their data suggests a direct relationship exists between TBI and PCS that is not due to the co-occurrence of TBI, PTSD, and depression. Further, it suggests that there are some postconcussive symptoms that are unique to mTBI as well as PTSD.

Maguen, Lau, Madden, and Seal (2012) assessed 1,549 OIF/OEF veterans and used factor analytic techniques to determine that there are both unique and shared symptoms among mTBI and PTSD. There was a weak correlation between the mTBI factor and the PTSD factor, suggesting that certain symptoms loading on said factors are unique to that diagnoses. Factors that were unique to mTBI were dizziness or balance deficits, headaches, memory problems, and visual issues. Irritability and sleep problems, two symptoms commonly associated with the residual effects of mTBI, overlapped with PTSD. Symptoms specific to PTSD included nightmares and avoidant behaviours. Emotional numbing, sleep issues, and hypervigilance overlapped with post-deployment issues. The study suggests that there are certain symptoms specific to PTSD, such as dizziness, headaches, memory problems, and sensitivity to light, as well as symptoms that are more specific to PTSD, such as nightmares and avoidant behaviours. That being said, certain instruments or tools examining non-specific symptoms may lead practitioners to conclude said symptoms are a product of an illness or injury that is not actually mediating the symptoms.

Dean, O’Neill, and Sterr (2012) found similar results when looking at mTBI and non-TBI groups. These groups were further divided based on whether individuals had PCS or PTSD. Thirty-three participants had mTBI with PCS (mTBI+PCS), 73 individuals had mTBI without PCS (mTBI-PCS), 83 individuals had no brain damage but exhibited PCS (control+PCS), and 161 had no brain damage and no PCS (control-PCS). Individuals reporting PCS scored higher on the Impact of Event Scale-Revised (IES-R; Dean et al., 2012) and therefore reported more
PTSD symptoms than those without PCS. While individuals with PCS had higher scores on all measurements (cognitive and psychiatric) regardless of having an mTBI, individuals with mTBI reported significantly more symptoms of headaches, nausea, taking longer to think, sensitivity to light, and double vision. There was a significant interaction between mTBI and PCS for headaches. These results support that there are unique postconcussive symptoms among those with mTBIs, specifically headaches, taking longer to think, balance issues, and vision problems. The authors conclude that it appears that cognitive deficits are responsible for most of the difference between mTBI and psychiatric disorders. They found that PTSD was significantly more present in individuals with mTBI+PCS than mTBI-PCS, and that there was no significant difference between PCS prevalence from the mTBI+PCS group and the control+PCS group (31% vs. 34%). What this suggests is that mTBI did not play a role in severity or reporting of PCS, but PCS played a role in the possible development of PTSD. In contrast to the study above, Hoge et al. (2008) found that, after adjusting for PTSD and depression, all health outcomes, including dizziness, nausea, balance issues, and memory deficits were associated with depression and PTSD over mTBI, with the exception of headaches.

In a similar multivariate analysis, MacGregor, Dougherty, Tang, and Galarneau (2012) found that there were symptoms unique to mTBI other than just headaches. Out of 334 soldiers with mTBI, 27% met criteria for PTSD and 20.7% met criteria for depression. This was significantly higher than the non-head injury group, suggesting that sustaining an mTBI may contribute to PTSD and depression. All symptoms, except muscle/joint pain and weakness, were significantly higher in the mTBI group than the non-TBI group even after considering demographic variables. Significant associations remained for headaches, back pain, memory problems, tinnitus, and dizziness. Macera, Aralis, MacGregor, Rauh, and Galarneau (2012) add support to the idea that certain symptoms are specific to TBI. While those with PTSD reported more neurobehavioural symptoms than those with TBI nearly a year after deployment, those with a dual diagnosis of TBI and PTSD reported a larger increase in neurobehavioural symptoms such as back pain, feeling weak, ringing in ears, dimming of vision, and dizziness. Comparing the groups, this suggests that having a TBI increased specific self-reported symptoms, namely back pain, feeling weak, sleeping difficulties, dizziness, and vision issues.

Some of the symptoms that have been commonly reported to be more associated with mTBI may actually be influenced by each other. For example, all studies above reported either sensitivity to light, dimming vision, or double vision, suggesting that vision in general could be affected by mTBI. While the literature above suggests that vision problems are more often associated with mTBI than PTSD, those with PTSD often report vision problems as well (Barlow-Ogden & Poynter, 2012). Barlow-Ogden and Poynter (2012) examined visual attention among three groups of OIF/OEF veterans: 17 diagnosed with PTSD, 15 diagnosed with PTSD and mTBI, and 15 with neither diagnosis. The authors found that those in the PTSD group performed significantly worse than the PTSD-only and control groups across all variables. In milliseconds, the mTBI+PTSD group had a response time (RT) of 989; the PTSD group had a RT score of 778, and the control group had a RT score of 668. With respect to accuracy, the mTBI+PTSD group had 80.1% accuracy; PTSD only group had 85.6% accuracy; and the control group had 88.1% accuracy. The above scores represent the left visual field (LVF), but the right visual field (RVF) scores were very similar, therefore, only LVF scores were reported in this report. Intrasubject response time SD scores were similar in trend, with mTBI+PTSD group performing significantly worse than the other groups. However, comparing LVF to RVF scores, the control group was the only group to have a consistent score. The mTBI+PTSD group had
more difficulty shifting attention to the LVF while the PTSD-only group had more difficulty shifting to the RVF. Barlow-Ogden and Poynter conclude that those with mTBI+PTSD may have right hemisphere deficits and those with PTSD only may have left hemisphere deficits. While the PTSD group did worse than the control group, indicating those with PTSD only have visual attention issues, the added presence of an mTBI seemed to impede visual attention even more. Unfortunately the study did not include an mTBI group only, and therefore conclusions as to whether visual attention deficits are better explained by mTBI or PTSD are limited.

According to the literature above, headaches also appear to be more attributable to mTBI than PTSD. Theeler, Flynn, and Erickson (2012) examined chronic daily headaches (CDH) in U.S. soldiers who sustained an mTBI during deployment to determine the characteristics and factors associated with CDH and episodic headaches. Out of 978 soldiers with mTBI, nearly 98% reported persistent headaches over the course of 3 months. Twenty percent of these individuals met criteria for CDH, having a median of 26.7 headache days per month. Seventy-eight percent of these individuals met criteria for episodic headache, having a median of 3.3 headache days per month. Looking at the general U.S. population, CDH is more than 4 times likely in soldiers after sustaining a concussion. Interestingly, symptoms of PTSD are strongly correlated with CDH as compared to episodic headaches, while cognitive performance, history of concussion, LOC, and mechanism of injury were not significantly different in those with CDH and those without. This suggests that post-traumatic stress may play a role in mediating chronic headaches, which is in contrast to some of the studies above.

A study by Vasterling et al. (2012) assessing PTSD, depression, and neuropsychological performance in U.S. Army soldiers, suggests that mTBI and other TBI were not associated with poor neuropsychological performance, even before adjusting for other psychiatric disorders. A study by Ponsford et al. (2012) also demonstrated that neuropsychological performance was not associated with PCS. In the Vasterling et al. (2012) study, the PTSD Checklist, Civilian Version (PCL-C) and the Center for Epidemiological Studies Depression Scale (CES-D) scores were significantly correlated with reaction time, code substitution learning, and delayed recall; the higher the scores of PTSD and depression, the poorer the cognitive performance. TBI was significantly and negatively correlated with the Veterans RAND 12-item Health Survey (VR-12) scores before and after adjusting for PTSD depression, indicating that mTBI and other TBI were associated with poorer self-reported health outcomes. However, both PTSD and depression instruments were also associated with poor self-reported health outcomes.

Glaesser, Neuner, Lutgehetmann, Schmidt, and Elbert (2004) assessed two groups of individuals with TBIs for PTSD based on whether they were allegedly conscious or unconscious at the time of their injury. They found that individuals conscious at the time of injury had a significantly higher proportion of PTSD symptoms than the group who were reportedly unconscious at the time of injury. Interestingly, the unconscious group also reported significantly more severe injuries than the conscious group. These findings suggest that loss of consciousness at the time of injury may impede the development of PTSD symptoms. It also suggests that the presence or absence of consciousness at trauma may be associated with the severity of the injury. In theory, those with severe brain injuries would be less likely to have memory of the event and those with less severe brain injuries would be more likely to have memory of the event. The same study demonstrated that the conscious group reported significantly more re-experiences with the traumatic event and significantly higher rates of anxiety on the Hopkins Symptoms Checklist (HSCL). A limitation of this study is that only four individuals from the conscious group were diagnosed with PTSD and only one individual from
the unconscious group was diagnosed with PTSD, so these findings may not be reflective of a more comprehensive sample of participants.

Jones, Harvey, and Brewin (2005) examined PTSD and acute stress disorder (ASD) symptom clusters in individuals with and without TBIs who experienced trauma and came to similar conclusions. At a 6-week follow-up, the non-TBI group reported significantly more re-experiencing symptoms than the TBI group. Within the TBI group, individuals with a TBI and a diagnosis of PTSD were more likely to report feelings of fear at the time of the traumatic event than individuals with a TBI without a diagnosis of PTSD. These findings, at the time, suggested that re-experiencing symptoms and responding with fear may be more attributable to PTSD than TBI. However, at a 3-month follow-up, there was no statistical difference between the groups. The TBI group began reporting more re-experiencing symptoms and fear symptoms than before. The reason for this is unknown, though the initial discrepancy could be explained by the level of unconsciousness survivors of TBIs usually experience, as results by Glaesser et al. (2004) indicate. It appears that the TBI with PTSD group experienced an increase in the symptom of revisiting the traumatic event. This could suggest that between the 6-week follow-up and the 3-month follow-up that the individuals with TBIs reconstructed memories based on information surrounding their trauma. Bryant, Marosszeky, Crooks, and Gurka (2000) found evidence to support similar conclusions. Out of 96 patients with severe TBI, 27.1% were diagnosed with PTSD. This supports the idea that PTSD can develop following a TBI, including a severe traumatic brain injury. Further, 19.2% of patients reported intrusive memories. However, at the time of the injury and the first few months afterwards, none of the participants had recollection of the event. Assessed 5 – 7 months later, 5 individuals reported intrusive memories of their accidents. The authors concluded that these memories were reconstructed based on information acquired after remittance of posttraumatic amnesia (PTA).

Previous literature shows that re-experiencing symptoms are more attributable to PTSD than mTBI. However, there are individuals with mTBI that do report re-experiencing symptoms. Kennedy, Leal, Lewis, Cullen, and Amador (2010) assessed and compared 586 soldiers with blast-related mTBIs and 138 individuals with non-blast related injuries for PTSD. There was no difference among blast and non-blast group in regards to symptom severity. However, when comparing symptom clusters, those who sustained blast-related mTBIs reported more re-experiencing symptoms, such as flashbacks and nightmares. One explanation for this is that perhaps explosions are considered more stressful than other injury mechanisms such as motor vehicle crashes or falls; Kennedy et al. (2010) note that explosions are usually a direct result of enemy attacks.

Reviewing the literature, it is apparent that postconcussive symptoms are not a direct result of a concussion, and that many of the symptoms associated with this label are non-specific. There does appear to be symptoms within the cluster that is PCS that are more attributable to mTBI and PTSD. It appears that headaches, dizziness or balance deficits, visual issues, and memory problems are more likely to be associated with mTBI, while symptom clusters such as intrusive memories and nightmares, emotional reactivity, and avoidance are more likely to be attributable to PTSD. This seems to be especially plausible in the initial stages of injury. However, after several months of persisting symptoms, PTSD (and other psychiatric conditions) seems to account for more of the postconcussive symptoms. This suggests that PTSD, or even anxiety in general, can maintain certain postconcussive symptoms. If this is the case and there are unique PCS symptoms between mTBI and PTSD, then instruments measuring these symptoms should be more sensitive to distinct symptoms rather than all PCS symptoms.
Contradictory results throughout the literature could be explained by using different measurement tools as well as different statistical methods to reach results. Although all studies attempted to understand postconcussive symptoms in the context of mTBI and PTSD, very few studies were replicated.

**Developmental Risk Factors**

As discussed, re-experiencing memories of the trauma is the essential characteristic of PTSD. One of the areas of research that has gathered a significant amount of attention is whether amnesia of the traumatic event serves as a risk factor for PTSD. Gil, Caspi, Ben-Ari, Koren, and Klein (2005) found a positive correlation between the development of PTSD and having memory of the event allegedly responsible for the PTSD. One hundred and twenty individuals with mTBI were assessed for PTSD and whether they had memory of the event. Sixty-five (55%) participants reported no memory of the event and 55 (45%) reported memory of the event. Among the 55 individuals with memory of the event, 13 (23%) developed PTSD. Among the 65 participants with no memory of the event, 4 (6%) were diagnosed with PTSD. Those with memory of the event are five times more likely to develop PTSD than those without memory of the event when looking at the crude relative risk for PTSD. These findings are consistent with the results reported by Glaesser et al. (2004), with regards to having consciousness of the injury. The association between memory of the event and the development of PTSD remained stable at a 6-month follow-up. Further, a MANOVA analysis revealed that the re-experiencing symptom cluster was responsible for most differences between groups. This suggests, not surprisingly, that the symptoms of re-experiencing the traumatic event are associated with having memory of the event. Gil et al. (2005) concluded that individuals with mTBI without memory of the event may serve as a protective factor against PTSD, though it is possible for PTSD to develop in the absence of memory of the traumatic event.

In contrast to the findings above, Hoge et al. (2008) found that 43.9% of those with mTBI with LOC met criteria for PTSD. This is a significant difference between the 6% of individuals with mTBI with no memory whom developed PTSD in the Gil et al. (2005) study. This difference could be a result of when the individuals were assessed. Participants in the Hoge et al. (2008) study were assessed 3-4 months after returning from a 1-year long deployment in Iraq, while participants in the Gil et al. (2005) study were assessed in the hospital, no later than 24 hours after their injury. It could be that the individuals in the Hoge et al. study developed memories of their injury over time, while those in the Gil et al. study did not have this time to experience recollections of the accident. Additionally, those in the Hoge et al. (2008) study may have lost consciousness for only a few minutes, while those in the Gil et al. (2005) study may have experienced LOC for closer to 30 minutes. Both groups of individuals would qualify for a diagnosis of a mild brain injury. It is also important to note that individuals in the Gil et al. (2005) study were categorically assigned to either having memories or not having memories based on the median cut-off score (2.1) of a 9-item questionnaire. The individuals in the Hoge et al. (2008) study were considered to have a loss of consciousness if they reported so.

The fact that some of those with mTBI developed PTSD is enough evidence to suggest that PTSD can develop after mTBI. However, there is still controversy as to whether mTBI is then a risk factor for developing PTSD. Nearly 27% of veterans with mTBI met criteria for PTSD and 20.7% met criteria for depression, compared to veterans with non-head injuries among whom 17.2% met criteria for PTSD and 13.2% met criteria for depression (MacGregor et al., 2012). Not only does this study show that PTSD and mTBIs can co-occur, but it also
suggests the presence of a mild traumatic brain injury increases the likelihood of developing PTSD. The study by Hoge et al. (2008) that revealed that 43.9% of individuals with mild traumatic brain injury met criteria for PTSD also supports the claim that PTSD can develop in those with mild traumatic brain injuries. The authors concluded that there was a strong association between the development of mild traumatic brain injury and posttraumatic stress disorder. Those with mTBI were more likely to develop PTSD than those without. This suggests that mTBI could be a predictor for PTSD.

Additionally, out of 124 individuals in the study by Hoge et al. (2008), 27.3% individuals with an mTBI and an altered mental status at the time of injury met criteria for PTSD; 16.2% of individuals with non-brain injuries met criteria for PTSD; 9.1% of individuals with no injuries met criteria for PTSD. First, this study suggests that an injury itself may increase the risk for PTSD. Second, individuals not conscious for some time of their injury were the most likely to develop PTSD, which is contradictory to findings by Gil et al. (2005) and Glaesser et al. (2012). It could be that those who reported not having consciousness of the injury experienced a more severe injury. As a result, memories reconstructed sometime after the initial injury may have been particularly stressful. The study by Hoge et al. (2008) had significantly more participants, which may be a better representation of true findings. Third, PTSD was strongly associated with mTBI.

In order to determine the role combat stress plays in mediating PCS, 472 veterans with mTBIs participated in self-report questionnaires regarding PCS and posttraumatic stress (Cooper et al., 2011). The sample was split into a low combat stress group and a high combat stress group. Cooper et al. found that rank, gender, and time of injury to assessment were associated with degree of stress during combat. Women were significantly more likely to experience high stress, while officers were significantly more likely to fall under the low stress group. Individuals who had longer wait times to be assessed from the time of their injury were also more likely to fall in the high stress group. Additionally, the high stress group had 3-8 times greater scores on the Neurobehavioural Symptom Inventory (NSI), indicating they reported significantly more PCS. This was true for all postconcussive symptoms suggesting that the severity of stress can influence the severity of postconcussive symptoms and PTSD symptoms in veterans with mTBI. While female veterans were more likely to report higher amounts of stress, which is indicative of PTSD and increased PCS, Morisette et al. (2011) found that male veterans were significantly more likely to sustain a TBI (50.8% vs. 14.3%); this may be due to women reporting lower levels of combat exposure.

The findings by Morisette et al. (2011) are further supported by Polusny et al. (2011) in that males were more likely to sustain a TBI. Additionally, Polusny et al. looked at military rank and found that officers were significantly less likely to sustain a brain injury and develop PTSD over non-officers. This is consistent with the findings by Cooper et al. (2011) that officers were significantly less likely to report high stress. In contrast to Cooper et al. (2011), however, Hoge et al. (2008) found that gender did not have an influence on mTBI, PTSD, or PCS. Rank and military service branch have consistently been associated with increased reports of mTBIs, PTSD, and PCS. Macera et al. (2012) found that military rank was correlated with TBI and PTSD; again, officers reported significantly fewer TBIs and PTSD. Nearly 70% of junior ranking participants reported a TBI or PTSD, 25% of midlevel soldiers reported a TBI or PTSD, and approximately 7% of officers reported a TBI or PTSD. Findings by Vasterling et al. (2012) support rank being a predictive factor of injury: 72.2% of participants were junior, 25.7% were mid-level (non-commissioned officers), and 2.2% were officers. Consistent with other reports,
rank was again associated with mTBI and general injuries in the MacGregor et al. (2012) study. Lower ranking soldiers were more likely to sustain injuries than higher ranking soldiers. Being in the Marines was also a stronger predictor of sustaining an injury over those in the Army. The study found that 69.8% of those with mTBI were Marines, 51.2% of those with non-TBI injuries were Marines; 23.4% of those with mTBI were Army, 38.3% of those with non-TBI injuries were Army.

Hoge et al. (2008) found that rank was also correlated with injury: Those low in rank were more likely to sustain an injury of any kind, meaning there was no difference in frequency of reported injuries between the mTBI group and the non-mTBI group. This suggests that being junior in rank and being in a combat situation is a risk factor for sustaining injury in general. However, those reporting mTBI were more likely to be hospitalized, report high intensity combat situations, exposed to explosions, and sustain their mTBI from an explosion. In addition, all participants in the Hoge et al. study reported seeing someone wounded or being killed. More than 75% of those with TBI, PTSD, or both reported experiencing significant fear of dying. MacGregor et al. (2012) found 49.7% of the mTBI cases reported at least 4 combat exposures. This study, along with the study by Hoge et al. (2008), suggest that more combat exposures is a risk factor for mTBI, PCS, and stress indicative of PTSD.

Mechanism of injury has also been shown to be associated with TBI or PTSD throughout the literature, although there are slight differences when comparing military to civilian populations. Schneiderman, Braver, and Kang (2008) found that 44% of veterans reported at least one mechanism of injury. Postconcussive symptoms were significantly associated with mTBI with LOC and not alteration of consciousness (AOC), injury mechanisms, and PTSD. PTSD symptoms were correlated with female veterans, which is consistent with some of the literature above. Interestingly, falls and sports/physical training were the most common mechanism of injury in the veterans assessed. Falls were also the most common among those reporting mTBI with LOC, though blasts were second most common among these veterans. About 11% of participants met criteria for PTSD. PTSD was most common among females, veterans from Iraq, those sustaining multiple injuries, and those with either mild or severe mTBI. Contrary to other findings, there was no association between injury and military service branch. Of the 275 veterans that sustained mild traumatic brain injuries mTBI, 35% reported three or more persistent PCS symptoms. Veterans exposed to blasts had an increase in PCS symptom reporting, but this increase was not significant. PTSD was associated with three or more PCS symptoms.

Polusny et al. (2011) assessed 953 veterans in a longitudinal study to determine long-term psychosocial outcomes and relationships between concussion and PTSD symptoms. Out of sixty veterans with mTBI, 42 (~70%) reported blast-related injuries; twenty-one veterans out of 26 (~80.8%) with mTBI and PTSD reported explosion related injuries. Approximately 24% of those in the PTSD only group reported falls as the most common injury (Polusny et al., 2011). Motor vehicle accidents were the second most common mechanism of injury, with 20% of TBI participants reporting this method of injury, and 23.1% of TBI participants with PTSD. Hoge et al. (2008) found that 79% of veterans with mTBI with LOC reported blasts or explosions as injury mechanism and 72.7% of those with mTBI with altered mental status reported blasts/explosions as the injury mechanism. These results appear to be consistent within the literature. MacGregor et al. (2012) also found that 88.9% of mTBI cases were due to battle related explosions. Those with other injuries reported falls as the most common injury, although the difference between groups was not significant (Hoge et al., 2008). Approximately 30% of
soldiers with mTBI with LOC reported motor vehicle crashes (MVC) as their injury mechanism, 18.1% of mTBI with altered mental status, and 13.3% with other injuries also reported MVC as their mechanism of injury. Ponsford et al. (2012) compared individuals residing in the emergency department with mTBI to other traumatic injuries and found that the mTBI group was significantly more likely to receive their brain injury from being assaulted (13.3% vs. 2%) as well as from being in motor vehicle accidents (40.9% vs. 28%). The authors also report that 78.9% of those with brain injuries sustained significantly more soft tissue damage than 59% of those sustaining other injuries. The authors report that being involved in litigation and having a history of head injuries were not predictors of PCS. Comparing individuals with mTBI to those with other brain injuries, Landre et al. (2006) found that 86% of those with mTBI reported MVC as their mechanism of injury, and 65% of individuals in the trauma control group reported MVC as injury mechanism. Sheedy et al. (2006) reported that assault (34.5%) was the most common mechanism of injury in their study, followed by low falls (20.7%), collisions with an object (17.2%), and road trauma, including MVC, accounting for 13.8%. Interestingly, motor vehicle accidents were least common, which appears to contrast most civilian studies.

These results suggest that, in a military context, blasts are the most common mechanism of TBI, and the development of PTSD. Outside of the veteran population, it appears that motor vehicle crashes are the most common mechanism of injury. Mechanism of injury is a predictive factor in that during these types of injuries the chances of sustaining an mTBI or developing PTSD are increased.

According to Hoffman et al. (2012), their study was the first to examine possible pre-injury personality characteristics that are risk factors for the development of PTSD within individuals with mTBI. Those who developed PTSD were more likely to report more worry and less happiness, both before the injury and six months after the injury. They were also significantly more likely to blame others for their injury. Those with mTBIs reported the opposite; they reported more happiness and less worry both pre- and post-injury, and they were more likely to claim responsibility for their injury rather than blaming others. The above study suggests that certain personality characteristics, such as worry, happiness, and locus of control may be predictors of PTSD after mTBI. Currently, it is unknown why those with PTSD reported a less positive report of their injury than those with mild traumatic brain injuries. It could be that the symptoms more associated with PTSD influenced a more negative view than the symptoms associated with mTBI. More research is needed to support these findings. Interestingly, Barlow-Ogden and Poynter (2012) found that when examining visual attention, those who complained more about vision issues performed significantly slower than those who did not complain. Of course, the individuals who complained more may have had worse individual vision problems, but it could also be that their perception of their vision influenced their performance.

Williams, Evans, Needham, and Wilson (2002) assessed 66 severe TBI survivors and found that there was no relationship between reporting PTSD symptoms and length of LOC. The severity of injury was not correlated with the severity of PTSD, or memory impairment. However there was a positive correlation between severity of PTSD and attribution of external control for the event. These findings suggest that it may not be the severity of the event, but the nature of the event in terms of the individual's perception of their ability to control it.

The study by Macera et al. (2008) also revealed PTSD was associated with scores indicative of poor health on both the Post Deployment Health Assessment (PDHA) and Post Deployment Health Reassessment (PDHRA): Approximately 45% of those with co-morbid TBI
and PTSD and 31% of those with PTSD reported poor health on at least one of the instruments, compared to 18% of the TBI only group and 13% of those with neither condition.
Chapter 3: Methodology

A literature search was conducted using the EBSCOhost databases that were available to Kingston’s St. Lawrence College (CINAHL with full text, ERIC, MEDLINE, Military and Government Collection, PSYCARTICLES, PSYCBOOKS, and PSYCINFO) and Kingston’s Providence Care Regional Community Brain Injury Service (CINAHL with full text, MEDLINE, Psychology and Behavioral Sciences Collection, Biomedical Reference Collection: Comprehensive, Nursing & Allied Health Collection: Comprehensive). A-Z database provided by Providence Care was also used, although only to obtain one article.

Key search terms in all databases, with the exception of A-Z, included: “PCS”, “postconcussion/concussive symptoms/syndrome”, “mild traumatic brain injury”, “mTBI”, “traumatic brain injury”, “TBI”, “posttraumatic stress disorder”, “PTSD”, and “combat stress”. In the beginning stages of research, information and peer-reviewed journal articles were gathered for thesis proposal purposes. Out of the first 20 articles that were considered relevant for thesis proposal purposes, 11 were used for introduction and background purposes with searching the above keywords. These articles were used for the introduction based on whether the information was relevant to the issue of overlapping symptoms between TBI and PTSD, and the co-occurrence of these diagnoses. During research for the literature review, the above keywords were paired with areas of interest in the literature review, namely epidemiology, symptom overlap, developmental risk factors, long-term consequences, treatment outcomes, and brain functionality. However, only three areas of interest were included in the literature review: Epidemiology, overlapping and distinct postconcussive symptoms, and developmental risk factors. In total, 45 articles were used for the literature review; 29 of said articles were used as empirical evidence (see Appendix A), while other articles were used for general information.

Articles were viewed if full-text versions were provided and were relevant to the association of PCS, TBI, and PTSD based on reading the title and abstract. If articles provided significant and relevant information from a secondary source, efforts were made to obtain the primary source by checking the references and searching the title of the secondary source through Google Scholar®. Only one article was unavailable and was therefore used as a secondary source. Selected articles primarily focused on the U.S. veteran population because most current research regarding the relationship between TBI, PTSD, and PCS revolves around veterans. However, civilian articles were also included because they may not experience variables that are present in a combat setting; strictly examining TBI and PTSD within a combat setting may not accurately reflect the true relationship between these diagnoses and PCS. Articles that were directly relevant for empirical purposes were summarized in a table (see Appendix A).
Chapter 4: Results

The following section summarizes the descriptive data gathered in the literature review, highlighting key findings and themes. Data is organized and presented to reflect central findings and ideas, but also follows the layout of the literature review, providing information on epidemiology, PCS as a diagnosis, which symptoms are better explained by mTBI and PTSD, and finally risk factors. Afterwards, the discussion section provides inferences and implications of the following results.

In terms of the epidemiology, the prevalence rates of mTBI and PTSD suggested in the literature are unclear. While estimations of rates are given, it appears that none are absolute and professionals often disagree about the true rates of prevalence. This is due, in part, to using instruments that necessarily include some error, using different tools to estimate in different studies, using clinical interviews to diagnose, which may have inter-rater reliability issues, and the fact that there are likely a percentage of individuals who have never been assessed. Finally, a common complaint within the literature is the weak and inconsistent use of definitions for brain injuries, which makes identification and discrimination between the disorders difficult. This will be further discussed in the discussion section.

The literature suggests that experiences that reflect post concussive symptoms do not appear to be directly and exclusively related to concussions. While there is evidence to show that PCS develop after an mTBI, or even brain injuries of other severities, PCS have been shown to develop in university students without severe and long lasting physical injuries (Wang et al., 2006). A subgroup of students with depression reported PCS more frequently. Additionally, postconcussive symptoms have been shown to be better explained by psychiatric illnesses than mild traumatic brain injuries (Sheedy et al., 2008; Ponsford et al., 2012). Although Ponsford et al. (2012) found that PCS were better explained by mTBI in the first week of injury and long term postconcussive symptoms were more likely to be maintained by psychiatric conditions. The results suggest that postconcussive symptoms alone are not sufficient as a diagnosis, but possibly related to the severity of the mTBI along with interactions with psychiatric illnesses. This conclusion is reflected by the refusal of post concussive syndrome as a diagnosis in the DSM-IV-TR and DSM-V.

Statistical analyses in the literature suggest that there are certain symptoms within the cluster of PCSs that are more unique to either mTBI or PTSD respectively, though the degree of specificity is unknown. Symptoms found to be more associated with PTSD include re-experiencing symptoms such as nightmares, intrusive and frequent memories, as well as avoidant symptoms. Symptoms found to be more associated with mTBI include dizziness or balance deficits, nausea, headaches, memory problems, visual issues, and taking longer to think. That is not to say that the symptoms associated with mTBI cannot be found in those with PTSD, but that among the literature reviewed, these symptoms were more likely to be a product of mTBI. For example, Hoge et al. (2008) found that all health outcomes, including the symptoms listed above, with the exception of headaches, were more likely to be associated with PTSD than mTBI.

Level of consciousness, or amnesia, has also been suggested to be a risk factor for increasing or impeding the development of PTSD after a brain injury (Gil et al., 2005; Bryant et al., 2000). The literature suggests that individuals conscious during their brain injuries were more likely to develop PTSD. However, those without consciousness during their brain injuries can develop PTSD as well. Other studies suggest that those without consciousness were more likely to develop PTSD than those with consciousness. Brain injuries, specifically mild traumatic brain injuries, have also been suggested as a risk factor for developing PTSD.
(MacGregor et al., 2012; Hoge et al., 2008). In fact, non-head injuries increase the chances to develop PTSD (Hoge et al., 2008). In the military literature, gender (Cooper et al., 2011) and rank (Macera, 2012; Polusny, 2011) were common risk factors for developing high stress and mild traumatic brain injuries. Mechanisms of injury have also been suggested to be risk factors for developing brain injuries and PTSD, such as falls and training accidents, blasts and explosions, and motor vehicle accidents (Schneiderman et al., 2008).

It is common that studies within the literature have contradictory findings. For example, having consciousness during a brain injury is a risk factor for developing PTSD in one study, while other studies support the opposite finding that being conscious during a brain injury is less likely to result in the development of PTSD. These discrepancies throughout the literature are further examined in the discussion section, where implications and inferences are given with respect to the qualitative data in the literature review.
Chapter 5: Discussion

The following discussion section examines implications of the results; both from the results section and literature review. Afterwards, conclusions will be offered with respect to the research question. Strengths and limitations of the thesis are discussed from a multilevel systems perspective, which focuses on the creation of the academic project on a client level, program level, and societal level. Provided in the strength and limitations section will be potential contributions of the thesis to the field of Behavioural Psychology field, which envelope other related human service fields as well. Finally, recommendations for future projects, research, and/or programs are proposed.

Implications of Results

As stated earlier in the results section, contradictory conclusions exist within the literature with regards to the relationship of PCS to mTBI and PTSD. This applies to most research included in the present literature review. According to Tanielian and Jaycox (2008), 220,000 – 300,000 veterans have sustained mild traumatic brain injuries. Their report has been cited in many studies regarding mTBI prevalence rates. However, Hoge et al. (2009) claim that this number is a gross misrepresentation because the only criterion to qualify for an mTBI was whether the veterans in this study were dazed or confused at the time of the injury. In other words, the definition of a mild traumatic brain injury used in this study is too broad, and likely encompasses subjects who may not in fact have experienced a mild traumatic brain injury. This is in contrast to the PTSD literature, which adheres to one definition of PTSD. The mTBI literature is littered with varying, and sometimes overly broad or contradictory definitions of mTBI. This affects the validity of mTBI literature as instruments used to collect data are based on the definitions of mild traumatic brain injuries. This is further discussed in the section reviewing the discrepancy between which symptoms of PCS are specific to either mTBI or PTSD.

While there are some studies that support that postconcussive symptoms are better explained by mTBI (Sheedy et al., 2006), most in the literature review conclude that some sort of psychiatric disorder better accounts for this constellation of symptoms (Ponsford et al., 2012; Landre et al., 2006; Lange et al., 2008). For example, university students reported high frequencies of five specific postconcussive symptoms; these symptoms were more intense among a subset of students with depression (Wang et al., 2006). This indicates that postconcussive symptoms, once thought of as symptoms associated solely with concussion, can seemingly develop in otherwise healthy students, with no reports of head trauma. Additionally, while substance use and stress were not assessed in this study, it is possible that, given the nature of university life, these factors could also be related to the students’ reports of postconcussive symptoms. Certainly, this raises the question as to the extent that stress or substance use could impact the development of postconcussive symptoms among those with an mTBI and/or PTSD. At present, it is unknown how general anxiety and stress impact PTSD and PCS; perhaps it is general anxiety and/or stress responsible for postconcussive symptoms.

Attributing certain postconcussive symptoms to PTSD is also cautioned. Most authors cited in the literature review conclude that psychiatric conditions in general, rather than PTSD, explain PCS better than brain injuries. It is unknown how general anxiety and stress impact PTSD and PCS; perhaps it is general anxiety and/or stress responsible for postconcussive symptoms.

Results from the Wang et al. (2006) study also calls into question whether instruments designed to measure PCS after a mild traumatic brain injury, or a traumatic event, may in fact be measuring postconcussive symptoms that were potentially present in the individual prior to their accident. There is a possibility that an accident exacerbates existing symptoms. At present,
research has not accounted for the possible existence of PCS before an accident, and as such there is no clear causal relationship between brain injury or traumatic event and the development of postconcussive symptoms.

However, statistical methods suggest that certain postconcussive symptoms, and not postconcussive syndrome as a whole, can be attributed to either mTBI or PTSD. There appears to be discrepancies that exist about which symptoms can be attributed to each condition. Some studies suggest that symptoms such as headaches, dizziness, balance deficits, and visual issues can be attributed to a brain injury rather than PTSD, while some researchers report that only headaches can be attributed to brain injuries. One explanation for these discrepancies is the difference in statistical methods used, as well as differences in experiments. Differing experimental designs and statistical methods will be further discussed as a topic for future research recommendations.

Suggestions and Recommendations

There are several suggestions for future research regarding this topic. First, practitioners concerned with brain injuries need to reach a consensus on what constitutes a brain injury. This will allow practitioners to validate brain injury instruments, as well as increase the validity for these instruments and, as a result, the practitioners’ studies. Second, it has been shown that PCS as a diagnosis is not yet an efficient diagnosis but certain symptoms associated with PCS can be attributable to either mTBI or PTSD and other psychiatric conditions. Therefore instruments, as well as definitions, should focus on symptoms, which are more likely to be attributable to the diagnosis that the instrument is concerned with rather than PCS as a whole. For example, mTBI instruments or definitions should place more emphasis on symptoms such as headaches, dizziness, or visual issues.

The above suggestions also pave the way for replication of past studies. A lot of the research in the literature review, specifically in the brain injury field, lack replication. When experimental designs and statistical methods are replicated in a study, the authors of said study should cite additional studies that have used exact or similar methods. This would create a clear link of continuous research that has been rigorously tested and built upon within a body of literature that currently lacks consistency. Additionally, studies lacked sufficient control groups, adding difficulty to making conclusions. It is suggested that more research replicate a method similar to Dean et al. (2012) whereby the authors divided participants into several groups: mTBI with PCS, mTBI without PCS, PCS but no brain damage, and no PCS and no brain damage.

As stated earlier, one of the barriers to inferring a causal relationship between a brain injury/traumatic event and the development of PCS is the lack of research examining PCS prior to an injury or traumatic event. While this information would be difficult to obtain from the civilian population, the military population provides opportunity because soldiers are likely to be exposed to possible traumatic events, such as seeing death, killing enemy combatants, or a brain injury itself. Currently, there are researchers interested in assessing militants before being deployed. However, often practitioners only assess prior psychiatric conditions and prior brain injuries rather than specific post-concussive symptoms. Unfortunately, as the results demonstrate, knowing whether an individual received an mTBI during deployment is not sufficient evidence to assume an mTBI caused current postconcussive symptoms. Those interested in causal relationships between PCS and mTBI or PTSD should conduct pre- and post-deployment measurements of PCS if possible.
**Strengths and Limitations**

This paper is limited in that this literature review is not an exhaustive review of all the literature regarding mTBI, PTSD, and PCS. The literature review was limited to three main areas of research: epidemiology, PCS symptom overlap, and risk factors. Even within these three domains of research this literature review does not review all the literature. Additionally, the research gathered focused mostly on military populations over civilian populations. This is partly due to the large body of research of mTBI and PTSD in the military population. Further, all data collected was descriptive, and therefore it is difficult to make conclusions about said data.

A strength of this literature review is the method of which information and articles were gathered, which can be found in Appendix A. The reader can see the variety of definitions, instruments, and methods used in the TBI literature and the consistency in the PTSD literature. This also provides additional information about the studies that may not have been deemed necessary to include in the literature review.

From a multilevel challenges perspective, one of the strengths of this thesis is providing current and relevant information to organizations and professionals concerned with mental health and brain injuries, specifically PTSD and concussions. Results show that most practitioners concluded that postconcussive symptoms were better explained and more likely maintained by psychiatric conditions, rather than a brain injury. This raises the question of whether individuals experiencing postconcussive symptoms should seek mental health or brain injury services, or both. When working with clients with a brain injury, some practitioners struggle with how much mental health impacts problematic behaviour. Likewise, professionals in the mental health field may be quick to attribute symptoms to brain injury. This literature review provides practicality in the behavioural psychology, and surrounding fields, by bringing awareness to a cluster of similar symptoms associated with TBI and PTSD, and reviewing evidence of the differences and similarities between these two concepts for practitioners. Understanding the difference could improve diagnostic accuracy and provide insight into appropriate treatments. Further, it may help the referral process; a brain injury service may be able to find adequate and appropriate mental health services for an individual exhibiting these symptoms, rather than treating the symptoms from a purely behavioural (e.g. brain injury) point of view when not needed. Likewise, mental health professionals may be able to provide, or at least refer, an individual to brain injury services if necessary. Additionally, this thesis will aid in future research regarding the overlap of PCS among mTBI and PTSD, as well as surrounding topics.
References


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Appendices

Appendix A: Method – Summarized Articles

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<th>Article and Authors</th>
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<td>Schneiderman, A. I., Braver, E. R., &amp; Kang, H. K. (2008). Understanding sequelae of injury mechanisms and mild traumatic brain injury incurred during the conflicts in Iraq and Afghanistan: Persistent postconcussive symptoms and posttraumatic stress disorder. American Journal of Epidemiology, 167(12), 1446-1452. Doi: 10.1093/aje/kwn068</td>
<td>Cross-sectional study of military personnel following deployment Iraq/Afghanistan with TBIs. Assessed for PTSD and PCS. Mailed questionnaire: injury mechanisms, immediate symptoms post-injury, and PCS. (2235 surveys returned) Brief Traumatic Brain Injury Screen (3 items) injury mechanisms, severity of mTBI: level 1 – dazed/confused, and having immediate PCS (headaches, dizziness); level 2 – difficulty remembering injury, LOC, or self-reported head injury). PCL-17 (scores of 50 or more indicative of PTSD) PCS 3+ 3 or more PCS</td>
<td>Most commonly reported mechanism for TBI: sports/physical training, falls, blasts, and motor vehicle crashes. Falls more likely to be categorized as mTBI.</td>
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<td>Injury mechanisms: Sports/physical training, falls, blasts, and motor vehicle crashes. mTBI increased with more reported injury mechanisms.</td>
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<td>Factors Associated with PTSD: 250 (11%) classified as having PTSD. PTSD 50+ scores “were significantly more prevalent” in females, OIF veterans, those having multiple injuries, and those classified as having level 1 or 2 mTBI.</td>
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<td>Factors Associated with PCS: 35% of 275 vets with in-theatre mTBI reported PCS3+ PCS3+ associated with 3 or</td>
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mTBI level 2: LOC, trouble remembering the injury
PCL-17 (score 50+ considered PTSD)
PCS3+ (3 or more PCS)

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<thead>
<tr>
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</tr>
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<tr>
<td>Cooper, D. B., Kennedy, J. E., Cullen, M. A., Critchfield, R., Amador, R. R., &amp; Bowles, A. O. (2011). Association between combat stress and post-concussive symptom reporting in OEF/OIF service members with mild traumatic brain injuries. Brain Injury, 25(1), 1-7</td>
<td>Co 472 vets with mTBI completed self-reports of posttraumatic stress (separated by high and low stress) and PCS. Screened with 3item mTBI questionnaire mTBI defined as: 1 or more of the following: LOC &lt;30 minutes, loss of memory before event (RTA), or after, (PTA) &lt;24 hrs, alteration in mental state (confusion, dizziness), presence of focal neurological deficits; GCS score &gt;13 Posttraumatic stress: PCL-C. PCS: NSI (Neurobehavioural Symptom Inventory) 22 items, 5 item likert scale (none – very severe)</td>
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724 participants with mTBI (586 blast related vs. 138 non-blast related). Non-blast = injury sustained from deployment-related events (motor vehicle accidents, assaults, and falls not result of blast explosion).

PTSD = PCL-C score of 50+ mTBI definition consistent with American Congress of Rehabilitation Medicine.

Positive symptom clusters = high stress

Of 3 clusters, only re-experiencing symptoms significantly differed among blast-TBI and non-blast TBI (t = 2.3, p<0.02). Re-experiencing symptoms: disturbing intrusive memories, dreams, flashbacks, feeling distressed by cues resembling the traumatic event, and physiological reaction to cues resemb. Traumatic event.

LOC and AOC groups tended to differ on avoidant symptoms (F = 3.773, p = 0.052).

Blast and nonblast groups did not differ in prevalence of PTSD: 38.2% (blast) vs. 33.3% (nonblast) score of 50+ on PCL-C scores.

When blast vs. nonblast injuries are compared, severity of symptoms does not differ. When symptom clusters are examined, those with blast related injuries report more re-experiencing symptoms: nightmares, flashbacks. It may be that blast related injuries are more stressful than motor vehicle crashes, or falls. Blasts are often a result of direct enemy attacks.


Purpose: relationships between the reporting of symptoms of PTSD following TBI and potentially predictive and or protective factors, such as severity of injury, neuropsychological profiles, and causal attributions for the event.

66 survivors of TBI (severe). Based on significant disturbance in consciousness involving either a coma (6hr or more) and/or posttraumatic amnesia (PTA) of

Correlational analyses done to examine relationship between neurological, cognitive, and attributional variables and total IES scores.

No relationship between length of disturbance of consciousness and reporting of PTSD symptoms; negative correlation between severity of PTSD and the discrepancy score; there was no relationship between severity of memory functioning on the
<p>| one day or more. | RBMT and the severity of PTSD; positive correlation between the attribution of external control for the event and severity of PTSD. There was no relationship between perceived avoidability of the event and PTSD severity. | PTSD symptoms not related to severity of injury (severe TBI, keep in mind). Authors suggest that the nature of the accident and not the severity major factor for reporting PTSD symptoms in severe TBI. |
| IES (impact of event scale) – avoidant systems associated with PTSD | | Survivors with moderate to severe dysexecutive disorder may either be protected from developing PTSD symptoms because of a lack of insight, or they may be less able to report on such symptoms. |
| DEX (dysexecutive questionnaire) – 20 item, cognitive, behavioural and emotional problems that characterize a frontal lobe or dysexecutive syndrome. RBMT (Rivermead Behavioral Memory Test) – tests of everyday memory problems. Causality: Their fault (5) no ones fault (0) | | |</p>
<table>
<thead>
<tr>
<th>Benge, J. F., Pastorek, N. J., &amp; Thornton, G. M. (2009). Postconcussive symptoms in OEF/OIF veterans: Factor structure and impact of posttraumatic stress. Rehabilitation Psychology, 54(3), 270-278. Doi: 10.1037/a0016736</th>
<th>Record review of 345 of participants. mTBI: individuals screened for past history of TBI (4 items). Positives were referred for an evaluation by specialty provider or team NSI, PCL-C, injury severity self-report questionnaire. Principal-components analysis using SPSS 16.0</th>
<th>PCS are complex phenomena that likely involve biological and psychological mechanisms that may be affected by a host of factors. “The finding of a history of alteration in consciousness and a current report of somatic, cognitive, and affective complaints in not sufficient to establish a causal link between PCS and mTBI.</th>
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<td><strong>Purpose:</strong> ASD and PTSD symptom profiles in those that sustained TBIs. Total sample: 131 ASD assessed using Acute Stress Disorder Interview PTSD assessed with PTSD Symptom Scale (PSS, 17 items) TBI assessed based on duration of PTA. Asked to recall the accident… “and then…and then….” procedure used by Gronwell and Washington. Used definition of mTBI by ACRM… focused on mTBI (PTA less than 24 hours). Assessed at: as soon as possible, 6 weeks post, and 3 months post.</td>
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<td>At 6 weeks, participants who sustained a TBI reported fewer re-experiencing symptoms relative to those who did not sustain a TBI. Participants with PTSD (even if they had a TBI) recalled more symptoms of fear at the time of trauma (A2) and more symptoms of re-experiencing (B), avoidance (C) and arousal (D) relative to those not diagnosed with PTSD. Those with PTSD and no TBI reported significantly more re-experiencing symptoms (4.00 (1.171)) than those with PTSD and a TBI (2.71). At 3 month follow up: Non-TBI (PTSD only) group reported 3.45 (0.93) vs. TBI-PTSD group which reported more re-experiencing symptoms than at 6 week follow-up (3.50) Individual symptoms: 1st interview, fewer individuals with TBI reported feeling intense fear and helplessness, and reported experiencing recurrent intrusive thoughts and images relative to the non-TBI group. More TBI participants reported “numbing” compared since the trauma relative to non-TBI group. No differences in difficulty sleeping, poor concentration, and irritability.</td>
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120 ind. With mTBI assessed immediately after the trauma, 1 week follow up, 3 months, and 6 months.

No subjects were unconscious during injury, which excludes LOC.

PTSD symptoms assessed with Clinician Administered PTSD Scale, Posttraumatic Stress Scale.

Depression and anxiety symptoms assessed with BDI, BAI.

Automated Neuropsychological Assessment Metrics

RESULTS CONTINUED

Our findings indicate that at least for TBI survivors, forgetting may be protective, in which case the process of deliberate recollection and remembering may be harmful rather than therapeutic.

It is noteworthy that 6% of the participants in this study without memory of the TE did meet PTSD criteria, suggesting PTSD can develop in the absence of memory of the traumatic event.

Cannot rule out possibility that false memories can influence PTSD symptoms (increase risk).

Severity of PTSD

Subjects with memory of the traumatic event significantly more likely to develop PTSD than those without memory of the event; logistic regression analysis revealed that memory of the traumatic event within the first 24hrs is a strong predictor of PTSD 6 months later. Differences in groups primarily from re-experiencing cluster.

55 participants with memory of the traumatic event, 13 (23%) had PTSD, whereas 4 (6%) of the 65 participants without memory of the TE developed PTSD. Thus, the crude relative risk for PTSD among the participants with memory of the TE was almost 5x higher than among those without memory of the TE. This remained stable over 6 months.

No significant differences were found on the avoidance and hyperarousal symptom clusters.

Memory, or lack of, of event did not have impact on cognitive abilities – no differences. Those with memory of event more than 2x likely to have PTSD than those with no memory.

Acute posttraumatic symptoms, and depressive symptoms, anxiety symptoms, within 1 week of the TE as well as history of psychiatric disorder were all associated with an increased risk for PTSD at 6 months.

Participants: 208 (104 head trauma, 104 substance abuse). TBI patients selected based on GCS scores, skull ex-rays, and CT scan results. Mean GCS score: 14.8 (14-15). 64% had LOC based on ambulance crew reports.

Cognitive measures: Trail Making Test (TMT), subtests of Wechsler Memory Scale-Revised (WMS-R), Digit Span Forward, Digit Span Backward, Logical Memory 1, Logical Memory 2, Visual Reproduction1, VR 2.

No significant differences between mTBI and substance abuse groups on any of the 8 cognitive measures.

*could not differentiate patients with mTBI and substance abuse based on tests of memory, concentration, and processing speed; similar on all 8 cognitive tests; no difference in cognitive profile.

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<td>Hoffman, J. M., Dikmen, S., Temkin, N., Bell, K. R. (2012). Development of posttraumatic stress disorder after mild traumatic brain injury. Arch Phys Med Rehabil 93, 287-292</td>
<td>Secondary analysis of randomized controlled trial of telephone follow-up vs. usual care to reduce mTBI symptoms and improve function. 239 participants PCL-C mTBI = likely circumstances for mTBI (car crash, fall) GCS score of 13-15, documented or self-reported LOC, period of impaired consciousness (e.g. confusion) for 24 hrs or less, PTA for 24 hours or less. Def consistent with CDC def.</td>
<td>Those with PTSD likely to be non-white, Hispanic, no grade 12. With PTSD at 6 mos reported stronger beliefs at baseline that the injury would affect their lives, and they were less likely to blame themselves but significantly more likely to blame others for their injury. Those with PTSD described themselves as significantly more worried and less happy; reported having less control in their lives. Self-reported levels of worry and happiness suggest personality differences between those who later developed PTSD and those who did not. Early after injury those that said they would be more impacted by injury were more likely to develop PTSD.</td>
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| 2525 U.S. Army infantry soldiers 3-4 months after their return from a year long deployment to Iraq. Measures: TBI based on 3 questions about mental state after injury, based on definition provided by CDC Combat intensity – Combat Experience Scale Physical Health – Physical health questionnaire (PHQ15) with additional questions regarding PCS (memory, balance, concentration, ringing in the ears, and irritability. RESULTS CONTINUED Headache was only physical health outcome associated with LOC; in contrast, PTSD and depression or both were strongly associated with all the physical health outcomes in these adjust models |
| 124 (4.9%) injury with LOC (lasting between a few seconds and 2-3 minutes); 260 (10.3%) reported injury with alteration in mental status (not LOC); 4 soldiers reported LOC longer than 30 minutes; 435 (17.2%) reported some other injury with no LOC or alteration in mental status Soldiers with mTBI significantly more likely to report high intensity combat, blast mechanism of injury, more than one exposure to explosions, and hospitalizations during deployment; also young and junior in rank. PTSD strongly associated with mTBI; 43.9% of those who reported LOC met the criteria for PTSD, as compared with 27.3% of those with altered mental status, 16.2% with other injuries, 9.1% no injuries. In a logistic-regression model only LOC and combat intensity remained significantly associated with PTSD. Injuries with no LOC associated with Depression; Mental status alteration associated with ONLY PTSD, not depression: same as combat intensity |

MORE RESULTS SEE ARTICLE
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<td>760 U.S. Army soldiers assessed pre- and post-deployment: Outcomes included neuropsychological performances and subjective functional impairment.</td>
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<td>692 TBI – and 68 TBI +</td>
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<td>Most vets had mTBI, but some had more severe TBIs as well (86.7% had mTBI)</td>
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<td>PTSD measured by PCL-C</td>
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<td>Depression measured by CES-D (9 items; Center for Epidemiological Studies Depression Scale)</td>
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<td>NDHS – sustained attention, executive functioning, inhibition, reaction time speed, learning, memory and cognitive efficiency.</td>
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<td>Physical and cognitive health-related functioning – Veterans RAND 12 Item Health Survey (VR-12).</td>
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<td>Hierarchical regression analyses revealed that inter-session TBI, even before adjusting for PTSD and depression was not significantly associated with neuropsychological performances (and there were 12 neurocognitive tests)</td>
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<td>mTBI (and not TBIs of other severity) were associated with decrements in subjective health-related functioning, which remained significant after accounting for psychiatric symptoms. PTSD and Depression associated with both decrements in cognitive tests and self-reported health-related functioning.</td>
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<td>The absence of such associations between TBI status and neuropsychological performance is all the more striking because we did not limit the sample to those unambiguously categorized as mild TBI.</td>
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<td>There was also no significance between time since most significant TBI, prior TBI or single v. multiple inter-session TBIs with outcomes.</td>
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<td>Postdeployment, participants reporting head injury with LOC, 18% screened positive for PTSD, and 31% screened positive for depression</td>
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<tr>
<td>Glaesser, J., Neuner, F., Lutgehetmann, R., Schmidt, R., &amp; Elbert, T. (2004). Posttraumatic stress disorder in patients with traumatic brain injury. BMC Psychiatry, 4(5), 1-6</td>
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<tr>
<td>Barlow-Ogden, K., &amp; Poynter, W. (2012). Mild traumatic brain injury and posttraumatic stress disorder: Investigation of visual attention in Operation Iraqi Freedom/Operation Enduring Freedom veterans. JRRD, 49(7), 1101-1114</td>
</tr>
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</table>
stimuli to either LVF (RH) or RVF (LH)

Three metrics calculated: alerting, executive, and orienting networks.

2 screens: Vision and attention

TBI+PTSD poorer accuracy as well

Found evidence that suggests mTBI+PTSD group had more difficulty covertly shifting attention to the LVF using RH mechanisms, whereas PTSD participants had more difficulty shifting attention to the RVF using LH mechanism (orienting metrics).

Before the study, asked participants whether they noticed any problems or complaints with attention or concentration; complaint or no complaint.

Found a significant VF x patient group interaction effect on accuracy scores (F(1, 14) =11.0, p = .005; n² = 0.44) indicating that participants who complained of attention problems performed significantly worse than the no-complaint participants when stimuli were projected to the LVF, suggesting an RH performance deficit.

RT measures showed same pattern…

No significant differences between control and PTSD regarding attentional performance

| 213 OEF/OIF returning war veterans over two studies… 1: 132 (47% TBI) and 2:81 (44.4% TBI).

Interviews of TBI and PCS and self-reports for PTSD and depression

TBI – Brief Traumatic Brain Injury Screen (B TBIS), which is used by Defense and Veterans Brain Injury Center (DVBIC); head injury that lead to alteration in consciousness, LOC, or PTA |

Among those who screened positive for deployment related TBI, 15.3% denied any PCS that did not overlap with PTSD or depression, 25.5% reported one current non-overlapping PCS, 19.4% reported two current nonoverlapping PCSs, and 39.8% endorsed all three current nonoverlapping PCSs.

Male veterans significantly more likely to screen positive for TBI; nonsignificant trend suggesting that White veterans were also
PCS: dizziness or balance problems, headaches, memory difficulties
PTSD – PCL-M, PTSD if score 50+
Depression – BDI
Combat exposure - FCES

more likely to endorse TBI.

Vets screening + for TBI reported significantly lower levels of education, higher levels of combat exposure, more PTSD symptoms, more depression symptoms than those screened – TBI.

Presence of PCS fully mediated the effects of TBI on current levels of PTSD and depression symptoms, and better accounted for the relations among these variables compared to the two alternative models tested. TBI and combat exposure combined to account for 50% of variance of PCS in model 1, combat exposure, PTSD, and depression combined to account for 40% of variance in model 3, suggesting that PCS and TBI have direct relationship.

PCS better explained by TBI than PTSD or depression.

Model 1: hypothesized full mediation model – PCS fully mediate the effects of TBI on PTSD and depression symptoms
Model 2: partial mediation model
Model 3: PTSD and depression symptoms mediate the effects of TBI status on PCS

<table>
<thead>
<tr>
<th>Article and Authors</th>
<th>Method</th>
<th>Results/Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>longitudinal cohort study. Time 1: 2677 soldiers from National Guard Brigade Combat Team completed a questionnaire 1 month before their return home from an extended 16 month combat deployment to Iraq. 1935 agreed to be invited to participate in future research. After sending out mail, response rate was 953 participants (50%). There was no difference in time 1 and time 2 results.</td>
<td>mTBI only: 42/60 (~70%) reported blasts as most common mechanism for mTBI. 21/26 (~80.8%) of those with mTBI+PTSD report blasts as most common injury. Interestingly, 25/103 (24.3%) in PTSD only group report falls as most common, compared with 75/748 (10% control) and 6/60 (10% TBI) and 9/26 (34.6 mTBI+PTSD)</td>
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<td>PTSD=PCL-M, score of 50+ Depression=BDI2, score of 20+</td>
<td>At time 2, PTSD only group reported more PCS (memory, balance, ringing in ears, concentration, irritability) than TBI group and control. On par with TBI+PTSD, though TBI+PTSD slightly more, except balance problems.</td>
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<td>Time 1= concussion/mTBI, time 2= probable PTSD</td>
<td>Time 1 PCL-M scores more “potent” predictor of all postdeployment PCS than time 1 mTBI status</td>
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<td>4 groups: (1) those who reported no mTBI or PTSD (control group); (2) those who reported mTBI/concussion but no PTSD; (3) those with probable PTSD but no mTBI; (4) those with both mTBI and PTSD. Groups were compared</td>
<td>“After accounting for PTSD, we found that a history of concussion/mTBI alone was not associated with postdeployment PCSs, depression, problematic drinking, nonspecific somatic complaints, social adjustment, or quality of life.”</td>
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<td>Perhaps repeated concussions are a different story.</td>
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**Purpose:** to determine factors associated with chronic headaches.

Cross-sectional, questionnaire-based study conducted with 978 U.S. soldiers screened + with concussion.

MREC – Madigan Redeployment Evaluation of Concussion (0-30) below 25 defined as abnormal cognitive function.

MACE – Military Acute Concussion Evaluation is a neurocognitive screening instrument intended for evaluating soldiers with a deployment related concussion after returning stateside.

PCL – PTSD symptom checklist.

CDH – defined as headaches occurring 15 or more days per month for the previous 3 months Fisher’s exact test was used to test for differences in proportions; unpaired t-test was used to test for differences in ordinal variables with a normal distribution; correlation coefficients were calculated to test the relationship between individual variables were appropriate; P-values less than .05 were considered significant.

Data from CDH group compared with group with episodic headaches not defined as CDH.

196 (20%) met criteria for CDH; 761 (78%) met criteria for episodic headache. Those with CDH had a median of 26.7 headache days per month, those with episodic headaches had 3.3.

55% of CDH group significantly more headache onset within 1 week of concussion over 33% of episodic. Soldiers with CDH reported taking headache medication more than 15 days in a month (49% vs. 2%).

Cognitive scores were the same in both groups as measured by MREC (no significant difference; averages were 26.5 CDH and 27.0 episodic.

Soldiers with CDH higher average scores on PTSD checklist, vs. soldiers with episodic (significant at the P<.0001 level). 41% of CDH group screened + for PTSD based on PCL score greater than 50 vs. 18% of those with episodic headaches.

Migraine prevalent in 66% of CDH population, with 28% having features of migraines. There was a significant difference between CDH group and episodic group (49%).

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<td>PTSD diagnosed in 26 (27.1%) of patients. + predictive power was defined as the probability of PTSD developing when a PTSD symptom was present; negative predictive power was defined as the probability of not developing PTSD when a PTSD symptom was absent.</td>
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<td>Symptoms that had the highest positive predictive powers were intrusive memories (1.00), nightmares (1.00) and emotional reactivity (0.86).</td>
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<td>Evidence against the idea that PTSD cannot occur after severe traumatic brain injury.</td>
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<td>The content of intrusive memories from those with severe TBI was of trauma-related images that they had apparently reconstructed on the basis of information acquired after remittance of PTA. One patient reported that his intrusive memories were of images that he had seen in a photograph of his wrecked car.</td>
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<td>“It is possible that the deficient coping skills associated with severe traumatic brain injury resulted in patients who suffered trauma re-experiencing being unable to manage the distress caused by the symptoms.”</td>
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96 patients, 5-7 months post-injury. Duration of PTA established by Westmead Posttraumatic Amnesia Scale. GCS average score indicate that TBI was severe (8.00). Participants “had no cohesive recall of events that occurred in the first months after the trauma”.

PTSD = PTSD Interview

Individuals with severe TBI are useful population to study implicit memories for traumatic experiences.
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<td>Ruff, R. L., Riechers II, R. G., Wang, X., Piero, T., &amp; Ruff, S. S. (2012). For veterans with mild traumatic brain injury, improved posttraumatic stress disorder severity and sleep correlated with symptomatic improvement. JRRD, 49(9), 1305-1320</td>
<td>63 participants with mTBI related to explosions; headaches, neurological deficits (NDs) and PTSD. Seen on average 2.5 year after their last mTBI. Treated with sleep hygiene counseling and oral prazosin. Monitored headache severity, day time sleepiness (Epworth Sleepiness Scale); cognitive performance using Montreal Cognitive Assessment test. PTSD assessed with PCL-M</td>
<td>Out of 12,046… 473 (3.9%) screened + for TBI 2,144 (17.8%) + for PTSD 644 (5.3%) + for both TBI and PTSD 8,785 (72.9%) had neither TBI or PTSD TBI individuals, with or without PTSD, significantly more likely to be younger and more likely to be in the Marines than sub. Without TBI. presence of TBI without PTSD did not appear to increase the chances of screening + for depression. PTSD was associated with poorer health (self-rated) on the PDHA or PDHRA. All subjects reported high rates of having witnessed someone wounded</td>
</tr>
<tr>
<td>Macera, C. A., Aralis, H. J., MacGregor, A. J., Rauh, M. J., Galarneau, M. R. (2012). Postdeployment symptom changes and traumatic brain injury and/or posttraumatic stress disorder in men. JRRD, 49(8), 1197-1208</td>
<td>TBI = service members were asked if they experienced a blast, explosion, vehicle accident, fragment or bullet wound, or any other injury involving the head. Following + response, they were asked about AOC or LOC. If + response, considered to have screened + for TBI. PTSD = PC-PTSD (primary care PTSD screen). Yes to at least 2/4 questions they screened + 15 symptoms assessed – questions about persistent headaches, sleeping, cognitive functions, ringing in the ears,</td>
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hearing, vision, and musculoskeletal problems. or killed; more than 75% of those with TBI, PTSD, and/or both reported having felt in great danger of being killed. Less common in group with neither TBI or PTSD Military rank associated with TBI and PTSD (officers reported significantly fewer TBI and PTSD)

The added presence of TBI appeared to be associated with an even greater increase in reporting symptoms such as problems sleeping, feeling weak, back pain, ringing in the ears, dimming of vision, and dizziness. Those with neither condition appeared to have reduced symptoms across time, while having either TBI, PTSD, or both was associated with increased or worsening conditions across time (with the exception of MS symptoms).

**NEUROBEHAVIOURAL SYMPTOMS**
See article, RCBIS EBSCOhost.

Does not support finding that once PTSD is taken into account, few symptoms associated with TBI

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<tr>
<th>Maguen, S., Lau, K. M., Madden, E., &amp; Seal, K. (2012). Relationship of screen-based symptoms for mild traumatic brain injury and mental health problems in Iraq and Afghanistan veterans: Distinct or overlapping symptoms?</th>
<th>Factor analytic techniques to differentiate distinct from overlapping screen-based symptoms of TBI, PTSD, and depression in Iraq and Afghanistan veterans. 1,549 veterans OIF/OEF TBI = the VA TBI screen (5 items modified from Brief TBI Screen) PTSD = PC-PTSD (primary care PTSD screen) Depression = PHQ-2</th>
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avoidance, emotional numbing, and hypervigilance)  
Depression (little interest, down and/or hopeless)

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| Wang, Y., Chan, R. C. K., Deng, Y. (2006). Examination of postconcussion-like symptoms in health university students: Relationships to subjective and objective neuropsychological function performance. Archives of Clinical Neuropsychology, 21, 339-347 | 124 university students, considered healthy, participated (49 m, 75 f). This sample was compared to a sample of 84 Hong Kong patients with brain injury (severity?). PCS – Rivermead Post-Concussion Symptoms Questionnaire (RPQ) measured frequency and intensity of symptoms (16 most common symptoms) Neuropsychological tests: attention, memory, and executive function Depression – BDI Lower 25% of participants as low symptom reporters and the upper 25% of P as high symptom reporters (28 v 30). | Subgroup of individuals falling in the mild depression range (N=16); cut off score of 14. Reporting average scores on RPQ, the whole group of participants (minus subgroup of depression) had 9.38; depressed subgroup (16) had 19.38, which is 10 points higher. Both groups reported same “most common” symptoms, but the depression subgroup had higher frequencies. “At the total score level, PCS were not much evident in the students. However, the students had relatively high endorsements of specific symptoms such as fatigue (76.9%), longer time to think (60.3) poor concentration (58.7%), sleep disturbance (50.4%) and frustration and impatience (46.3%). In mildly depressed + group, fatigue (93.7), frustration and impatience (93.7%), poor concentration (87.5%), sleep disturbance (81.2%), and longer time to think (81.2%).
<table>
<thead>
<tr>
<th>Source</th>
<th>Study Design</th>
<th>Participants</th>
<th>Symptoms</th>
<th>Findings</th>
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<td>Chan, R. C. K. (2010)</td>
<td>Cross sectional study, purpose to explore the base rates of PCS among participants without head injury.</td>
<td>85 participants without head injury, other psychiatric illnesses.</td>
<td>PCS – British Columbia Post-Concussion Symptom Inventory (BC-PSI) 16 items, 13 symptoms</td>
<td>The most commonly reported symptoms were longer time to think (65.9%), poor concentration (58.9%) and forgetfulness (58.9%), fatigue easily (53.5%), sleep disturbance (50.6%), and irritability (43.6%).</td>
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<td>Garden, N., &amp; Sullivan, K. A. (2010)</td>
<td>96 “healthy” participants (49 university students, 47 from general community).</td>
<td>PCS endorsement rate ranged from 37% to 81%; most frequently endorsed symptoms were headache (81%), fatigue (81%), irritability (78%), feeling nervous or tense (76%), poor concentration (73%), and feeling sad (70%).</td>
<td>92% endorsed 3 or more symptoms, 74% endorsed 7 or more symptoms, 30% endorsed 11/13 symptoms.</td>
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<td>Subgroup (N=24) with elevated BDI scores indicating mild depression (14+)</td>
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**Purpose:** assess the prevalence of PCS-like symptoms in depressed individuals. 64 participants with depression (via physician or Structured Clinical Interview for DSM-IV [SCID]).

PCS - British Columbia Postconcussion Symptom Inventory (BC-PSI), 16 items unpublished – frequency and intensity of 13 symptoms of PCS.

No significant difference between men and women on PCS scores. 13/64 reported history of concussion or TBI with LOC at some point in their lives. No significant difference between those with and without concussion with respect to PCS scores. 89% = 3+ symptoms 75% = 7+ symptoms 19% = 11+ (Liberal^, see article for conservative rates) “When looking at more clinically relevant symptoms” (?) or symptoms with more severity… 72% = 3+ symptoms 27% = 7+ symptoms 2% = 11+

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<td>Iverson, G. L. (2006). Misdiagnosis of the persistent postconcussion syndrome in patients with depression. Archives of Clinical Neuropsychology, 21, 303-310. Doi: 10.1016/j.acn.2005.12.008</td>
<td>Purpose: assess the prevalence of PCS-like symptoms in depressed individuals. 64 participants with depression (via physician or Structured Clinical Interview for DSM-IV [SCID]). PCS - British Columbia Postconcussion Symptom Inventory (BC-PSI), 16 items unpublished – frequency and intensity of 13 symptoms of PCS.</td>
<td>No significant difference between men and women on PCS scores. 13/64 reported history of concussion or TBI with LOC at some point in their lives. No significant difference between those with and without concussion with respect to PCS scores. 89% = 3+ symptoms 75% = 7+ symptoms 19% = 11+ (Liberal^, see article for conservative rates) “When looking at more clinically relevant symptoms” (?) or symptoms with more severity… 72% = 3+ symptoms 27% = 7+ symptoms 2% = 11+</td>
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Cross-sectional study of 106 participants with mTBI and 244 without head injury. Online questionnaire collected data about post-concussion symptoms, cognitive failures, anxiety, depression, and PTSD. Variability in the sample was addressed by splitting by PCS diagnosis into 4 groups: mTBI+PCS, mTBI-PCS, Control+PCS, and Control-PCS. PCS diagnosed with ICD-10 criteria.

mTBI defined using ICD-10 criteria: had to report one of the following: dizziness/confusion, LOC for 30 minutes or less, PTA for less than 24 hours.

PCS diagnosed based on ICD-10 criteria, RPQ used to assess PCS. PCS was diagnosed if reported 3+ symptoms.

CFQ (cognitive failures questionnaire), PTSD, IES-R (impact of event scale revised), daytime sleepiness (Epworth Sleepiness Scale ESS), anxiety and depression (Hospital Anxiety and Depression Scale: HADS)

Two groups: TBI and no TBI. Further divided…

Authors also sought to determine if diagnostic criteria (definition) had an effect on control populations, as well as those with mTBI.

mTBI+PCS (n=33), mTBI-PCS (n=73), control+PCS (n=83), control-PCS (n=161).

PTSD, as measured by the IES-R sum score, was present to a greater degree in the mTBI+PCS group than the mTBI-PCS group. (suggests that PCS plays role in development of PTSD?)

There was no statistical difference between the mTBI group and the control group in terms of PCS prevalence (31% mTBI+PCS and 34% control).

ANOVA revealed that individuals with mTBI had substantially greater scores on the CFQ (cognitive failures) than the control group.

Individuals with PCS had higher scores on all measures (anxiety, depression, CFQ, and RPQ). Individuals with PCS scored higher on each item regardless of whether they had mTBI. Those with mTBI had significantly higher scores for headaches, nausea, taking longer to think, light sensitivity, and double vision. Significant interaction between PCS and mTBI was observed for headaches.

TBI participants had higher average scores on majority of the somatic symptoms and 1 of the cognitive symptoms (taking longer to think).

Headaches and cognitive “failures” make up most difference. Therefore, combining these symptoms in the definition may create a
more precise definition and diagnosis of PCS.

Those in mTBI-PCS group more likely to have dyslexia (does this have anything to do with more or less reporting of PCS?)

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<td>MacGregor, A. J., Dougherty, A. L., Tang, J. J., &amp; Galarneu, M. R. (2012). Postconcussive symptom reporting among U.S. combat veterans with mild traumatic brain injury from Operation Iraqi Freedom. Journal of Head Trauma Rehabilitation,</td>
<td>Postdeployment health assessments and reassessments used to assess self-reported health and PCS (PDHA, PDHRA). Both instruments contain validated PTSD and depression instruments. PCS symptoms identified from PDHRA. EMED (deployment health database) was queried for PDHA and PDHRA. Both surveys had been completed within 1 year. 992 injured personnel, 334 with mTBI and 658 with non-head injury. Symptoms = headache, dizziness, memory problems, sleep issues, back pain, joint and muscle pain, weakness, irritability, and tinnitus. Variable created for PCS+3</td>
<td>Those with mTBI: 92/334 had concussion with LOC of 30 mins or less, 1 had concussion with LOC of 31 to 59 minutes, 10 had concussion with LOC of unspecified duration, 150 had concussion with no LOC, and 81 had mTBI characterized as “concussion, unspecified”. 88.9% of mTBI cases were battle related blasts (mechanism of injury). 49.7% of TBI cases had 4 “combat exposures” 27.8% screened + for PTSD; 20.7% screened + depression.</td>
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123 with mTBI and 100 in control group that have experienced trauma not resulting in a brain injury. Measured in ED 1 and 3 months post-injury.

ImPACT Post-Concussional Symptom Scale and cognitive concussion battery, (including attention, Verbal and Visual memory, processing speed and reaction time modules, pre- and postinjury SF-36 and MINI psychiatric status ratings, VAS Pain Inventory, Hospital Anxiety and Depression Scale, PTSD checklist-specific, and revised social readjustment scale.

RESULTS CONTINUED

3 factors associated with PCS at 1 week after injury; being female, presence of pre-injury psychiatric disorder, and having amTBI. The experience of mTBI renders a person 3 times more likely to experience PCS in first week after injury than a general trauma patient requiring no surgery.

“Therefore it would seem erroneous to conclude that mTBI does not cause PCS in the early days after injury”

Performance on cognitive tests failed to predict PCS at 1 week and 3 month post-injury. This suggests that PCS are not influence by cognitive deficits mTBI patients may experience.

mTBI does contribute significantly to PCS in the acute phase, but psychiatric disorders more significantly related to

111/123 completed the 1 week assessment, and 90 completed the 3 month follow-up. 90/100 TCs completed the 1 week follow-up, and 80 (80%) completed the 3 month follow up. No significant difference between genders.

Those with mTBI more likely to be assaulted than Trauma Control group [16(13.3)* vs. 2(2)] as well as be in motor vehicle collisions 49(40.9) vs. 28(28). Soft tissue laceration/damage 97(78.9)** vs. 59(59) History of head injury 51(41.5) vs. 28(28) Involved in litigation 15(17.2) vs. 7(8.9)

During the acute phase (ED) and 1 week post-injury, TBI group more than double scores for PCS than TC group 32.5 vs. 13.5 (acute) and 16 vs. 7.5 (1 week). No significant difference between scores on 3 month follow up on any measure (depression and anxiety). TBI group reported decreased PCS reporting substantially from 16 at 1 week to 4 at 3 months (same as TC).

No association between education and PCS at anytime; history of head injuries was not significant predictor of PCS at any time; nor was there an association between litigation and PCS at 3 month follow-up.

mTBI predictor of PCS during the acute phase after injury, but not at 3 months post-injury. At this 3 month time mark, psychiatric factors were strongest predictors for PCS.

37 hospitalized mTBI subjects vs. 39 hospitalized trauma subjects (4.5 days after injury).

Emergency Department

mTBI group performed significantly worse on all cognitive measures, but did not differ in report of PCS. Cog. Performance unrelated to pain severity and emotional distress. PCS unrelated to pain severity, but consistently related to emotional distress. 86% of mTBI in MVC, 65% of trauma control PCS scores (frequency, intensity, duration) unrelated to the SF-36 Bodily Pain rating; no relationship between PCS and Cognitive Index composite variable. PCS significantly correlated with the SF-36 Mental Health ratings (higher PCS scores associated with lower (worse) SF-36 Mental Health ratings.

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<td>Sheedy, J., Geffen, G., Donnelly, J., &amp; Faux, S. (2006). Emergency department assessment of mild traumatic brain injury and prediction of post-concussion symptoms at one month post injury. Journal of Clinical and Experimental Neuropsychology, 28, 755-772</td>
<td>mTBI participants compared to individuals with orthopedic injuries… neurocognitive testing 29 ind in mTBI and 30 ind in ortho injury group. 30 ind in balance control group. RSC – neurocognitive test computer based series of tasks presented through headphones. 5 tests: immediate and delayed</td>
<td>29 participants in sample, 21 experienced LOC (72.4%), 13 (44.8%) had at least one previous head injury. Most common mechanism: assaults (34.5%), low falls (20.7%) and collision with an object (17.2%). Road trauma (MVC, cyclists, pedestrians) accounted for 13.8% of the group.</td>
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verbal recall, speed of sentence comprehension, orientation and the Digit Symbol Substitution Test (DSST) from the WAIS-R
BESS (balance error scoring system)
Pain (visual analog scale)
Alcohol
RPQ – PCS

Emergency Department

RESULTS CONTINUED
The authors conclude that the most reported symptoms appear to be associated with neurocognitive deficits than emotional problems. (however, this assumption implies that neurocog deficits cannot be mediated by emotional problems, which does not appear to be the case).

Although PCS scores correlated with RPQ scores (neurocog scores).

Control group (ortho) completed significantly more years of school. Concussed group reported significantly more history of drinking than control (balance) group (51.7% vs. 6.7%). No difference in alcohol consumption between Ortho and mTBI groups.

Within the mTBI group, males RSC performance tended to be worse, but this was a non-significant trend.

mTBI as a whole compared with Ortho group performed significantly worse on tests: mTBI group recalled significantly fewer immediate memory words, delayed words, judged fewer sentences, and completed fewer DSST items. After adjusting for alcohol consumption/no. of standard drinks, the mTBI group recalled significantly fewer immediate memory items. No other sig. Differences.

mTBI could not perform balance tests, given maximum scores. mTBI group performed significantly worse on balance tests than balance control group. This remained consistent even when accounting for alcohol consumption. Balance issues likely to be related to pathophysiology of injury.

After about 30 days follow up was performed. mTBI reported significantly more PCS than ortho group: 17.9 (14.32) and 5.76 (5.44). PCS reported in greatest frequency were fatigue, frustration, memory problems, concentration difficulty, slower thinking, headache/irritability