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Predictors of Depression after Traumatic Brain Injury during Early and Late Recovery

By

Elmar Gardizi

A Dissertation

Submitted to the Faculty of Graduate Studies through the Department of Psychology in Partial
Fulfillment of the Requirements for the Degree of Doctor of Philosophy at the University of
Windsor

Windsor, Ontario, Canada

2015

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Predictors of Depression after Traumatic Brain Injury during Early and Late Recovery

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ABSTRACT

Cognitive, neurological, and psychosocial predictors of depression after TBI were investigated in an Early and a Late Recovery group. The Early Recovery group consisted of 80 participants who were 1.3 years removed from their TBI, while the Late Recovery Group consisted of 107 participants who were 10.1 years removed from their TBI. Participants were enrolled in the Southeastern Michigan Traumatic Brain Injury System (SEMTBIS). Depression was measured using the Brief Symptom Inventory Depression subscale. The cognitive domains that were assessed included attention, executive functioning, and memory. Injury severity was used as a measure of neurological damage while psychosocial variables of interest included emotion-focused coping, problem-focused coping, perceived social support, and alcohol use. Five models were run using multiple linear regression and the best fitting models were selected using the Bayesian Information Criterion. For the Early Recovery group, the model that included only psychosocial variables was the best fit. Specifically, the use of emotion-focused coping and lack of perceived social support was associated with higher levels of depression. Conversely, the use of problem-focused coping was associated with lower levels of depression. For the Late Recovery group, a model that included cognitive functioning and psychosocial variables was the best fit. Specifically, the use of emotion-focused coping, lack of perceived social support, and better attention ability were associated with higher levels of depression. The findings suggest that psychosocial variables may be related to depression during early recovery. With time however, the role of cognitive functioning, namely better attention, may become an important factor in predicting depression. Also, the influence of problem-focused coping on depression may diminish with time. Conversely, emotion-focused coping and perceived social support may

become more important in predicting depression as time since injury increases. Generally, the results imply that treatment protocols that focus on improving coping and social skills throughout the recovery process may improve outcome. Similarly, cognitive screening several years after TBI may be useful in identifying persons who may be susceptible to the development of depression. Lastly, possible changes in the effectiveness of problem-focused coping over time may provide evidence in favour of creating interventions that are more relevant to specific stages of recovery.

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Predictors of Depression after Traumatic Brain Injury during Early and Late Recovery

Each year, 1.7 million people sustain a traumatic brain injury (TBI) in the United States (Faul, Xu, Wald, & Coronado, 2010). Approximately, 1.4 million of these individuals will be treated and released from emergency departments (ED), 275,000 will be hospitalized and discharged alive, and 50,000 will die as a result of their injuries (Faul et al., 2010). Overall, TBI accounts for 30% of injury-related deaths in the United States. Of those who survive, many are permanently disabled; in fact, estimates indicate that over 5 million Americans may be living with TBI related disabilities (Thurman, Alverson, Dunn, Guerrero, & Snieszek, 1999). Of these disabilities, psychological disturbances can be among the most impactful sequelae of TBI and may manifest in a variety of emotional and behavioural abnormalities; the most prevalent is clinical depression (Kennedy et al., 2005). Given the strong association between depression and TBI, it is critical that scientists and clinicians better understand the factors that may be predictive of this condition and how their impact may change during different stages of recovery.

Traumatic Brain Injury

Traumatic brain injury is defined as an injury resulting from external impact and/or rapid acceleration/deceleration of the brain (Lezak, Howieson, Bigler, & Tranel, 2012). The majority of TBIs are closed-head injuries meaning that the skull remains generally intact (Lezak et al., 2012). Open-head injuries have higher fatality rates and occur when the skull and the protective covering of the brain (i.e., dura) are crushed or penetrated by an external force (Lezak et al., 2012). The neurological consequences of a TBI occur in two stages: the primary injury and the secondary injury. The primary injury is used to describe the neurological damage resulting from

the external force. Primary injury complications include: brain contusions and lacerations, diffuse axonal damage, hematomas, and intracranial bleeding (Lucas & Addeo, 2006). The secondary injury is longer in duration and can be more harmful relative to the primary injury (Maas et al, 2008; Povlishock & Katz, 2005). Common secondary complications include: edema (swelling), ischemia (insufficient blood supply), brain infection, seizures, hypoxia (insufficient oxygen), and hydrocephalus (accumulation of cerebrospinal fluid; Lezak et al., 2012; Lucas & Addeo, 2009). On a cellular level, TBI can result in an initial increase in neurotransmitter (NT) levels, particularly glutamate and acetylcholine. This causes an influx of intracellular calcium into neurons which leads to cytotoxic injury and eventually cell death (McAllister, 2011). With time, these NT levels may become chronically depleted which may result in behavioural and emotional abnormalities (McAllister, 2011).

The severity of a TBI can be classified based on depth of coma and/or length of posttraumatic amnesia (PTA). The Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) is the most frequently used instrument to measure depth of coma. It yields a total score based on eye opening as well as verbal and motor responses. The scores range from 3 to 15 and the grading system is as follows: (a) mild TBI = 13-15; (b) moderate TBI = 9-12; and (c) severe TBI = 8 or fewer (Lucas & Addeo, 2006). Length of PTA is a term that describes the loss of memory for events that occur immediately after a TBI. It can also be used to classify injury severity and the grading system is as follows: (a) mild TBI = PTA less than 1 hour; (b) moderate TBI = PTA between 1 to 24 hours; and (c) severe TBI = PTA longer than 24 hours (Lezak et al., 2012). Individuals that are 75 or older and children aged 15-19 are most likely to sustain a TBI (Faul et al., 2010). From 2002-2006, the leading causes of TBI were falls (35.2%), motor vehicle accidents (17.3%), being struck by or against objects (16.5%), and assaults (10.2% ; Faul et al.,

2010). The cause of the remaining injuries was unknown. It should be noted that the cause of a TBI can vary according to the age group in question. For example, MVA and assault rates are highest among young adults while fall rates are highest among children and older adults. Being male also increases the lifetime risk for sustaining a TBI. In particular, men account for 62% of TBI-related hospitalizations in the United States (Faul et al., 2010). The higher rate for men has generally been attributed to their tendency to engage in more risky behavior relative to women (Coronado, McGuire, Faul, Sugerman, & Pearson, 2013). With respect to race and ethnicity, the highest rates of TBI-related emergency department (ED) visits were reported for African Americans and Caucasians followed by American Indian, Alaska Native, and Asian or Pacific Islander (Faul et al., 2010). Lower socioeconomic status, as measured by employment, education, and income level, has been associated with an elevated risk for TBI (Seel et al., 2003). Moreover, alcohol use is also a significant risk factor for TBI. Estimates indicate that 25 to 50% of persons who sustain a TBI may be intoxicated at the time of their injury (Shandro et al., 2009).

TBI and Depression

Traumatic brain injury related sequelae are wide ranging and can include cognitive, physical, emotional, interpersonal, and occupational disturbances. Of these, depression or post-TBI depression, as it will be referred to here, is one of the most common (e.g., Bombardier et al., 2010; Dikmen, Bombardier, Machamer, Fann, & Temkin, 2004; Jorge et al., 1993; Kennedy et al., 2005; Ownsworth & Oei, 1998) and persistent complications (Hoofien, Gilboa, Vakil, & Donovick, 2001). The *Diagnostic and Statistical Manual of Mental Disorders* defines Major depression as the presence of 5 or more of the following symptoms over a 2-week period:

(1) Depressed mood..., (2) markedly diminished interest or pleasure..., (3) significant weight loss when not dieting or weight gain..., (4) insomnia or hypersomnia nearly every day, (5) psychomotor agitation or retardation..., (6) fatigue or loss of energy..., (7) feelings of worthlessness or excessive guilt..., (8) diminished ability to think or concentrate or indecisiveness..., and (9) recurrent thoughts of death (DSM-IV; American Psychiatric Association, 2000, pp. 356).

Studies have reported that the prevalence of depression following TBI can range from 6% to 77% (as cited in Seel et al., 2003). One of the reasons for this variability is that many of the symptoms of depression overlap with symptoms that are experienced after acute TBI. For example, apathy, changes in appetite, and sleep disturbance are commonly reported by TBI patients (Babin, 2003). Nevertheless, a review by Rogers and Read (2007) examined data from several studies that focused on post-TBI depression. They found that the prevalence rate of depression after TBI is approximately 25%. In comparison, the life time prevalence rate for depression in the general population has been estimated at 16% (Kessler et al., 2003). Even when compared with other traumatic injury patient groups, the prevalence of depression continues to be elevated in persons with TBI (Jorge et al., 2004). This implies that factors that are unique to TBI such as neurological and cognitive disturbances may influence the development of depression. As it is currently conceptualized, however, the etiology of post-TBI depression has generally been attributed to psychosocial factors (Ownsworth & Oei, 1998). More specifically, the role that factors such as alcohol use, coping, and social support have on post-TBI depression has been well established (Rogers & Read, 2007). On the other hand, studies that have examined the influence of neurological factors such as injury severity have yielded mixed findings.

Similarly, the role of cognitive functioning on post-TBI depression is not well known. Evidence also suggests that the contribution of psychosocial, neurological, and cognitive factors may vary with time (Jorge, Robinson, Arndt & Forrester, 1993) such that the etiology of depression after acute TBI may be different than the etiology of depression several years after the injury. In what follows the literature regarding post-TBI depression will be discussed with a focus on the factors that may be responsible for the development of this condition and how their influence can change at different stages of recovery.

Psychosocial Factors

Coping. Sustaining a TBI can be a life altering event; not surprisingly, the ability to cope with the changes post injury is critical to recovery. Historically, coping strategies have been categorized in two ways. The first is problem-focused coping, which involves dealing directly with the environmental stressor. Individuals who use this strategy either seek out more information and skills in order to manage the situation (i.e., self-focused) or they alter the situation directly (Anson & Ponsford, 2006; Lazarus & Folkman, 1984). The second is, emotion-focused coping. This strategy involves the use of avoidance and denial strategies as a means of altering the way the situation is attended to or reappraising the situation or using acceptance in order to help interpret the situation differently (Anson & Ponsford, 2006; Lazarus & Folkman, 1984).

Emotion-focused coping is commonly used by persons with TBI and this coping strategy has been linked to depression (Tomberg, Toomela, Pulver, & Tikk, 2005). For example, Curran, Ponsford, and Crowe (2000) examined the influence of coping strategies on emotional outcome in TBI patients who were 1-5 years post injury. Level of depression was measured using the

Beck Depression Inventory (BDI). It was found that emotion-focused coping was associated with higher levels of depression relative to problem-focused coping.

Kendall, Shum, Lack, Bull, and Fee (2001) used a contextually sensitive assessment method to study coping styles in a TBI sample. Participants were presented with stressful video based scenarios and were asked to record how they would respond in that situation. The authors took these responses and classified them into four categories; these included emotional, problem, active, and passive coping styles. The use of active-problem -focused strategies was associated with higher self-esteem and positive affect; whereas, passive-emotion =focused strategies were associated with poorer self-esteem and negative affect.

The relationship between coping and depression was further demonstrated by Anson and Ponsford (2006). Their sample consisted of 33 individuals who had sustained TBI between 1.5 months and 7 years previously and had a mean PTA duration of 32 days. A variety of self-report measures were used in order to assess for coping style, depression, anxiety, anger, and self-esteem. They reported that over 50% of their sample endorsed clinically significant levels of depression and that emotion-focused coping was associated with higher levels of depression. In comparison, problem-focused coping was associated with higher self-esteem.

It is important to note, however, that problem-focused coping can be maladaptive in some cases. For instance, Kendall and Terry (2008) used a longitudinal design to study the relationship between coping and emotional outcome. Their sample consisted of 90 TBI patients with a mean GCS of 8.18 ($SD = 4.62$). Emotional outcome was measured using the Delusions-Symptom-States Inventory/States of Anxiety and Depression (DSSI/sAD; Bedford, Foulds, & Sheffield, 1976). Contrary to previously reviewed studies, they reported that problem-focused coping was

not associated with emotional well-being and that persistent use of this strategy can result in emotional distress in the long-term. A plausible explanation for this finding is that persons with TBI may not be as effective at using problem-focused coping strategies due to their diminished cognitive resources. By adopting this approach, these individuals may encounter frequent failure at their attempts to solve problems, which may lead to frustration, helplessness, and depression (Kendall et al., 2001; Kendall & Terry, 2008).

The reviewed studies emphasize the important role that coping can play in depression after TBI, although the effectiveness of specific coping styles (i.e., problem-focused vs. emotion-focused) requires further examination. Similarly, the extent to which coping strategies change over time and how this influences depression is not well known. The few studies that have focused on this issue suggest that maladaptive coping styles increase with time (Kendall & Terry, 2008; Wolters, Stapert, Brands, & van Heugten, 2010). More specifically, persons with TBI may be at an increased risk to resort to drugs, alcohol, and other avoidant-type strategies as time since injury increases (Tomberg, Toomela, Ennok, & Tikk, 2007).

Social support. Similar to coping, social support also influences recovery after TBI. Unfortunately, social isolation and loss of social contact are frequently reported within this population (Morton & Wehman, 1995). For instance, Oddy, Humphrey, and Uttley (1978) assessed changes in social relationships over 10 years in 49 TBI survivors. Information was obtained from a close family member or spouse in order to increase objectivity. The first follow-up occurred at 6 months and it was found that there was a significant reduction in friendships over this time span. At 12 months, the number of friendships continued to decrease and the participants received fewer visits. At 2 years participants continued to experience difficulties in their social life. They reported fewer relationships and did not engage in leisure activities when

compared to matched controls. Kozloff (1987) used a longitudinal design in order to study the social network characteristics of TBI survivors. The 37 participants were separated into two groups: an early recovery group (average 3 months post-injury) and a late recovery group (average 17 month post-injury). Regardless of time since injury, social networks reduced in size over time and most of the people lost were non-family members. Finset et al., (1995) used interviews and self-report questionnaires to examine social networks in 70 patients with severe TBI. Approximately 57% of the sample reported that their social networks had reduced following their injury while 33% reported that they did not have any close relationships. Zencius and Wesolowski (1999) compared the social networks of 70 TBI survivors living in a rehabilitation setting with those of non-injured people. It was found that the social support network of TBI survivors was 3-4 times smaller relative to non-injured persons; furthermore, the composition of the social networks for persons with TBI consisted mostly of family members. More recently, Strandberg (2009) used a qualitative design to examine the consequences of TBI in 15 individuals. A varied case sampling procedure was used, meaning that the participants differed on key variables of interest such as injury severity, age, sex, and time post-injury. In-depth interviews were conducted which focused on several themes, one of which included support from society. Most of the participants reported that social interaction with professional care providers, relatives, and friends was important and was altered to some degree after the brain injury. Relationships with friends had changed or been lost, which led to a reduction in their social network. Conversely, relationships with family members had improved despite the added burden of caregiver responsibility.

Social support is particularly important for persons with TBI because of its strong connection to psychological well-being. For example, Douglas and Spellacy (2000) used the

Instrumental-Expressive Social Support Scale (IESSS; Ensel & Woelfel, 1986) to measure the association between perceived social support and depression in 35 individuals with a history of TBI. Time post-injury varied from 3.5 to 10 years and severity of injury for all subjects was less than 7 days PTA. It was found that 60% of the sample was depressed and that perceived social support contributed significantly to depression. Gomez-Hernandez, Max, Kosier, Paradiso, and Robinson (1997) also examined the relationship between psychosocial factors and depression post-TBI. They interviewed 65 patients who were also administered the Hamilton Depression Rating Scale and the Social Functioning Exam at 3, 6, 9, and 12 months. In addition to fear of job loss, lack of personal close relationships was predictive of depression at 6, 9, and 12 months after TBI. As part of their study, Tomberg et al. (2007) sought to examine changes in social support and health related quality of life following TBI. Their sample consisted of 31 patients who were assessed at 2.3 and 5.7 years after TBI. Social support was measured using the Brief Social Support Questionnaire (SSQ; Sarason, Levine, Basham, & Sarason, 1983) and health related quality of life was measured using the RAND-36 survey. The authors reported that satisfaction with social support decreased over time and that social support was positively correlated with emotional-well being and general health. Similar results were reported by Smith, Magill-Evans, and Brintnell (1998) who examined the long-term impact of TBI on life satisfaction. Their sample consisted of 43 adults who had sustained TBI an average of 7 years previously. Participants completed a number of self-report questionnaires including measures of social support. The authors found that the strongest predictors of life satisfaction were perceived psychosocial dysfunction and perceived social support. Together, these variables accounted for 35% of the total variance.

In addition to demonstrating the direct relationship between social support and post-TBI depression, the reviewed findings indicate that social support may diminish with time (Godfrey & Shum, 2000; Kozloff, 1987). Part of the reason for this may be that friends and intimate partners unrealistically expect persons with TBI to reach pre-injury levels of functioning. When this does not happen, they may move on, which explains why the social network of TBI survivors is mainly composed of family members (Kozloff, 1987; Zencius & Wesolowski, 1999). Additionally, many individuals who sustain moderate-to-severe TBI engage in fewer social activities in general (Doig, Fleming, & Tooth, 2001; Wise et al., 2010). This isolation likely plays a role in the reduction of their social networks.

Alcohol use. Pre-injury heavy drinking has also been implicated as a risk factor for TBI (Taylor, Kreutzer, Demm, & Meade 2003). In fact, estimates indicate that between 50 to 60% of individuals who sustain a TBI may have a substance abuse problem and more than half of these individuals were intoxicated at the time of their injury (Shandro et al., 2009; West, 2011). Alcohol use is an important prognostic factor to consider because it can impact many aspects of recovery including disability, cognition, return to work, social functioning, and neurological changes (Glucksman, 1994; Jorge, 2005; Kelly, Johnson, Knoller, Drubach, & Winslow; 1997; Kreutzer, Witol, & Marwitz, 1996; Sparadeo & Gill, 1989). Similarly, alcohol use has been shown to be related to depression. For instance, the development of depression can be preceded by a substance abuse disorder in over 50% of cases (Kessler et al., 2003) which makes it the 3rd most common co-occurring disorder with depression (Kessler et al., 2003; Seel et al., 2010).

Dikmen et al., (2003) examined the risk factors and phenomenology of depression 3 to 5 years after TBI. One of the risk factors that they focused on was pre-injury substance abuse as measured by the Short Michigan Alcoholism Screening Test (SMAST; Selzer, Vinokur, & van

Rooijen, 1975). Depression was measured using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). Overall, it was found that higher rates of pre-injury substance abuse were associated with higher rates of depression. In a similar study, Hart et al., (2011) investigated the role of substance abuse on minor and major depression after TBI. Problematic substance abuse was coded as yes/no. The presence of depression was measured using the PHQ-9 (Kroenke, Spitzer, & Williams, 2001), which is a self-report questionnaire based on the DSM-IV. The findings revealed that patients with minor and major depression were significantly more likely to have substance abuse problems. Paul (1992) examined the prevalence of and risk factors associated with depression in a sample of 66 patients with acute TBI. The Hamilton Rating Scale (Hamilton, 1960) was used to measure depression while the presence of pre-injury alcohol problems was assessed by clinical interview. Seventeen of the patients met the criteria for depression and it was also found that poor social functioning, which included alcohol problems, was predictive of depression. In further support of this, McCarthy et al. (2006) examined the self-reported psychosocial health of persons with TBI. Archival data from 7612 participants from 62 acute care facilities were used. Psychosocial functioning was measured using scales from the SF-36 (Ware, Snow, & Kosinski, 1993) which also assesses symptoms of depression. The results showed that participants with pre-existing substance abuse problems, including heavy drinking, were more likely to report poorer psychosocial functioning.

Alcohol use *following* TBI is also associated with depression. To demonstrate this, Horner et al. (2005) examined the patterns of alcohol use 1-year after TBI. This was a population-based, epidemiological study that used data from 1606 adults who had a positive history of TBI. Telephone interviews were used to classify drinking patterns based on heavy use, moderate use, or light/abstinent use. Approximately 15.4% of the sample reported heavy

drinking the month before the interview and one of the factors associated with frequent alcohol use was the diagnosis of depression. Given the strong relationship between alcohol use and post-TBI depression, it is surprising that studies have yet to examine how this relationship changes over time. It is known however, that early in recovery most persons with TBI will reduce their alcohol intake significantly or engage in abstinence altogether (Corrigan, 1995; Dikmen, Machamer, Donovan, Winn, & Temkin, 1995). With time however, alcohol use may begin to increase (Corrigan, Smith-Knapp, & Granger, 1998; Taylor et al., 2003), and in some cases may reach pre-injury levels (Kolakowsky-Hayner et al., 1999). To illustrate this, Ponsford, Whelan-Goodinson, and Bahar-Fuchs (2007) examined alcohol use over a 3-year period after TBI. During the first year, they found that alcohol use declined; however, by the second year, nearly 26% of their participants were drinking at levels indicative of alcohol abuse.

Injury Severity

In contrast to coping, social support, and alcohol use, the relationship between injury severity and depression after TBI is less clear. This is surprising given that moderate-to-severe TBI is known to be associated with diffuse pathology in the frontotemporal regions of the brain (Lezak et al., 2012) and that these areas play a critical role in emotional functioning (Olson, Plotzker, & Ezzyat, 2007; Stuss & Knight, 2002). In addition, severe TBI can cause damage to the hypothalamic-pituitary-adrenal axis (HPA; Powner, Boccalandro, Alp, & Vollmer, 2006). This region of the brain comprises part of the neuroendocrine system which regulates many functions including mood and emotions. In particular, hyperactivity of the HPA axis has been consistently linked to major depression (Pariante & Lightman, 2008). Severe TBI can also lead to NT disturbances. During the initial stages of a TBI, there is an excess of excitatory NTs; however, during the course of recovery, these levels may become chronically depleted (Jorge &

Starkstein, 2005). This is important given the role that NTs such as serotonin, dopamine, and acetylcholine play in depression (Baldwin & Rudge, 1995; Janowski, El-Yousef, & Davis, 1974). Despite these neurological changes however, the severity of a TBI has not always been shown to predict depression.

For example, Rapoport, McCauley, Levin, Song, and Feinstein (2002) examined the role that injury severity plays in neurobehavioral outcome. The GCS was used to measure injury severity and neurobehavioral outcome was measured using the Neurobehavioral Rating Scale Revised (NRS-R; McCauley et al., 2001). The NRS-R measures cognitive, behavioral, and emotional sequelae that are commonly experienced after TBI. Their results revealed that severe TBI was associated with behavioral and cognitive dysfunction; however, it was not predictive of emotional functioning. Consistent with this, Malec, Brown, Moessner, Stump, and Monahan (2010) used structural equation modelling to evaluate a model for post-TBI depression in a sample of 158 adults. They concluded that injury severity as measured by length of PTA was not predictive of depression. As part of their multicenter study, Seel et al. (2003) also examined the correlation between injury severity and depressive symptoms. They used several variables to measure injury severity including PTA, GCS, length of hospital stay, and disability; while depressive symptoms were measured using the Neurobehavioral Functioning Inventory (NFI; Kreutzer, Seel, & Marwitz, 1999). Overall, it was found that depressive symptoms were not associated with any of the injury severity variables.

The uncertain role that injury severity plays in post-TBI depression is further illustrated by studies that report an inverse-dose-response relationship. For instance, Glenn, O'Neil-Pirozzi, Goldstein, Burke, and Jacob (2001) used the Beck Depression Inventory-II (BDI-II) to examine the incidence of depression and its predictors in 41 outpatients who had sustained TBI. They

found that mild TBI, as classified by the GCS, was more predictive of depression than were more severe injuries. Similarly, Dikmen et al. (2004) examined the relationship between several indices of injury severity such as PTA, GCS, and time to follow commands on post-TBI depression. Their results showed that greater depressive symptoms were associated with milder injuries.

Conversely, other studies have demonstrated that severe TBI is related to post-TBI depression. For instance, Holsinger et al. (2002) examined the medical records of WWII veterans 50 years after they suffered TBI and compared them with veterans who were hospitalized for other ailments (e.g., pneumonia, lacerations, puncture, and incision wounds). Participants were given a structured telephone interview to determine extent of depressive symptoms while injury severity was classified as follows: mild = loss of consciousness less than 30 minutes; moderate = loss of consciousness greater than 30 minutes but less than 24 hours; and severe = loss of consciousness greater than 24 hours. Relative to the other injuries, veterans with TBIs were more likely to be depressed, but more importantly, the life-time risk of depression increased with severity of TBI. Similarly, Levine and Grossman (1978) investigated whether injury severity influenced behavioral disturbances in a sample of 62 TBI patients. Injury severity was classified based on length of coma while behavioral disturbances were measured using the Brief Psychiatric Rating Scale. It was found that severe injuries were associated with greater depressive symptoms.

The association between injury severity and depression has also been shown in younger TBI populations. For example, Barker-Collo (2007) compared the behaviour profile of children who sustained TBI with those who had suffered orthopedic injuries. Their results showed that the TBI group had more problems with depression as measured by the Child Behavior Checklist

(CBCL; Achenbach, 1991). Importantly, these problems increased with injury severity.

Consistent with this, Max et al. (1998) examined the development of psychiatric and behavioral problems following TBI in a sample of 43 children and adolescents. Several psychiatric and behavioral assessments were administered, including the CBCL; while injury severity was classified as severe versus mild. Their results showed that TBI was predictive of novel psychiatric illness including depression. Furthermore, those with severe injuries were more likely to experience depression.

As reviewed, the direct impact of injury severity on post-TBI depression remains controversial. Given this, it should not be surprising that even less is known about the role of injury severity over different stages of recovery. Nevertheless, the natural course of a TBI may provide some insight into this issue. In particular, the pathological changes that result from TBI are greatest during the first months to years after the injury (Lezak et al., 2012). As a result, the association between injury severity and depression may be at its strongest earlier in recovery.

Cognitive Functioning

Similar to injury severity, the direct relationship between cognitive functioning and post-TBI depression is unclear. However, cognitive functioning is known to affect other factors that are associated with depression such as community integration, return to work, disability, and social functioning (Ponsford et al., 2008; Sigurdardottir, Andelic, Roe, & Schanke, 2009; Wood & Rutherford, 2006; Yeates et al., 2004). In addition, the degree to which a TBI survivor is aware of their deficits has been shown to be related to cognitive functioning. For example, Bivona et al. (2008) examined metacognitive self-awareness in a sample of 37 patients with severe TBI. They reported that decreased metacognitive self-awareness was significantly correlated with

aspects of executive functioning (EF) such as mental flexibility and the ability to change behaviour in response to feedback. Similarly, other studies have found that lack of self-awareness is associated with impairments in attentional abilities (McAvinue, O'Keeffe, McMackin, and Robertson, 2005; O'Keeffe, Dockree, Moloney, Carton, and Robertson, 2007). These findings are important to keep in mind because persons with intact self-awareness may be more susceptible to depression. To illustrate this, Crisp (1993, p. 398) asked a TBI survivor to discuss his sense of self-worth subsequent to his injury, and he replied "I'm bloody slower...I work slower...I hate saying that...I'm very evasive to admitting that...it frustrates me knowing what my abilities used to be...We know what we were like before the accident...That's the worst bloody thing." Even though many persons with TBI may have insight into their deficits, few studies have examined the direct relationship between cognitive functioning and post-TBI depression. This is concerning because moderate-to-severe TBI often results in persistent and/or permanent cognitive changes (Dikmen et al., 2003; Draper & Ponsford, 2008; Ruttan, Martin, Liu, Colella, & Green 2008).

Spitz, Schonberger, and Ponsford (2012) conducted one of the few studies that did focus on this relationship. They examined whether cognitive functioning was predictive of depression as measured by the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1994). Ninety-seven participants with mild-to-severe TBI were given a variety of neuropsychological tests that measured memory, attention, processing speed, and EF. The results of the study revealed that poor performance on measures of EF, memory, and processing speed were associated with higher rates of depression. Jorge et al. (2004) examined the clinical, neuropsychological, and structural factors that are associated with major depression 1 year after TBI. Their TBI group consisted of 91 patients while their control group consisted of 27 patients

with traumatic injury (excluding involvement of the CNS). Both groups were administered tests of memory and EF. The frequency of major depression was significantly higher in the TBI group relative to the trauma group. Furthermore, TBI patients with depression scored lower on all of the neuropsychological tests, particularly on measures of EF.

Similarly, Wood and Rutherford (2006) investigated the predictors of psychosocial outcome 10 years after TBI. A sample of 131 participants were administered the HADS as well as several neuropsychological tests. In addition to predicting other outcomes such as community integration and life satisfaction, deficits in working memory were also predictive of depression. Consistent with this, Rapoport, McCullagh, Shammi, and Feinstein (2005) compared the cognitive abilities of TBI patients with depression to those without depression. The presence of depression was diagnosed by a psychiatrist based on DSM-IV criteria. A variety of cognitive measures was given to assess memory, attention, processing speed, and executive functioning. The authors found that patients with depression performed poorly on tasks of working memory, processing speed, and verbal memory relative to patients without depression. Furthermore, patients with depression also had significantly more perseverative responses on the Wisconsin Card Sorting Task, which was used to measure executive functioning. The findings remained consistent when the authors controlled for age and past history of depression.

Hart et al. (2012) also provided some insight regarding the role of cognitive functioning on depression after TBI. They used a longitudinal design to examine the course of depression in a TBI sample over 2 years. In general, they were interested in examining the presence and severity of depression over time as well as exploring which factors predict the stability, deterioration, and improvement of depressive symptoms. Their sample consisted of 1089 participants enrolled in the Traumatic Brain Injury Model System (TBIMS) followed at 1 and 2

years post-injury. Depression was measured using the PHQ-9 and cognitive functioning was measured using the Functional Independence Measure (FIM). As part of their results, they reported that greater cognitive disability was related to worsening depressive symptoms at year 2. Their findings emphasize the important role of cognitive functioning in post-TBI depression, and, in fact, may suggest that cognitive impairment may be a risk factor for the development of new episodes of depression after TBI.

As demonstrated, there is a paucity of studies that examine the direct relationship between cognitive functioning and post-TBI depression. The studies that were reviewed indicate that there may be a correlation, but findings are mixed as to which cognitive skills are most important to consider. There is also a need to learn more about how the impact of cognitive functioning changes over time. To date, it is believed that the majority of cognitive recovery occurs within 2 years of sustaining a TBI, after which it begins to plateau (Ruttan et al., 2008). Given this, it would be anticipated that the impact of cognitive functioning would be greatest during the first 2 years after TBI. Beyond this point, individuals may come to terms with the persistent nature of their impairments and adjust accordingly.

Lastly, it is possible that cognitive functioning may indirectly affect depression by influencing coping. As discussed earlier, individuals with TBI may lack the cognitive resources required to engage in problem-focused coping. Assuming that problem-focused coping is negatively correlated with depression, it is possible that cognitive functioning may moderate this relationship. Support for this theory comes from studies that have found an association between cognition and coping. For example, Krpan, Stuss, and Anderson (2011) examined neuropsychological, physiological, and psychological differences in persons with TBI who adopt problem-focused coping versus emotion-focused coping. The participants were given a series of

questionnaires and neuropsychological tests. It was found that individuals who were more likely to use emotion-focused coping style were more likely to have poor executive functioning performance. In contrast, participants who adopted problem-focused coping were more likely to do well on measures of executive functioning. Similarly, Krpan, Levine, Stuss, and Dawson (2007) examined the influence of executive functioning on coping 1-year after TBI. Their study sample consisted of 21 TBI patients and 15 controls. Severity of injury ranged from mild to moderate as measured by the GCS. A composite score for EF was obtained by averaging the score on various tests based on correlational analysis. These tests included the Brown-Peterson Procedure, Trail Making Test (part A and B), Stroop Test, WCST, and the Revised Strategy Application Test (R-SAT). Among the TBI group, it was found that better executive functioning was related to greater use of problem-focused coping strategies. Conversely, lower executive functioning was related to emotion-focused coping. These studies emphasize the role that cognition, in particular EF, can play in coping style. To date however, no studies have examined whether cognitive impairments moderate the relationship between coping and post-TBI depression. In other words, it is possible that individuals with lower EF who engage in problem-focused coping are more depressed relative to individuals with higher EF who engage in problem-focused coping.

Purpose of the Current Study

The current investigation attempted to answer several important theoretical questions. Firstly, it examined whether injury severity and cognitive functioning predict post-TBI depression above and beyond coping, perceived social support, and alcohol use. Secondly, this investigation examined how the contribution of neurological, cognitive, and psychosocial variables to post-TBI depression changes at different stages of recovery. Thirdly, the

relationships between problem-focused coping, emotion-focused coping, and post-TBI depression were assessed. Fourthly, this study examined the direct role that cognitive functioning plays in post-TBI depression. Finally, this study also investigated whether EF moderates the relationship between problem-focused coping and depression.

Hypotheses

1. It was predicted that injury severity and cognitive functioning (i.e., attention, EF, and memory) would predict post-TBI depression above and beyond coping, social support, and alcohol use within two years post-injury. The reason for this is that neurological and cognitive changes are continuing to unfold during this time; as such, it was anticipated that their influence on the development of depression would be greatest during early recovery.
2. At 5 to 15 years after TBI, it was hypothesized that the predictive ability of injury severity and cognitive functioning would diminish as neurological and cognitive recovery would have neared or reached a plateau.
3. With respect to psychosocial variables, it was expected that both types of coping, perceived social support, and alcohol use would be associated with depression regardless of time since injury.
 - a. For coping style, it was hypothesized that the use of emotion-focused coping would be associated with higher levels of depression while the use of problem-focused coping would be associated with lower levels of depression.
 - b. It was hypothesized that more alcohol use would be predictive of higher levels of depression.

- c. It was hypothesized that lower perceived social support would be predictive of depression.
 - d. It was also anticipated that the cumulative predictive ability of these psychosocial variables and their individual contributions would increase with time.
4. It was hypothesized that EF would moderate the relationship between problem-focused coping and depression. Specifically, it was hypothesized that persons with EF impairments who engaged in problem-focused coping would be more depressed relative to persons with intact EF who engaged in problem-focused coping.

Method

Participants

This study used archival data from participants enrolled in the Southeastern Michigan Traumatic Brain Injury System (SEMTBIS), which is part of the NIDRR-funded Traumatic Brain Injury Model Systems (TBIMS) Project and has been described in detail by Corrigan et al. (2011). Originally, the sample consisted of 278 participants; however, listwise deletion was used to exclude cases with missing data (see appendix A and B for information regarding missing data). This reduced the sample to 187 participants who were then divided into 2 groups. The Time 1 group, also referred to as the Early Recovery group, consisted of 80 participants who were ≤ 2 years removed from their TBI, while the Time 2 group, also referred to as Late Recovery group, consisted of 107 participants who were ≥ 5 years removed from their TBI. Each of these groups was composed of different individuals. Two years or less was selected as a cut-off point for the Early Recovery group because this is approximately the amount of time it takes for cognitive and neurological changes to plateau (Lezak et al., 2012; Ruttan et al., 2008). Five

years or more was selected as a cut-off point for the Late Recovery group in order to maximize the time since injury. As part of the SEMTBIS project, some participants were assessed at multiple time points. For example, participants in the Early Recovery group were tested at year 1 and/or year 2 post-injury. Meanwhile, participants in the Late Recovery group were tested at year 5, 10, and/or 15. For the purpose of this study, data from year 1 was used for the Early Recovery group if the participant was tested multiple times. The reason for this was to keep time since injury to a minimum for this group. For the Late Recovery group, data from the last time they were tested was used if the participant was tested multiple times. The reason for this was to increase the time since injury for this group. By using the results from the second or third testing, there were some concerns regarding practice effects for the Late Recovery Group. However, the influence of this phenomenon was anticipated to be minimal given the extended time between test administration (Salthouse, Schroeder, & Ferrer, 2004).

All participants received acute care at the TBIMS site within 72 hours after injury and had a Glasgow Coma Scale score of 3-12 or 13-15 with intracranial hemorrhage. Participants in the latter group were classified as having moderate injuries because they had positive neuroimaging findings and research has shown that their injury characteristics are similar to those with moderate TBI (Kashluba, Hanks, Casey, & Millis, 2008). Neuropsychological tests of memory, attention, and EF, as well as measures of coping and social support were administered to each participant by trained research assistants. Informed consent was obtained by the participant or a designated proxy if the participant was still in posttraumatic confusion at the time of inpatient rehabilitation.

Measures

Brief Symptom Inventory 18 (BSI-18). The BSI-18 was used as the outcome measure for this study. This instrument is a concise but highly sensitive self-report questionnaire that is used to screen for psychological distress and psychiatric disorders in medical patients as well as the general population (Derogatis, 2001). It is made up of 3 subscales including: (a) depression; (b) anxiety; and (c) somatization (Derogatis, 2001). For this study however, only scores on the depression subscale were used. This subscale consists of 6 items which patients complete by rating their level of distress from 0 – 4 (higher scores indicate more distress) over the last 7 days. The specific items include: “(a) Feeling no interest in things; (b) Feeling lonely; (c) Feeling blue; (d) Feelings of worthlessness; (e) Feeling hopeless about the future; (f) Thoughts of ending your life” (Derogatis, 2001). The items on the BSI-18 were chosen based on the prevalence of the symptom, item analysis characteristics, and loading saturations in factor analysis of the Brief Symptom Inventory (BSI) and Symptom Checklist-90-Revised (SCL-90-R; Derogatis, 2001). Raw scores are calculated by summing the values that the patient endorses on each item. The maximum raw score that can be obtained on any of the subscales is 24. Subscale raw scores are subsequently converted into standardized T scores based on normative data from Derogatis (2001). Elevations above 65 are deemed clinically relevant.

There are two main reasons that support the use of the BSI-18 on a TBI sample. To begin with, standard instruments of depression such as the Beck Depression Inventory, the Hospital Anxiety and Depression Scale, and the Geriatric Depression Scale contain items that confound the measurement of this condition (Babin, 2003). The reason for is that these instruments query symptoms that overlap between depression and TBI such as memory complaints, poor concentration, restlessness, lack of energy, and crying (Babin, 2003). Not surprisingly, medical

patients who endorse these symptoms may not be depressed (Kathol et al., 1990). In comparison, items on the BSI-18 depression subscale do not inquire about neurological and somatic symptoms commonly experienced by persons with TBI. Additionally, the psychometric properties of the BSI-18 and in particular, the depression subscale, have been validated within a TBI sample (Meachen, Hanks, Millis, & Rapport, 2008). The internal consistency estimate, as measured by Cronbach alpha, for the depression subscale was found to be .84 while for the entire scale it was .91. The test-retest reliability for the depression subscale was .63 while for the entire scale it was .66 (Meachen et al., 2008). Although these values are lower, this is to be expected with any measure of affective state. In regards to validity, the BSI-18 is significantly correlated with other common measures of psychosocial functioning such as the Neurobehavioral Functioning Inventory (NFI; Kreutzer et al., 1999), the Positive and Negative Affect Schedule (PANAS; Tellegen, Watson, & Clark, 1988), and the Satisfaction with Life Scale (SWLS; Diener, Emmons, Larson, & Griffen, 1985; Meachen et al., 2008).

Coping Inventory for Stressful Situations (CISS). Coping style was measured using the CISS, which is a multidimensional self-report measure that can be used with a wide range of respondents including healthy adolescents and adults, as well as clinical populations (Endler & Parker, 1999; Hanks, Rapport, Wertheimer, & Koviak, 2012). It is comprised of 3 scales each containing 16 items that measure different coping styles; these include Task-Oriented coping, Emotion-Oriented coping, and Avoidance-Oriented coping (Endler & Parker, 1999). The last scale, Avoidance-Oriented coping, is further subdivided into two subcomponents; Distraction (8 items) and Social Diversion (5 items; Endler & Parker, 1999). In order to complete the CISS, respondents are instructed to rate the items on a 5-point scale such that 1 = “Not at all” and 5 = “Very much.” Administration time for the CISS is typically less than 10 minutes although this

can vary based on individual differences. Raw scores for each of the scales can range from 16-80. Higher scores suggest that an individual has the tendency to engage in that coping style more frequently relative to others who scored lower on that scale. This study used raw scores from the Emotion-Oriented coping scale and the Task-Oriented Coping scale.

The CISS has been validated on various populations including healthy adults, undergraduate students, psychiatric patients, and adolescents. For the healthy adults, the internal consistency, as measured by Cronbach alpha, was .88 for the Task-Oriented scale, .90 for the Emotion-Oriented, and .82 for the Avoidance-Oriented Coping scale (Endler & Parker, 1999). The test-retest reliability has been validated on undergraduate students. The estimates for males were .73 for the Task-Oriented scale, .68 for the Emotion-Oriented scale, and .55 for the Avoidance-Oriented scale. The estimates for women were .72 for the Task-Oriented scale, .71 for the Emotion-Oriented scale, and .60 for the Avoidance-Oriented scale (Endler & Parker, 1999). With respect to validity, the scales of the CISS have been compared to those of the Ways of Coping Questionnaire (WCQ; Folkman & Lazarus, 1985, 1988). This commonly used inventory is made up of a Problem-Focused scale, Social Support scale, and 6 Emotion-Focused scales. The 6 Emotion-Focused scales include the: (a) Wishful Thinking scale; (b) Distancing scale; (c) Emphasizing the Positives scale; (d) Self-Blame scale; (e) Tension-Reduction scale; (f) and Self-Isolation scale. It was found that the Task-Oriented scale on the CISS was significantly correlated with the Problem-Focused scale on the WCQ (males = .42; females = .49). The Emotion-Oriented scale of the CISS was significantly correlated with the Wishful Thinking scale (males = .69; female = .49), Distancing scale (males = .45; females = .36), Emphasizing the Positive scale (males = .48), Self-Blame scale (female = .55), Tension Reduction scale (males = .46), and the Self-Isolation scale (female = 31) of the WCQ. Similarly, the Avoidance-Oriented

scale of the CISS was correlated with the Wishful Thinking scale (males = .40; females = .25), Distancing scale (females = .39), Emphasizing the Positive scale (male = .38; females = .24), and Tension-Reduction (female = .49) of the WCQ.

Social Provisions Scale. The Social Provisions Scale was used to measure perceived social support (Cutrona & Russell, 1987). This instrument was designed to assess 6 aspects of social relationships including: (a) guidance (advice or information); (b) reliable alliance (assurance that others can be counted on in times of crisis); (c) reassurance of worth (recognizing one's competence); (d) attachment (emotional closeness); (e) social integration (a sense of belonging to a group of friends); and (f) opportunity for nurturance (providing assistance to others). Scores can be obtained for each of the subscales in addition to a total score. The total raw score was used for this study.

The internal consistency of the Social Provisions Scale, as measured by Cronbach alpha, has been estimated to be over .70 (Cutrona, Russell, & Rose, 1986), while the test-retest reliability can range from .37 to .66. With respect to validity, scores on the Social Provisions Scale have been shown to be significantly correlated to scores on the UCLA Loneliness Scale (Cutrona, 1982; Russell, Peplau, & Cutrona, 1980). The Social Provisions Scale is also correlated with number of relationships, frequency of contact, and satisfaction with social supports (Cutrona & Russell, 1987).

Neuropsychological Tests

Tests of attention, memory, and EF (Mitrushina, Boone, Razani, D'Elia, 2005; Strauss, Sherman, & Spreen, 2006) were chosen because impairments in these cognitive domains have been consistently demonstrated after TBI (e.g., Hart, Whyte, Kim, & Vaccaro, 2005; Niemann,

Ruff, & Kramer, 1996; Rapoport et al., 2005; Wiegner & Donders, 1999). Moreover, there is evidence to suggest that impairments in these cognitive domains are associated with post-TBI depression (Jorge et al., 2004; Spitz et al., 2012; Wood & Rutherford, 2006). Lastly, the inclusion of an EF test was further warranted because it has been shown to influence coping (Krpan et al., 2011), which in turn, may affect post-TBI depression.

Digit Vigilance Test (DVT). The DVT was developed by Lewis and Rennick (1979) with the purpose of measuring sustained attention and psychomotor speed (Mitrushina et al., 2005). This test has been validated on healthy adults, medical, psychiatric, and TBI patients (Grant et al., 1987; Heaton, Miller, Taylor, & Grant, 2004; Kwok, Lee, Leung, & Poon, 2008; Stein, Kennedy, & Twamley, 2002). The DVT consists of two pages; each one is made up of 59 rows of randomly placed numbers. The numbers on the first page are printed in red ink while the numbers on the second page are printed in blue ink. In order to complete the test, the participant is asked to cross out the number 6, which occurs randomly throughout both pages. The participant completes the first page as quickly as they can before they proceed to the second page. As an alternative, the number 9 can also be used instead of the number 6. The DVT yields 3 outcome scores, which include: the total time it takes to complete the test, the number of omission errors, and the number of commission errors. This study used the total time raw score, which was recorded in seconds.

Wisconsin Card Sorting Test (WCST-64). The WCST is frequently used to measure a range of EFs such as planning, organization, abstract reasoning, concept formation, cognitive set maintenance, shifting ability, and inhibiting impulsive responses in healthy adults and clinical populations (Demakis, 2003; Heaton et al., 2004; Mitrushina et al., 2005; Stratta et al., 1993; Strauss et al., 2006). Participants are presented with a deck of 64 cards and asked to sort each

one to four key cards. They do this by placing the cards under the key cards that are set up in front of the participant in a predetermined order. However, participants are not told how to sort the cards. The WCST yields several outcome scores; this study used the total number of perseverative errors. This raw score reflects the examinee's tendency to make repetitive errors despite feedback.

California Verbal Learning Test – Second Edition (CVLT-II). The CVLT-II is a widely administered list-learning test of memory (Mitrushina et al., 2005; Strauss et al., 2006). Structurally, the CVLT-II consists of several parts, but the current study focused on the acquisition trial. During this phase of the test, a word list is read to the examinee over 5 trials. Each time, the examinee is asked to “repeat back as many of the words as you can remember.” The total raw score for the number of words recalled over the 5 trials was used for this study.

Statistical Analysis

To describe demographic differences between the two groups, participants in the Early Recovery group were compared with participants in the Late Recovery group using chi-squared and two sample *t* tests. Demographic and injury variables of interest included: age, sex, education, employment history, injury severity, and disability. Multiple linear regressions were used to examine a priori hypotheses. The statistical significance, R^2 , adjusted R^2 , and effect size (f^2) were reported for each model. It should be noted that the usefulness of R^2 for this study was limited because this estimate always favours the model with the most parameters. Consequently, more importance was given to the adjusted R^2 because it penalizes for the number of included parameters. For this reason, adjusted R^2 was also used to calculate the f^2 . Additionally, the Bayesian Information Criterion (BIC) was used to select the best fitting model. This fit statistic is

commonly used to compare generalized linear models whereby lower BIC values are thought to reflect the better fitted model (Raftery, 1995). It considered to be one of the most conservative methods of model selection because it takes the number of parameters and sample size into consideration and tends to favour simpler models. The absolute difference between the BIC statistics for the models was used to assess degree of model preference. It is generally accepted that a difference of 0-2 = weak preference; difference of 2-8 = positive preference; difference of 6-10 = strong preference; and difference of >10 = very strong preference (Raftery, 1995).

Coefficient beta weights and squared semipartial correlations were used to determine the direction of the relationship and the unique contribution of each predictor variable to depression. Furthermore, independent sample *t* tests were conducted for each predictor variable to determine whether there were any differences between the early and late recovery group.

Hypothesis 1 analysis. Two regression models were run to examine whether cognitive functioning and injury severity predict post-TBI depression above and beyond psychosocial variables during early recovery. All of the predictor variables were entered into the first/complete model. This included emotion-focused coping, problem-focused coping, perceived social support, alcohol use, attention score, memory score, EF score, and injury severity as measured by the GCS score at admission. Due to the lack of variability in the participants' responses, alcohol use was coded dichotomously, such that: 0 = Abstaining and 1 = Drinking. For the second/reduced model, the cognitive scores and injury severity were excluded from the regression equation. If cognitive functioning and injury severity were predictive of depression at the Early Recovery, it was expected that both models would be statistically significant; however, the variance accounted for (adjusted R^2) and the effect size would be greater for the complete model. Furthermore, the BIC value for the complete model would be lower than the BIC value

for the reduced model thereby suggesting a very strong preference for the complete model. With respect to the individual predictors, it was anticipated that injury severity and cognitive functioning would be significantly associated with depression. More specifically, greater injury severity and cognitive impairment would be associated with higher levels of depression.

Hypothesis 2 analysis. In order to test whether the impact of cognitive functioning and injury severity reduced with time, the same analysis was run for the Late Recovery group. Given the a priori hypothesis, it was expected that both models would be statistically significant; however, there would be a strong preference for the reduced model (i.e., greater adjusted R^2 , effect size, and lower BIC). The reason being, that injury severity and cognitive functioning would not be significantly associated with depression at later stages of recovery.

Hypothesis 3 analysis. To determine whether psychosocial variables were predictive of depression regardless of time since TBI, the significance level, beta value, standardized beta value, and squared semipartial correlation for each predictor variable were assessed. For both groups, it was expected that alcohol use, and emotion-focused coping would be positively associated with depression. In contrast, perceived social support and problem-focused coping were expected to be negatively associated with depression.

To examine whether the influence of psychosocial variables was greater at time 2, the reduced model from the Late Recovery group was compared with the reduced model from the Early Recovery group. If psychosocial variables contribute more to depression later in recovery, it was expected that the Late Recovery group reduced model would have a greater adjusted R^2 , effect size, and a lower BIC value. The squared semipartial correlations and standardized beta

weights for each psychosocial variable were also anticipated to be greater for the Late Recovery group.

Hypothesis 4 analysis. To test whether EF moderates the association between problem-focused coping and depression, EF and problem-focused coping were centered so that they had a mean of 0. An interaction term was then created and entered into the regression equation with all of the other variables. The presence of a moderating relationship would be supported if the interaction term was statistically significant.

Results

A power analysis was conducted to ensure that the sample size was sufficient enough to detect any significant results. As discussed, published research has consistently shown that coping, social support, and alcohol use are predictive of post-TBI depression (Rogers & Read, 2007; Anson & Ponsford, 2006; Ownsworth & Oei, 1998; West, 2011). In particular, the unique variance of coping and social support on depression in some of these studies has exceeded 35% (Curran et al., 2000; Douglas & Spellacy, 1999). Therefore, for the current investigation, it was conservatively estimated that the minimum R^2 that the predictor variables would account for would be .20. The power analysis produced a sample size of 68 when an R^2 value of .20 was used in combination with an alpha level of .05, 8 predictor variables, and a power of .80. This suggests that the current study design had sufficient power to detect a minimum R^2 of .20 since the smallest sample size was 80 for the Early Recovery group.

The data were then examined to ensure that the assumptions of linear regression were met. For the Early Recovery group, the skewness = .22, $p < .001$, and kurtosis = 1.93, $p < .001$. For the Late Recovery group, the skewness = .39, $p < .001$, and kurtosis = 2.16, $p < .001$.

Therefore, the distribution of the dependent variable (i.e., depression) was not normal for both groups. Linear regression however, is robust to violations of normality (Cohen et al., 2003). In addition, robust regressions were conducted which produce similar R^2 , beta, and standardized beta values, but have standard errors that do not assume normality (Acock, 2012). Robust regression also produces slightly smaller t -values (Acock, 2012). Scatterplots showed that the residuals for both groups were normally distributed. Furthermore, tolerance and VIF values revealed no issues with multicollinearity. Three outlying cases were identified, all of which were in the Late Recovery group. When these cases were removed, the results remained consistent. This was one of the main reasons they were not excluded from the analysis. Furthermore, there was no evidence of miscoding or mistakes with data collection and there was also no way of verifying the validity of the participant's responses. This provided further justification to include these cases in the analysis, especially given that uncontaminated outlying responses are commonly encountered in research (Cohen et al., 2003).

The mean depression score for the Early Recovery group was 55 ($SD = 11$, range = 40-81), while the mean depression score for the Late Recovery group was 54 ($SD = 10$, range = 40-81). Information regarding demographic and injury characteristics for both groups can be found in Table 1, Table 2, and Table 3. Information regarding psychosocial variables as well as neurological and cognitive variables can be found in Table 4 and Table 5 respectively.

Table 1

Demographic Characteristics for Both Groups

	Early Recovery Group (N=80)		Late Recovery Group (N=107)	
	M (SD)	Range	M (SD)	Range
Age at Time of Injury	38.8 (13.8)	16 – 66	35.7 (11.6)	16 – 75
Age at Time of Testing	40.1 (13.8)	17 – 68	46.1 (11.1)	21 – 80
Total Years of Education at Time of Testing	11.7 (2.1)	6 – 18	12.0 (2.1)	7 – 18
Time Since Injury (Years)	1.3 (0.5)	1 – 2	10.1 (4.0)	5 – 15
Disability Level	2.4 (1.8)	0 – 7.5	1.8 (1.7)	0 – 7.5

Table 2

Cause of TBI for the Early and Late Recovery Groups

Early Recovery Group (N=80)	Frequency	Percentage (%)
Motor vehicle accident	16	20.00
Motorcycle accident	11	13.75
Gunshot wound	10	12.50
Assault related injury	22	27.50
Pedestrian vs. automobile	6	7.50
Fall related injury	15	18.75
Late Recovery Group (N=107)		
Motor vehicle accident	35	32.71
Motorcycle accident	4	3.74
Gunshot wound	8	7.48
Assault related injury	44	41.12
Pedestrian vs. automobile	9	8.41
Fall related injury	7	6.54

Table 3

Sex and Ethnicity of Participants in Both Recovery Groups

Early Recovery Group (N=80)		Late Recovery Group (N=107)	
	Percentage (%)		Percentage (%)
Sex		Sex	
Male	80	Male	81
Female	20	Female	19
Ethnicity		Ethnicity	
African American	73	African American	78
Caucasian	26	Caucasian	19
Hispanic/Native American/Pacific Islander	1	Hispanic/Native American/Pacific Islander	3

Table 4

Psychosocial Information for Participants in Early and Late Recovery Groups

Early Recovery Group (N=80)	M	SD	Range
Emotion-focused coping	45.3	13.9	20 – 80
Problem-focused coping	57.1	12.6	24 – 80
Perceived social support	46.2	6.9	26 – 60
Alcohol use at time of testing	Percentage (%)		
Abstaining	84		
Drinking	16		
Late Recovery Group (N=107)			
Emotion-focused coping	42.5	13.1	16 – 80
Problem-focused coping	56.9	12.4	18 – 80
Perceived social support	46.2	6.8	26 – 60
Alcohol use at time of testing	Percentage (%)		
Abstaining	78		
Drinking	22		

Table 5

Cognitive and Neurological Information for Participants in Early and Late Recovery Groups

Early Recovery Group (N=80)	M	SD	Range	T-Score (M)
Injury severity (GCS at admission)	9.4	4.3	3 – 15	
Attention (DVT score)	525.9	182.2	280 – 1112	34.0
Memory (CVLT2 total acquisition trial score)	35.9	11.7	4 – 69	37.1
EF (WCST perseverative errors score)	14.8	9.8	3 – 46	40.5
Late Recovery Group (N=107)				
Injury severity (GCS at admission)	8.1	4.5	3 – 15	
Attention (DVT score)	557.4	277.3	257 – 1836	30.4
Memory (CVLT2 total acquisition trial score)	34.1	11.2	4 – 60	37.1
EF (WCST perseverative errors score)	13.6	9.6	2 – 46	42.0

Note. The mean, standard deviation, and range for the cognitive tests are based on raw scores. The T-scores are provided for comparative purposes and were calculated using Heaton et al. (2004) demographically adjusted norms.

Chi-squared and two-sample t tests were used to examine differences in demographic and injury variables between individuals in the Early Recovery group relative to individuals in the Late Recovery group. There were no group differences for employment status $\chi^2(1, 187) = .60, p > .05$; education level at time of testing $t(185) = -.67, p > .05$; sex $t(185) = -.22, p > .05$; injury severity $t(185) = 1.9; p > .05$; age at injury $t(185) = 1.6 p > .05$, and age at time of testing $t(185) = -3.25, p > .05$. However, individuals in the Early Recovery reported higher levels of disability, as measured by the Disability Rating Scale (DRS; Rappaport, Hall, Hopkins, Belleza, & Cope, 1982), at follow-up relative to individuals in the Late Recovery group $t(185) = 2.1, p < .05$. This was not surprising given that they were 1.3 years removed from their injury. Regarding the cause of TBI, individuals in both groups were further classified as either having a violence-related injury or a nonviolence- related injury (i.e., violence-related injury = TBI caused by gunshot wound or assault). The reason for this was that violence-related TBIs have consistently been shown to be associated with important socioeconomic factors such as living in low income areas, unemployment rate, and minority status (Boshnak, Hanks, Kreutzer, & Rosenthal, 2003; Dunn, Henry, & Beard, 2003; Wagner, Sasser, Hammond, Wierciszewski, and Alexander, 2000). For this sample, there was no difference in the rates of violent vs nonviolent cause of injury across the Early and Late recovery groups $\chi^2(1, 187) = 1.37, p > .05$. Similarly, there were no between group differences for endorsement of depression $t(185) = .68, p > .05$; emotion-focused coping $t(185) = 1.4, p > .05$; problem-focused coping $t(185) = -.10, p > .05$; social support $t(185) = -.002, p > .05$; and alcohol use at time of testing $\chi^2(1, 187) = .81, p > .05$. There were also no differences between the two groups on tests of attention $t(185) = -.88, p > .05$; memory $t(185) = -.003, p > .05$; and EF $t(185) = -1.12, p > .05$.

Hypothesis 1

Two regression models were run to determine whether injury severity and cognitive functioning predict depression above and beyond psychosocial variables for the Early Recovery group. All of the predictor variables were entered into a full model while the depression score was entered as the dependent variable. The full model was statistically significant $F(8, 71) = 11.29, p < .001$ and yielded an $R^2 = .44$ (adjusted $R^2 = .37; f^2 = .59$; see Table 6). The goodness-of-fit as measured by the BIC was 251. Importantly, neither injury severity nor any of the cognitive domains were predictive of depression (see Table 7).

For the reduced model, injury severity and cognitive scores were excluded from the regression equation. This model was also statistically significant $F(4, 75) = 22.00, p < .001$ and yielded an $R^2 = .43$ (adjusted $R^2 = .40; f^2 = .67$; see Table 6). The goodness-of-fit as measured by the BIC was 235. The difference between the BIC values for the two models was 16, thereby suggesting a very strong preference for the reduced model.

Hypothesis 2

The same analysis was conducted for the Late Recovery group to examine whether the predictive ability of injury severity and cognitive functioning reduces as time since injury increases. The full model was statistically significant $F(8, 98) = 8.14, p < .001$ and yielded an $R^2 = .40$ (adjusted $R^2 = .36; f^2 = .56$; see Table 6). The BIC value for the full model was 285. In contrast to the Early Recovery group, better attention performance was predictive of depression. Of equal interest was that problem-focused coping was not associated with depression for the Late Recovery group (See Table 8).

The Late Recovery group reduced model, which excluded injury severity and cognitive scores, was also statistically significant $F(4, 102) = 11.76, p < .001$ and yielded an $R^2 = .36$ (adjusted $R^2 = .33; f^2 = .49$; see Table 6). The BIC value for the reduced model was 274. Interestingly, while the difference between the two BIC values was 11, thereby suggesting a very strong preference for the reduced model, the adjusted R^2 and the f^2 were larger for the full model. The reason for these mixed results was that the full model included attention, which was found to be an important predictor of depression.

As a result, an additional linear regression was conducted to determine if including this variable would improve the fit of the reduced model. Consequently, the *new* model included a combination of the following predictors: emotion-focused coping, perceived social support, alcohol use, and attention performance. Problem-focused coping was excluded because it was not associated with depression. Overall, this new model was statistically significant $F(4, 102) = 13.58, p < .001$ and yielded an $R^2 = .39$ (adjusted $R^2 = .37; f^2 = .59$; BIC = 268; see Tables 6 and 9). The lower BIC value as well as the increase in the adjusted R^2 and effect size implied that including attention performance and excluding problem-focused coping produces the best fitting model for the Late Recovery group.

Hypothesis 3

Each of the predictor variables was examined individually to determine whether psychosocial variables were predictive of depression regardless of time since TBI. For the Early Recovery group, the beta weights and the squared semipartial correlations showed that lower perceived social support and the reduced tendency to engage in problem-focused coping were moderately predictive of higher levels of depression. In contrast, the tendency to engage in

emotion-focused coping was moderately predictive of depression (see Table 7). For the Late Recovery group, lower perceived social support and the tendency to engage in emotion-focused coping were moderately predictive of depression. Interestingly however, problem-focused coping was not associated with depression (see Table 8). This lack of a relationship partially contributed to the higher BIC for the Late Recovery group reduced model relative to the Early Recovery group reduced model. The former model also had a significantly lower adjusted R^2 and effect size. Generally, this suggested that the cumulative impact of psychosocial variables was more strongly related to depression during the Early Recovery. On the other hand, by focusing on the squared semipartial correlations and beta weights for emotion-focused coping and perceived social support, it was evident that individually the relation of these variables with depression became slightly stronger during Late Recovery.

Hypothesis 4

The interaction term for EF and problem-focused coping was not statistically significant when it was entered into the regression equation. As such, it appears that the results do not support the hypothesis that EF moderates the relationship between problem-focused coping and depression.

Table 6

Early Recovery and Late Recovery Group Regression Models

Regression model	R²	Adj. R²	f²	BIC	Sig.
Early recovery full model	.44	.37	.59	251	.001
Early recovery reduced model	.43	.40	.67	235	.001
Late recovery full model	.41	.36	.56	285	.001
Late recovery reduced model	.36	.33	.49	274	.001
Late recovery new model	.39	.37	.59	268	.001

Table 7

Full and Reduced Regression Models for the Early Recovery Group (N=80)

Model	Variable	B	SE	β	Squared semipartial correlations	
						Sig.
1 (Full)	Injury severity	-.030	.251	-.012	.001	.904
	Attention	.005	.007	.095	.009	.427
	EF	-.045	.085	-.040	.001	.604
	Memory	.061	.095	.066	.003	.520
	Emotion-focused coping	.354	.090	.451	.161	.001
	Problem-focused coping	-.193	.093	-.222	.046	.041
	Perceived social support	-.368	.146	-.233	.045	.014
	Alcohol use	3.36	2.66	.114	.012	.211
2 (Reduced)	Emotion-focused coping	.340	.086	.434	.163	.001
	Problem-focused coping	-.194	.091	-.223	.047	.036
	Perceived social support	-.357	.141	-.227	.043	.013
	Alcohol use	3.54	2.63	.120	.014	.182

Note. Injury severity = GCS score at admission; Attention = DVT raw score, EF = WCST-64 perseverative errors raw score; Memory = CVLT-2 total acquisition trial raw score; Emotion-Focused Coping = CISS Emotion Scale raw score; Problem-Focused Coping = CISS Task Scale raw score; Perceived Social Support = Social Provisions Scale raw score.

Table 8

Full and Reduced Regression Models for the Late Recovery Group (N=107)

Model	Variable	B	SE	β	Squared semipartial correlations	Sig.
1 (Full)	Injury severity	.083	.187	.036	.001	.659
	Attention	-.008	.004	-.241	.040	.036
	EF	.083	.084	.078	.004	.330
	Memory	-.065	.083	-.072	.004	.430
	Emotion-focused coping	.353	.060	.455	.190	.001
	Problem-focused coping	-.029	.048	-.036	.001	.543
	Perceived social support	-.392	.123	-.267	.063	.002
	Alcohol use	3.33	1.96	.135	.017	.092
2 (Reduced)	Emotion-focused coping	.348	.065	.449	.190	.001
	Problem-focused coping	-.041	.052	-.051	.002	.423
	Perceived social support	-.381	.117	-.259	.062	.002
	Alcohol use	3.32	1.94	.135	.018	.089

Note. Injury severity = GCS score at admission; Attention = DVT raw score; EF = WCST-64 perseverative errors raw score; Memory = CVLT-2 total acquisition trial raw score; Emotion-Focused Coping = CISS Emotion Scale raw score; Problem-Focused Coping = CISS Task Scale raw score; and Perceived Social Support = Social Provisions Scale raw score.

Table 9

New Regression Model for the Late Recovery Group (N=107)

Model	Variable	B	SE	β	Squared semipartial correlations	Sig.
1 (New)	Emotion-focused coping	.354	.058	.457	.200	.001
	Perceived social support	-.416	.121	-.282	.075	.001
	Alcohol use	2.91	1.90	.119	.014	.127
	Attention	-.007	.003	-.187	.034	.035

Note. This new model includes attention as a predictor variable while problem-focused coping was excluded from the regression equation.

^aAttention = DVT raw score; Emotion-Focused Coping = CISS Emotion Scale raw score; and Perceived Social Support = Social Provisions Scale raw score.

Discussion

One of the main purposes of this study was to examine the role of cognitive functioning and injury severity on post-TBI depression above and beyond well established psychosocial variables. Contrary to what was expected, cognitive functioning and injury severity were not predictive of post-TBI depression during early recovery. On the other hand, better attention performance was associated with higher levels of depression during late recovery. Unlike the current findings however, these studies have typically reported that greater attention impairments are predictive of depression. In addition, studies that have found a relationship between attention and depression have reported this pattern for samples that were less than 2 years removed from their TBI (Rapoport et al., 2005; Spitz et al., 2012).

One reason as to why there was not a relationship between attention and depression for the Early Recovery group may be that most cognitive improvement occurs earlier in the recovery process (Ruttan et al., 2008; Spitz et al., 2012). As such, persons with TBI may not be as emotionally distraught about their deficits because they may be under the assumption that this rapid improvement will continue to pre-injury levels. Theoretically, this idea makes sense; however, more studies focusing on the perspective of individuals with TBI and their expectations for recovery need to be conducted before it can be given more credence. Another contributing factor that may explain why there was no relationship between attention and depression during early recovery could be that persons with TBI become more independent with respect to performing activities of daily living between 2 to 5 years post injury (Olver, Ponsford, & Curran, 1996). In other words, individuals in the Early Recovery group may not have had the opportunity to encounter challenging situations that require intact attentional abilities. With time however, they may become more aware of their attentional deficits (Powell, Machamer, Temkin, & Dikmen, 2001) as they encounter difficulties attempting to return to their pre-injury activities. In turn,

increases in their levels of awareness may leave them susceptible to experiencing emotional distress (Fleming, Strong, & Ashton, 1998). This theory would explain why relatively better attention was predictive of depression for the Late Recovery group.

The association between attention and awareness has been documented in the literature. For example, McAvinue et al. (2005) examined error awareness and sustained attention in 18 TBI-participants at 37.7 months post injury. They found that degree of error awareness was strongly correlated with sustained attention ability, even when injury severity was included as a covariate. Similarly, O'Keeffe et al. (2007) investigated awareness of deficits in 31 TBI-participants at 36.2 months post injury. They reported that performance on a test of sustained attention predicted the ability of participants to describe their deficits as they happen as well as to describe how their deficits may lead to difficulties on future cognitive tests. Given the potential relationship between attention and awareness, future studies should examine whether relatively better attention can lead to improved awareness and increased susceptibility to depression while also examining whether poor attention may act as a protective barrier against depression by reducing awareness. In other words, it is plausible that attention may act as a moderator variable between awareness and depression.

All of the psychosocial variables with the exception of alcohol use were predictive of depression for the Early Recovery group. Cumulatively, emotion-focused coping, problem-focused coping, and perceived social support accounted for a significant proportion of the variance in post-TBI depression. As hypothesized, it was found that persons who engaged in emotion-focused coping and reported less social support were more likely to be depressed. Conversely, persons who engaged in problem-focused coping were less likely to be depressed.

Emotion-focused coping and perceived social support were also predictive of depression for the Late Recovery group.

The relationship between these psychosocial variables and depression regardless of time since injury has important implications. Firstly, coping style is amenable to change and many interventions are designed to help people improve their adaptive coping skills. For persons with TBI, many of these interventions are based in the principles of Cognitive Behavioural Therapy (CBT; Anson & Ponsford, 2006; Bradbury et al., 2008; Gurr & Coetzer, 2005; Ownsworth, 2005; Tiersky et al., 2005). For example, Anson and Ponsford (2006) conducted a coping skills group using CBT with 31 individuals with TBI. The intervention was implemented twice a week over a five week period. Overall, the depression levels remained consistent; however, participants reported using more adaptive coping skills to better manage emotional issues. Bradbury et al. (2008) also used CBT in-person and over the phone to help individuals with TBI better cope with emotional distress. Both forms of treatment delivery significantly reduced emotional distress. In addition to CBT, approaches based on mindfulness meditation (Bedard et al., 2003; McMillan, 2002) and comprehensive-rehabilitation programs have been used to improve emotional functioning after TBI (Cattelani, Zettin, & Zoccolotti, 2010; Powell et al., 2002).

Similar to coping, adaptive social skills can also be taught to individuals with TBI. The general purpose of these social skills training (SST) programs are to teach individuals appropriate verbal and nonverbal behaviours so they can gain acceptance from peers and family members, establish friendships, and meet the demands of work and school (Ylvisaker, Turkstra, & Coelho, 2005). Research has shown that SST is generally effective in improving social communication and quality of life (Dahlberg et al., 2006). Furthermore, promoting community

integration such as return to work for persons with TBI may also improve outcome (O'Neill et al., 1998). While many of these interventions may be promising, it should be emphasized that very few psychotherapeutic and rehabilitation studies have been conducted that specifically focus on depression after TBI (Fann, Hart, & Schomer, 2009). Rather, they tend to focus on general emotional functioning which could include anxiety and other comorbid issues. Similarly, their study samples were heterogeneous with respect to variables such as injury severity and time since injury (Fann et al., 2009). Lastly, many of these interventions did not lead to reductions in levels of depression, and of those that were more effective, a limited number of them provided specialized treatment manuals (Fann et al., 2009).

While emotion-focused coping and perceived social support were predictive of depression for the Late Recovery group, the cumulative impact of the psychosocial variables was weaker. An interesting reason for this finding was that problem-focused coping was not predictive of depression during late recovery. This was not completely unexpected given that the effectiveness of problem-focused coping has been shown to reduce with time (Hinkeldey & Corrigan, 1990; Folkman & Moskowitz, 2004; Kendall et al. 2001). In other words, problem-focused coping is most useful for situations that can be controlled or managed. However, for persons with severe TBI, many of their permanent disabilities limit them from functioning as they once did. Consequently, the continued use of problem-focused coping to deal with situations that they do not have the resources to change can become counterproductive (Hinkeldey & Corrigan, 1990; Terry & Hynes, 1998). This may become particularly evident later in recovery as the person gains more independence and begins to reintegrate into the community.

Conversely, the responsibilities associated with early recovery may be less demanding and this may explain why the use of problem-focused coping was effective for the Early Recovery group. In support of this theory, studies that have reported similar findings have done so for samples that were less than 5 years removed from their TBI (Anson & Ponsford, 2006; Curran, Ponsford, & Crowe, 2000). As such, the results of the current study may suggest that the usefulness of problem-focused coping can be classified into 3 stages. The first occurs earlier in recovery when the person is making major cognitive and physical improvements. During this stage, the responsibilities that they are confronted with are less demanding, and as a result, problem-focused coping is an effective strategy to protect against depression. The second stage occurs when the person begins to reintegrate into the community and begins to gain more independence. During this time, the demands of their responsibilities increase and the use of problem-focused coping can become counterproductive if they do not have the resources to successfully manage the obstacles with which they are confronted (Hinkeldey & Corrigan, 1990; Terry & Hynes, 1998). The persistent use of problem-focused coping would be expected to be predictive of depression during this stage. By the third stage, the person may be more willing to accept their disabilities and become more familiar with situations that they cannot alter. Consequently, it would be anticipated that the impact of problem-focused coping on depression during this stage would diminish. In fact, the use of this strategy may only be beneficial as it pertains to dealing with easily manageable tasks and/or compensating for limitations (Willer, Allen, Durnan, & Ferry, 1990; Willer, Allen, Liss, & Zicht, 1991). For example, rather than going grocery shopping, an individual with TBI may use problem-solving to allocate that responsibility to a primary caregiver.

To the author's knowledge, this is the first study to demonstrate a lack of association between problem-focused coping and one aspect of emotional well-being in a chronically injured TBI sample. Interestingly, however, similar results have been reported in other patient groups. For instance, Bombardier, D'Amico, and Jordan (1990) examined the relationship between coping responses and illness adjustment in a sample of patients suffering from chronic medical and psychiatric complications. The participant's average duration of illness was 10 years. While it was found that emotion-focused coping was related to poor psychosocial adjustment and depression, there was no relationship between these outcome variables and problem-focused coping. Keefe et al. (1987) reported similar findings with respect to coping and psychological distress in a sample of patients with longstanding arthritis pain. Overall, the results of the current investigation, along with those of the aforementioned studies, suggest that using problem-focused coping strategies may be less important than avoiding the use of emotion-focused coping strategies when dealing with chronic conditions such as moderate to severe TBI.

If there is indeed a shift in the usefulness of problem-focused coping over time, these findings would have important treatment implications. For example, some aspects of CBT, which is commonly used to help persons with TBI, emphasize skills that are associated with problem-focused coping (Beck, 1995). However, if it is found that this coping strategy is not adaptive for all stages of recovery, alternative techniques need to be explored and implemented.

While the cumulative impact of psychosocial variables was greatest for the Early Recovery group, individually, the impact of emotion-focused coping and perceived social support on depression slightly increased for the Late Recovery group. With respect to emotion-focused coping, this finding may be related to locus of control. The persistent use of emotion-focused coping may cause persons with TBI to externalize their problems and to abandon any

hope of changing stressful situations. This learned helplessness may leave them more susceptible to depression during later stages of recovery. Partial support for this theory comes from studies that have demonstrated the strong relationship between external locus of control and depression in the general population (e.g., Benassi, Sweeney, & Dufour, 1988; Burger, 1984; Wiersma et al., 2011). In addition, the association between external locus of control and learned helplessness has also been widely accepted (e.g., Cohen, Rothbart, & Phillips, 1976; Ross & Mirowsky, 2013). The reason why social support becomes more predictive of depression during later stages of recovery may be related to the quality of the interactions between the individual with TBI and their friends and family. For instance, Tomberg et al. (2007) found that persons with TBI may become less satisfied with their support network as time since injury increases. A possible explanation for this trend may be that social interactions become more transient and superficial with time given that individuals with TBI may never regain the cognitive resources required for in-depth communication. While these are preliminary hypotheses and more research into these areas is required, the findings imply that interventions focusing on improving coping skills and social functioning are important and should be made available several years after TBI and immediately thereafter.

Limitations

This study had some notable limitations. Firstly, the sample size was small, particularly for the Early Recovery group. The sample size was limited because data with missing values was excluded from the analyses using listwise deletion. While this procedure can limit power due to a reduction in sample size (McKnight, McKnight, Sidani, & Figueredo, 2007), it is often considered the method of choice when dealing with missing data (Lynch, 2003). There are several reasons for this; to begin with, imputation methods can result in biased standard errors

and parameter estimates because they underestimate the variability of the missing values (Lynch, 2003). They also require many uncertain decisions such as choosing which imputation procedure to use (i.e., mean imputation, hotdecking, regression-based imputation, or multiple imputation). In the case of multiple imputation, several data sets need to be created using different imputed values. As a result, questions arise as to how many data sets are enough? Maximum likelihood estimation could have been used to deal with the missing data; however, this method would have required the linear regressions to be run using structural equation modelling (SEM). This approach is limited because it does not yield an overall *p* value for the regression equations and it also does not provide information related to regression diagnostics (Kolenikov, 2013).

The results of this study would have been strengthened if depression was measured using multiple instruments rather than solely relying on the BSI-18. Similarly, cognitive performance in the domains of attention, EF, and memory were also defined using scores from single tests (i.e., DVT, WCST-64, CVLT-2). It should be noted however, that in clinical practice, the pattern of performance over a battery of tests is used to assess cognitive functioning. Furthermore, using injury severity as a measure of neurological damage was a limitation. Methods such as neuroimaging may have been more accurate at measuring extent of brain damage relative to GCS score. Another weakness of this study was that most of the non-cognitive measures used were self-report inventories, which may not reflect the underlying construct in a TBI population as they do in a neurologically intact population, given the high potential for impairment in awareness of deficits. Similarly, for the cognitive measures response bias is always a concern when using neuropsychological tests. The validity of the cognitive test scores could have been verified if effort measures were included.

One of the main purposes of this investigation was to compare two different time points; early recovery and late recovery using a cross-sectional design. However, both of these groups consisted of different individuals. Although some in-between group differences were accounted for by conducting chi-squared and *t* tests, there are likely many other important variables that were not examined. In order to control for important individual differences, it would have been ideal to use a longitudinal design.

Conclusions

This investigation revealed several interesting and important results. It was shown that injury severity as well as memory and EF ability did not predict depression above and beyond psychosocial factors regardless of time since injury. On the other hand, better attention functioning may be related to more depression later in recovery. The mechanism behind this relationship may stem from improved awareness of deficits. Screening for cognitive functioning after TBI may be useful in better understanding persons who may be susceptible to depression.

The tendency to engage in emotion-focused coping and lower levels of perceived social support were moderately correlated with depression. It should also be noted that the influence of these variables on depression may increase with time. To this end, future studies should continue to improve and create new treatment protocols that focus on altering coping and social skills. These programs should also be made available to persons with TBI from the time that they are injured until at least several years post injury. Finally, the current study revealed that the relationship between problem-focused coping and depression may reduce with time. Examining when this shift occurs during recovery could improve the effectiveness of interventions for persons with TBI.

References

- Achenbach, T. M. (1991). *Child behavior checklist/4-18*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Acock, A. C. (2012). *A gentle introduction to stata* (revised 3rd ed.). College Station, Texas: Stata Press Publication.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington DC: American Psychiatric Association.
- Anson, K. & Ponsford, J. (2006). Coping & emotional adjustment following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 21(3), 248-259.
- Anson, K., & Ponsford, J. (2006). Evaluation of a coping skills group following traumatic brain injury. *Brain Injury*, 20(2), 167-178.
- Babin, P. R. (2003). Diagnosing depression in persons with brain injuries: A look at theories, the DSM-IV, and depression measures. *Brain Injury*, 17(10), 889-890.
- Baldwin, D., & Rudge, S. (1995). The role of serotonin in depression and anxiety. *International Clinical Psychopharmacology*, 9(4), 41-45.
- Barker-Collo, S. L. (2007). Behavioural profiles and injury severity following childhood traumatic brain injury. *Brain Impairment*, 8(01), 22-30.
- Beck, J. S. (1995). Cognitive therapy: Basics and beyond. New York: The Guilford Press.
- Bedard, M., Felteau, M., Mazmanian, D., Fedyk, K., Klein, R., Richardson, J., Parkinson, W. & Minthorn-Biggs, M. B. (2003). Pilot evaluation of a mindfulness-based intervention to

improve quality of life among individuals who sustained traumatic brain injuries. *Disability & Rehabilitation*, 25(13), 722-731.

- Bedford, A., Foulds, G. A., & Sheffield, B. F. (1976). A new personal disturbance scale (DSSI/sAD). *British Journal of Social and Clinical Psychology*, 15(4), 387-394.
- Benassi, V. A., Sweeney, P. D., & Dufour, C. L. (1988). Is there a relation between locus of control orientation and depression? *Journal of Abnormal Psychology*, 97(3), 357-367.
- Bivona, U., Ciurli, P., Barba, C., Onder, G., Azicnuda, E., Silvestro, D., ... & Formisano, R. (2008). Executive function and metacognitive self-awareness after severe traumatic brain injury. *Journal of the International Neuropsychological Society*, 14(05), 862-868.
- Bombardier, C. H., D'Amico, C., & Jordan, J. S. (1990). The relationship of appraisal and coping to chronic illness adjustment. *Behaviour Research and Therapy*, 28(4), 297-304.
- Bombardier, C. H., Fann, J. R., Temkin, N. R., Esselman, P. C., Barber, J., & Dikmen, S. S. (2010). Rates of major depressive disorder and clinical outcomes following traumatic brain injury. *Journal of the American Medical Association*, 303(19), 1938-1945.
- Bradbury, C. L., Christensen, B. K., Lau, M. A., Ruttan, L. A., Arundine, A. L., & Green, R. E. (2008). The efficacy of cognitive behavior therapy in the treatment of emotional distress after acquired brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(12), S61-S68.
- Burger, J. M. (1984). Desire for control, locus of control, and proneness to depression. *Journal of Personality*, 52(1), 71-89.
- Bushnik, T., Hanks, R. A., Kreutzer, J., & Rosenthal, M. (2003). Etiology of traumatic brain injury: Characterization of differential outcomes up to 1 year postinjury. *Archives of Physical Medicine and Rehabilitation*, 84(2), 255-262.

- Cattelani, R., Zettin, M., & Zoccolotti, P. (2010). Rehabilitation treatments for adults with behavioral and psychosocial disorders following acquired brain injury: A systematic review. *Neuropsychology Review*, 20(1), 52-85.
- Cohen, S., Rothbart, M., & Phillips, S. (1976). Locus of control and the generality of learned helplessness in humans. *Journal of Personality and Social Psychology*, 34(6), 1049-1056.
- Coronado, V. G., McGuire, L. C., Faul, M., Sugerman, D. E., & Pearson, W. S. (2013). Traumatic brain injury epidemiology and public health issues. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain Injury Medicine: Principles and practice* (2nd ed., pp. 84-100). New York: Demos Medical Publishing.
- Corrigan, J. D. (1995). Substance abuse as a mediating factor in outcome from traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 76(4), 302-309.
- Corrigan, J.D, Cuthbert, J.P., Whiteneck, G.G., Dijkers, M.P., Coronado, V., Heinemann, A.W., Harrison-Felix, C., Graham, J.E. (2011). Representativeness of the Traumatic Brain Injury Model Systems Database. *Journal of Head Trauma Rehabilitation*, 26 (5), 1-9.
- Corrigan, J. D., Smith-Knapp, K., & Granger, C. V. (1998). Outcomes in the first 5 years after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 79(3), 298-305.
- Crisp, R. (1993). Personal responses to traumatic brain injury: A qualitative study. *Disability, Handicap & Society*, 8(4), 393-404.
- Curran, C. A., Ponsford, J. L., & Crowe, S. (2000). Coping strategies and emotional outcome following traumatic brain injury: A comparison with orthopedic patients. *Journal of Head Trauma Rehabilitation*, 15(6), 1256-1274.

- Cutrona, C. E. (1982). Transition to college: Loneliness and the process of social adjustment. In L. A. Peplau & D., Perlman (Eds.). *Loneliness: A sourcebook of current theory, research, and therapy*, (pp. 291-309). New York: Wiley.
- Cutrona, C. E., & Russell, D. W. (1987). The provisions of social relationships and adaptation to stress. In W. H., Jones & D., Perlman (Eds.). *Advances in personal relationships*, (pp. 37-67). Greenwich, CT: JAI.
- Cutrona, C., Russell, D., & Rose, J. (1986). Social support and adaptation to stress by the elderly. *Psychology and Aging, 1*(1), 47-54.
- Dahlberg, C., Hawley, L., Morey, C., Newman, J., Cusick, C., Harrison-Felix, C., & Coll, J. (2006). Social communication skills training after traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 21*(5), 425.
- Demakis, G. J. (2003). A meta-analytic review of the sensitivity of the Wisconsin Card Sorting Test to frontal and lateralized frontal brain damage. *Neuropsychology, 17*(2), 255-264.
- Derogatis, L. R. (2001). *Brief Symptom Inventory 18: Administration, scoring, and procedures manual*. Minneapolis, MN: NCS Pearson Inc.
- Diener, E. D., Emmons, R. A., Larsen, R. J., & Griffin, S. (1985). The satisfaction with life scale. *Journal of Personality Assessment, 49*(1), 71-75.
- Dikmen, S. S., Bombardier, C. H., Machamer, J. E., Fann, J. R., & Temkin, N. R. (2004). Natural history of depression in traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 85*(90), 1457-1464.
- Dikmen, S. S., Machamer, J. E., Donovan, D. M., Winn, H. R., & Temkin, N. R. (1995). Alcohol use before and after traumatic head injury. *Annals of Emergency Medicine, 26*(2), 167-176.

Dikmen, S. S., Machamer, J. E., Powell, J. M., & Temkin, N. R. (2003). Outcome 3 to 5 years after moderate to severe traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84(10), 1449-1457.

Doig, E., Fleming, J., & Tooth, L. (2001). Patterns of community integration 2-5 years post-discharge from brain injury rehabilitation. *Brain Injury*, 15(9), 747-762.

Douglas, J. M., & Spellacy, F. J. (2000). Correlates of depression in adults with severe traumatic brain injury and their carers. *Brain Injury*, 14(1), 71-88.

Draper, K., & Ponsford, J. (2008). Cognitive functioning ten years following traumatic brain injury and rehabilitation. *Neuropsychology*, 22(5), 618-625.

Dunn, L., Henry, J., & Beard, D. (2003). Social deprivation and adult head injury: a national study. *Journal of Neurology, Neurosurgery & Psychiatry*, 74(8), 1060-1064.

Endler, N. S., & James, D. A. (1999). *Coping inventory for stressful situations manual* (2nd ed.). New York: Multi-Health Systems Inc.

Ensel, W., & Woelfel, M. Measuring the instrumental and expressive functions of social support. In N. Lin, A. Dean & W. Ensel (Eds.). *Social Support, Life Events, and Depression* (pp. 129-152). Orlando, FL: Academic Press.

Fann, J. R., Hart, T., & Schomer, K. G. (2009). Treatment for depression after traumatic brain injury: a systematic review. *Journal of Neurotrauma*, 26(12), 2383-2402.

Faul, M., Xu, L., Wald, M. M., & Coronado, V. G. (2010). Traumatic brain injury in the United States: emergency department visits, hospitalizations, and deaths. Atlanta (GA): Centers for Disease Control and Prevention. *National Center for Injury Prevention and Control*.

Finset, A., Dyrnes, S., Krogstad, J. M., & Berstad, J. (1995). Self-reported social networks and interpersonal support 2 years after severe traumatic brain injury. *Brain Injury*, 9(2), 141-150.

Fleming, J. M., Strong, J., & Ashton, R. (1998). Cluster analysis of self-awareness levels in adults with traumatic brain injury and relationship to outcome. *The Journal of Head Trauma Rehabilitation*, 13(5), 39-51.

Folkman, S., & Lazarus, R. S. (1985). If it changes it must be a process: A theoretical analysis. *Journal of Personality and Social Psychology*, 46(1), 150-170.

Folkman, S., & Lazarus, R. S. (1988). *Manual for the ways of coping questionnaire*. Palo Alto, CA: Consulting Psychologists Press.

Folkman, S., & Moskowitz, J. T. (2004). Coping: Pitfalls and promise. *Annual Review of Psychology*, 55, 745-774.

Glenn, M. B., O'Neil-Pirozzi, T., Goldstein, R., Burke, D., & Jacob, L. (2001). Depression amongst outpatients with traumatic brain injury. *Brain injury*, 15(9), 811-818.

Glucksman, E. (1994). Alcohol and accidents. *British medical bulletin*, 50(1), 76-84.

Godfrey, H. P., & Shum, D. (2000). Executive functioning and the application of social skills following traumatic brain injury. *Aphasiology*, 14(4), 433-444.

Gomez-Hernandez, R., Max, J. E., Kosier, T., Paradiso, S., & Robinson, R. G. (1997). Social impairment and depression after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 78(12), 1321-1326.

Grant, I., Prigatano, G. P., Heaton, R. K., McSweeny, A. J., Wright, E. C., & Adams, K. M. (1987). Progressive neuropsychologic impairment and hypoxemia: relationship in chronic obstructive pulmonary disease. *Archives of General Psychiatry*, 44(11), 999-006.

Gurr, B., & Coetzer, B. R. (2005). The effectiveness of cognitive-behavioural therapy for post-traumatic headaches. *Brain Injury*, 19(7), 481-491.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, 23(1), 56-62.

Hanks, R. A., Rapport, L. J., Wertheimer, J., & Koviak, C. (2012). Randomized controlled trial of peer mentoring for individuals with traumatic brain injury and their significant others. *Archives of Physical Medicine and Rehabilitation*, 93(8), 1297-1304.

Hart, T., Brenner, L., Clark, A. N., Bogner, J. A., Novack, T. A., Chervoneva, I., Nakase-Richardson, R., & Arango-Lasprilla, J. C. (2011). Major and minor depression after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 92(8), 1211-1219.

Hart, T., Hoffman, J. M., Pretz, C., Kennedy, R., Clark, A. N., & Brenner, L. A. (2012). A longitudinal study of major and minor depression following traumatic brain injury. *Archives of physical medicine and rehabilitation*, 93(8), 1343-1349.

Hart, T., Whyte, J., Kim, J., & Vaccaro, M. (2005). Executive function and self-awareness of "real-world" behavior and attention deficits following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 20(4), 333-347.

Heaton, R. K., Miller, S. W., Taylor, M. J., & Grant, I. (2004). *Revised comprehensive norms for an expanded Halstead-Reitan Battery: Demographically adjusted neuropsychological norms for African American and Caucasian Adults*. Florida: Psychological Assessment Resources Inc.

Hinkeldey, N. S., & Corrigan, J. D. (1990). The structure of head-injured patients' neurobehavioural complaints: a preliminary study. *Brain Injury*, 4(2), 115-133.

- Holsinger, T., Steffens, D. C., Phillips, C., Helms, M. J., Havlik, R. J., Breitner, J., Guralnik, J. M., & Plassman, B. L. (2002). Head injury in early adulthood and the lifetime risk of depression. *Archives of General Psychiatry*, 59(1), 17-22.
- Hoofien, D., Gilboa, A., Vakil, E., & Donovick, P. J. (2001). Traumatic brain injury (TBI) 10–20 years later: A comprehensive outcome study of psychiatric symptomatology, cognitive abilities and psychosocial functioning. *Brain Injury*, 15(3), 189–209
- Horner, M. D., Ferguson, P. L., Selassie, A. W., Labbate, L. A., Kniele, K., & Corrigan, J. D. (2005). Patterns of alcohol use 1 year after traumatic brain injury: a population-based, epidemiological study. *Journal of the International Neuropsychological Society*, 11(3), 322-330.
- Janowsky, D. S., El-Yousef, M. K., & Davis, J. M. (1974). Acetylcholine and depression. *Psychosomatic Medicine*, 36(3), 248-257.
- Jorge, R. E. (2005). Neuropsychiatric consequences of traumatic brain injury: a review of recent findings. *Current Opinion in Psychiatry*, 18(3), 289-299.
- Jorge, R. E., Robinson, R. G., Arndt, S. V., & Forrester, A. W., (1993). Comparison between acute – and delayed – onset depression following traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences*, 5(1), 43-49.
- Jorge, R. E., Robinson, R. G., Moser, G., Tateno, A., Crespo-Faccorro, B., & Arndt, S. V. (2004). Major depression following traumatic brain injury. *Archives of General Psychiatry*, 61(1), 42-50.
- Jorge, R. E., Robinson, R. G., Arndt, S. V., Starkstein, S. E., Forrester, A. W., & Geisler, F. (1993). Depression following traumatic brain injury: a 1 year longitudinal study. *Journal of Affective Disorders*, 27(4), 233-243.

- Jorge, R. E., & Starkstein, S. E. (2005). Pathophysiological aspects of major depression following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 20(6), 475-487.
- Kashluba, S., Hanks, R. A., Casey, J. E., & Millis, S. R. (2008). Neuropsychologic and disability after complicated mild traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(5), 904-911.
- Kathol, R. G., Noyes, R., Williams, J., & Mutgi, A. (1990). Diagnosing depression in patients with medical illness. *Psychosomatics: Journal of Consultation Liaison Psychiatry*, 31(4), 434-440.
- Keefe, F. J., Caldwell, D. S., Queen, K. T., Gil, K. M., Martinez, S., Crisson, J. E., Ogden, W., & Nunley, J. (1987). Pain coping strategies in osteoarthritis patients. *Journal of Consulting and Clinical Psychology*, 55(2), 208-212.
- Kelly, M. P., Johnson, C. T., Knoller, N., Drubach, D. A., & Winslow, M. M. (1997). Substance abuse, traumatic brain injury and neuropsychological outcome. *Brain injury*, 11(6), 391-402.
- Kendall, E., Shum, D., Lack, B., Bull, S., &, Fee, C. (2001). Coping following traumatic brain injury: the need for contextually sensitive assessment. *Brain Impairment*, 2(2), 81–96.
- Kendall, E., & Terry, D. J. (2008). Understanding adjustment following traumatic brain injury: Is the Goodness-of-Fit coping hypothesis useful? *Social Science and Medicine*, 67(8), 1217-1224.
- Kennedy, R. E., Livingston, L., Riddick, A., Marwitz, J. H., Kreutzer, J. S., & Zasler, N. D. (2005). Evaluation of the Neurobehavioral Functioning Inventory as a depression screening tool after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 20(6), 512-526.

- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., Rush, J., Walters, E. E., & Wang, P. S. (2003). The epidemiology of major depressive disorder. *The Journal of the American Medical Association*, 289(23), 3095-3105.
- Kolakowsky-Hayner, S. A., Gourley III, E. V., Kreutzer, J. S., Marwitz, J. H., Cifu, D. X., & McKinley, W. O. (1999). Pre-injury substance abuse among persons with brain injury and persons with spinal cord injury. *Brain Injury*, 13(8), 571-581.
- Kolenikov, S. (2013, March 7). Re: Regression in SEM programs vs regression in statistical packages such as SPSS [Online forum comment]. Retrieved from <http://stats.stackexchange.com/questions/51558/regression-in-sem-programs-vs-regression-in-statistical-packages-such-as-spss>
- Kozloff, R. (1987). Networks of social support and the outcome of severe head injury, *Journal of Head Trauma Rehabilitation*, 2(3), 14–23.
- Kreutzer, J. S., Seel, R. T., & Marwitz, J. H. (1999). *Neurobehavioral Functioning Inventory: NFI*. San Antonio, TX: Psychological Corporation.
- Kreutzer, J. S., Witol, A. D., & Marwitz, J. H. (1996). Alcohol and drug use among young persons with traumatic brain injury. *Journal of Learning Disabilities*, 29(6), 643-651.
- Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9. *Journal of General Internal Medicine*, 16(9), 606-613.
- Krpan, K. M., Levine, B., Stuss, D. T., & Dawson, D. R. (2007). Executive function and coping at one-year post traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 29(1), 36-46.

- Krpan, K. M., Stuss, D. T., & Anderson, N. D. (2011). Coping behaviour following traumatic brain injury: What makes a planner plan and an avoider avoid? *Brain Injury*, 25(10), 989-996.
- Kwok, F. Y., Lee, T. M., Leung, C. H., & Poon, W. S. (2008). Changes of cognitive functioning following mild traumatic brain injury over a 3-month period. *Brain Injury*, 22(10), 740-751.
- Lazarus R, & Folkman S. (1984). *Stress, Appraisal and Coping*. New York: Springer.
- Levin, H. S., & Grossman, R. G. (1978). Behavioral sequelae of closed head injury: a quantitative study. *Archives of Neurology*, 35(11), 720-727.
- Lewis, R., & Rennick, P. (1979). *Manual for the repeatable cognitive-perceptual-motor battery*. Grosse Pointe Park, MI: Axon.
- Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). Neuropsychological Assessment (5th ed.). New York: Oxford University Press.
- Lucas, J. A., & Addeo, R. (2006). Traumatic brain injury and postconcussion syndrome. In P. J. Snyder, P. D. Nussbaum, & D. L. Robins (Eds.), *Clinical neuropsychology: A pocket handbook for assessment* (2nd ed., pp. 351-380). Washington DC: American Psychological Association.
- Lynch, S. M. (2003). *Missing data (Soc 504)*. Retrieved from:
<http://www.princeton.edu/~slynch/soc504/missingdata.pdf>
- Maas, A. I. R., Stocchetti, N., & Bullock, R. (2008). Moderate and severe traumatic brain injury in adults. *Lancet Neurology*, 7(8), 728-741.

- Malec, J. F., Brown, A. W., Moessner, A. M., Stump, T. E., & Monahan, P. (2010). A preliminary model for posttraumatic brain injury depression. *Archives of Physical Medicine and Rehabilitation*, 91(7), 1087-1097.
- Max, J. E., Arndt, S., Castillo, C. S., Bokura, H., Robin, D. A., Lindgren, S. D, Smith Jr., W. L., Sato, Y., & Mattheis, P. J. (1998). Attention-deficit hyperactivity symptomatology after traumatic brain injury: a prospective study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 37(8), 841-847.
- McAllister, T. W. (2011). Neurobiological consequences of traumatic brain injury. *Dialogues in Clinical Neuroscience*, 13(3), 287-300.
- McAvinue, L., O'Keeffe, F., McMackin, D., & Robertson, I. H. (2005). Impaired sustained attention and error awareness in traumatic brain injury: Implications for insight. *Neuropsychological Rehabilitation*, 15(5), 569-587.
- McCarthy, M. L., Dikmen, S. S., Langlois, J. A., Selassie, A. W., Gu, J. K., & Horner, M. D. (2006). Self-reported psychosocial health among adults with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 87(7), 953-961.
- McCauley, S. R., Levin, H. S., Vanier, M., Mazaux, J. M., Boake, C., Goldfader, P. R., Rockers, D., Butters, M., Kareken, D. A., Lambert, J., & Clifton, G. L. (2001). The neurobehavioural rating scale-revised: sensitivity and validity in closed head injury assessment. *Journal of Neurology, Neurosurgery & Psychiatry*, 71(5), 643-651.
- McKnight, P. E., McKnight, K. M., & Figueiredo, A. J. (2007). *Missing data: A gentle introduction*. New York: Guilford Press.

- McMillan, T., Robertson, I. H., Brock, D., & Chorlton, L. (2002). Brief mindfulness training for attentional problems after traumatic brain injury: A randomised control treatment trial. *Neuropsychological Rehabilitation*, 12(2), 117-125.
- Meachen, S. J., Hanks, R. A., Millis, S. R., & Rapport, L. J. (2008). The Reliability and Validity of the Brief Symptom Inventory- 18 in Persons with Traumatic Brain Injury. *Archives of Physical Medicine and Rehabilitation*, 89(5), 958-965.
- Mitrushina, M. (2005). *Handbook of normative data for neuropsychological assessment*. New York: Oxford University Press.
- Morton, M. V., & Wehman, P. (1995). Psychosocial and emotional sequelae of individuals with traumatic brain injury: a literature review and recommendations. *Brain Injury*, 9(1), 81-92.
- Niemann, H., Ruff, R. M., & Kramer, J. H. (1996). An attempt towards differentiating attentional deficits in traumatic brain injury. *Neuropsychology Review*, 6(1), 11-46.
- Oddy, M., Humphrey, M., & Uttley, D. (1978). Stress upon the relatives of head-injured patients. *The British Journal of Psychiatry*, 133(6), 507-513.
- O'Keeffe, F., Dockree, P., Moloney, P., Carton, S., & Robertson, I. H. (2007). Awareness of deficits in traumatic brain injury: A multidimensional approach to assessing metacognitive knowledge and online-awareness. *Journal of the International Neuropsychological Society*, 13(01), 38-49.
- Olson, I. R., Plotzker, A., & Ezzyat, Y. (2007). The enigmatic temporal pole: a review of findings on social and emotional processing. *Brain*, 130(7), 1718-1731.
- Olver, J. H., Ponsford, J. L., & Curran, C. A. (1996). Outcome following traumatic brain injury: a comparison between 2 and 5 years after injury. *Brain Injury*, 10(11), 841-848.

- O'Neill, J., Hibbard, M. R., Broivn, M., Jaffe, M., Sliwinski, M., Vandergoot, D., & Weiss, M. J. (1998). The effect of employment on quality of life and community integration after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 13(4), 68-79.
- Ownsworth, T. (2005). The impact of defensive denial upon adjustment following traumatic brain injury. *Neuropsychoanalysis*, 7(1), 83-94.
- Ownsworth, T. L., & Oei, T. P. (1998). Depression after traumatic brain injury: conceptualization and treatment considerations. *Brain Injury*, 12(9), 735-751.
- Paul, J. (1992). Depression in patients with acute traumatic brain injury. *American Journal of Psychiatry*, 149(7), 918-923.
- Pariante, C. M., & Lightman, S. L. (2008). The HPA axis in major depression: classical theories and new developments. *Trends in Neurosciences*, 31(9), 464-468.
- Ponsford, J., Draper, K., & Schonberger, M. (2008). Functional outcome 10 years after traumatic brain injury: its relationship with demographic, injury severity, and cognitive and emotional status. *Journal of the International Neuropsychological Society*, 14(2), 233-242.
- Ponsford, J., Whelan-Goodinson, R., & Bahar-Fuchs, A. (2007). Alcohol and drug use following traumatic brain injury: A prospective study. *Brain Injury*, 21(13-14), 1385-1392.
- Povlishock, J. T., & Katz, D. I. (2005). Update of neuropathology and neurological recovery after traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 20(1), 76-94.
- Powell, J. M., Machamer, J. E., Temkin, N. R., & Dikmen, S. S. (2001). Self-report of extent of recovery and barriers to recovery after traumatic brain injury: a longitudinal study. *Archives of Physical Medicine and Rehabilitation*, 82(8), 1025-1030.

- Powner, D.J., Boccalandro, C., Alp, M. S., & Vollmer, D. G. (2006). Endocrine failure after traumatic brain injury in adults. *Neurocritical Care*, 5(1), 61-70.
- Radloff, L. S. (1977). The CES-D scale A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1(3), 385-401.
- Raftery, A. E. (1995). Bayesian model selection in social research. P. V. Marsden (Eds.). *Sociological Methodology* (Vol. 25, pp. 111-163). Oxford: Blackwell.
- Rapoport, M.J., McCauley, S., Levin, H., Song, J., & Feinstein, A. (2002). The role of injury severity in neurobehavioral outcome 3 months after traumatic brain injury. *Cognitive and Behavioral Neurology*, 15(2), 123-132.
- Rapoport, M. J., McCullagh, S., Shammi, P., & Feinstein, A. (2005). Cognitive impairment associated with major depression following mild and moderate traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 17(1), 61-65.
- Rappaport, M., Hall, K. M., Hopkins, K., Belleza, T., & Cope, D. N. (1982). Disability rating scale for severe head trauma: coma to community. *Archives of Physical Medicine and Rehabilitation*, 63(3), 118-123.
- Rogers, J. M., & Read, C. A. (2007). Psychiatric comorbidity following traumatic brain injury. *Brain injury*, 21(13-14), 1321-1333.
- Ross, C. E., & Mirowsky, J. (2013). The sense of personal control: Social structural causes and emotional consequences. *Handbook of the sociology of mental health* (pp. 379-402). Netherlands: Springer.
- Russell, D., Peplau, L. A., & Cutrona, C. E. (1980). The revised UCLA Loneliness Scale: Concurrent and discriminant validity evidence. *Journal of Personality and Social Psychology*, 39(3), 472-480.

Ruttan, L., Martin, K., Liu, A., Colella, B., & Green, R. E. (2008). Long-term cognitive outcome in moderate to severe traumatic brain injury: a meta-analysis examining timed and untimed tests at 1 and 4.5 or more years after injury. *Archives of Physical Medicine and Rehabilitation*, 89(12), S69-S76.

Salthouse, T. A., Schroeder, D. H., & Ferrer, E. (2004). Estimating retest effects in longitudinal assessments of cognitive functioning in adults between 18 and 60 years of age. *Developmental Psychology*, 40(5), 813-822.

Sarason, I. G., Levine, H. M., Basham, R. B., & Sarason, B. R. (1983). Assessing social support: The social support questionnaire. *Journal of Personality and Social Psychology*, 44(1), 127-139.

Seel, R. T., Kreutzer, J. S., Rosenthal, M., Hammond, F. M., Corrigan, J. D., & Black, K. (2003). Depression after traumatic brain injury: A national institute on disability and rehabilitation research model systems multicenter investigation. *Archives of Physical Medicine and Rehabilitation*, 84(2), 177-184.

Seel, R. T., Macciocchi, S., & Kreutzer, J. S. (2010). Clinical considerations for the diagnosis of major depression after moderate to severe TBI. *The Journal of Head Trauma Rehabilitation*, 25(2), 99-112.

Selzer, M. L., Vinokur, A., & Rooijen, L. V. (1975). A self-administered short Michigan alcoholism screening test (SMAST). *Journal of Studies on Alcohol and Drugs*, 36(01), 117-126.

Shandro, J. R., Rivara, F. P., Wang, J., Jurkovich, G. J., Nathens, A. B., & MacKenzie, E. J. (2009). Alcohol and risk of mortality in patients with traumatic brain injury. *The Journal of Trauma and Acute Care Surgery*, 66(6), 1584-1590.

- Sigurdardottir, S., Andelic, N., Roe, C., & Schanke, A. K. (2009). Cognitive recovery and predictors of functional outcome 1 year after traumatic brain injury. *Journal of the International Neuropsychological Society*, 15(05), 740-750.
- Smith, J. L., Magill-Evans, J., & Brintnell, S. (1998). Life satisfaction following traumatic brain injury. *Canadian Journal of Rehabilitation*, 11(3), 131-140.
- Sparadeo, F. R., & Gill, D. (1989). Effects of prior alcohol use on head injury recovery. *The Journal of Head Trauma Rehabilitation*, 4(1), 75-81.
- Spitz, G., Ponsford, J. L., Rudzki, D., & Maller, J. J. (2012). Association between cognitive performance and functional outcome following traumatic brain injury: A longitudinal multilevel examination. *Neuropsychology*, 26(5), 604-612.
- Spitz, G., Schonberger, M., & Ponsford, J. (2012). The relations among cognitive impairment, coping style, emotional adjustment following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 21(3), 248-259.
- Stein, M. B., Kennedy, C. M., & Twamley, E. W. (2002). Neuropsychological function in female victims of intimate partner violence with and without posttraumatic stress disorder. *Biological Psychiatry*, 52(11), 1079-1088.
- Strandberg, T. (2009). Adult with acquired traumatic brain injury: Experiences of a changover process and consequences in everyday life. *Social Work & Health Care*, 48(3), 276-297.
- Stratta, P., Rossi, A., Mancini, F., Cupillari, M., Mattei, P., & Casacchia, M. (1993). Wisconsin Card Sorting Test performance and educational level in schizophrenic and control samples. *Cognitive and Behavioral Neurology*, 6(3), 149-153.
- Strauss, E., Sherman, E. M., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary (3rd ed.)*. New York: Oxford University Press.

- Stuss, D. T., & Knight, R. T. (Eds.). (2002). *Principles of frontal lobe function*. New York: Oxford University Press.
- Taylor, L. A., Kreutzer, J. S., Demm, S. R., & Meade, M. A. (2003). Traumatic brain injury and substance abuse: A review and analysis of the literature. *Neuropsychological Rehabilitation, 13*(1-2), 165-188.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: a practical scale. *The Lancet, 304*(7872), 81-84.
- Tellegen, A., Watson, D., & Clark, L. A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063-1070.
- Terry, D. J., & Hynes, G. J. (1998). Adjustment to a low-control situation: Reexamining the role of coping responses. *Journal of Personality and Social Psychology, 74*(4), 1078-1092.
- Thurman, D. J., Alverson, C., Dunn, K. A., Guerrero, J., & Sniezek, J. E. (1999). Traumatic brain injury in the United States: a public health perspective. *The Journal of Head Trauma Rehabilitation, 14*(6), 602-615.
- Tiersky, L. A., Anselmi, V., Johnston, M. V., Kurtyka, J., Roosen, E., Schwartz, T., & DeLuca, J. (2005). A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 86*(8), 1565-1574.
- Tomberg, T., Toomela, A., Ennok, A., & Tikk, A. (2007). Changes in coping strategies, social support, optimism, and health-related quality of life following traumatic brain injury: A longitudinal study. *Brain Injury, 21*(5), 479-488.

- Tomberg, T., Toomela, A., Pulver, A., & Tikk, A. (2005). Coping strategies, social support, life oriented and health-related quality of life following traumatic brain injury. *Brain Injury*, 19(14), 1181-1190.
- Wagner, A. K., Sasser, H. C., Hammond, F. M., Wierciszewski, D., & Alexander, J. (2000). Intentional traumatic brain injury: epidemiology, risk factors, and associations with injury severity and mortality. *Journal of Trauma and Acute Care Surgery*, 49(3), 404-410.
- Ware, J. E., Snow, K. K., Kosinski, M., & Gandek, B. (1993). *SF-36 health survey: manual and interpretive guide*. Boston: Health Institute, New England Medical Centre.
- West, S. L. (2011). Substance use among persons with traumatic brain injury: A review. *NeuroRehabilitation*, 29(1), 1-8.
- Wiegner, S., & Donders, J. (1999). Performance on the California Verbal Learning Test after traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 21(2), 159-170.
- Wiersma, J. E., van Oppen, P., Van Schaik, D. J., van der Does, A. J., Beekman, A. T., & Penninx, B. W. (2011). Psychological characteristics of chronic depression: a longitudinal cohort study. *The Journal of Clinical Psychiatry*, 72(3), 288-294.
- Willer, B., Allen, K., Durnan, M. C., & Ferry, A. (1990). Problems and coping strategies of mothers, siblings and young adult males with traumatic brain injury. *Canadian Journal of Rehabilitation*, 3(3), 167-173.
- Willer, B. S., Allen, K. M., Liss, M., & Zicht, M. S. (1991). Problems and coping strategies of individuals with traumatic brain injury and their spouses. *Archives of Physical Medicine and Rehabilitation*, 72(7), 460-464.

- Wise, E. K., Mathews-Dalton, C., Dikmen, S., Temkin, N., Machamer, J., Bell, K., & Powell, J. M. (2010). Impact of traumatic brain injury on participation in leisure activities. *Archives of Physical Medicine and Rehabilitation*, 91(9), 1357-1362.
- Wolters, G., Stapert, S., Brands, I., & van Heugten, C. (2010). Coping styles in relation to cognitive rehabilitation and quality of life after brain injury. *Neuropsychological Rehabilitation*, 20(4), 587-600.
- Wood, R. L., & Rutherford, N. A. (2006). Demographic and cognitive predictors of long-term psychosocial outcome following traumatic brain injury. *Journal of the International Neuropsychological Society*, 12(3), 350-358.
- Yeates, K. O., Swift, E., Taylor, H. G., Wade, S. L., Drotar, D., Stancin, T., & Minich, N. (2004). Short- and long-term social outcomes following pediatric traumatic brain injury. *Journal of the International Neuropsychological Society*, 10(3), 412-426.
- Ylvisaker, M., Turkstra, L. S., & Coelho, C. (2005, November). Behavioral and social interventions for individuals with traumatic brain injury: A summary of the research with clinical implications. In *Seminars in speech and language* (Vol. 26, No. 04, pp. 256-267). New York: Thieme Medical Publishers, Inc.
- Zencius, A. H., & Wesolowski, M. D. (1999). Is the social network analysis necessary in the rehabilitation of individuals with head injury? *Brain Injury* 13(9), 723–727.
- Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica*, 67(6), 361-370.

Appendices

Appendix A

Information Regarding Missing Data for the Early Recovery Group (N=119)

Psychosocial variables	Number of missing cases	Percentage missing
Depression (BSI-18 depression subscale)	4	3%
Emotion-focused coping	1	1%
Problem-focused coping	1	1%
Perceived social support	2	2%
Cognitive and neurological variables		
Attention (DVT score)	27	22%
Memory (CVLT-2 total acquisition trial score)	10	8%
EF (WCST perseverative errors score)	25	21%

Appendix B

Information Regarding Missing Data for the Late Recovery Group (N=159)

Psychosocial variables	Number of missing cases	Percentage missing
Depression (BSI-18 depression subscale)	9	6%
Emotion-focused coping	3	2%
Problem-focused coping	3	2%
Perceived social support	6	4%
Cognitive and neurological variables		
Attention (DVT score)	34	21%
Memory (CVLT-2 total acquisition trial score)	17	11%
EF (WCST perseverative errors score)	30	19%

Appendix C

Pearson and Point-Biserial Correlations between Dependent and Independent Variables for the Early Recovery Group (N=80).

	Injury severity	Alcohol use	Perceived social support	Problem-focused coping	Emotion-focused coping	EF	Attention	Memory	Depression
Injury severity	1.00								
Alcohol use	0.03	1.00							
Perceived social support	-0.02	-0.12	1.00						
Problem-focused coping	-0.03	0.09	0.20	1.00					
Emotion-focused coping	-0.04	0.13	-0.35	-0.08	1.00				
EF	0.05	-0.10	0.01	-0.03	0.05	1.00			
Attention	-0.09	0.06	0.08	0.03	0.02	0.04	1.00		
Memory	0.16	-0.10	0.05	-0.04	-0.29	-0.21	-0.34	1.00	
Depression	-0.01	0.18	-0.44	-0.29	0.54	-0.03	0.06	-0.10	1.00

Appendix D

Pearson and Point-Biserial Correlations between Dependent and Independent Variables for the Late Recovery Group (N=107).

	Injury severity	Alcohol use	Perceived social support	Problem-focused coping	Emotion-focused coping	EF	Attention	Memory	Depression
Injury severity	1.00								
Alcohol use	0.07	1.00							
Perceived social support	-0.06	-0.14	1.00						
Problem-focused coping	0.02	0.04	0.13	1.00					
Emotion-focused coping	0.11	0.12	-0.20	0.11	1.00				
EF	-0.01	-0.12	-0.02	-0.05	-0.01	1.00			
Attention	-0.21	-0.05	-0.08	0.04	0.09	0.43	1.00		
Memory	0.15	0.11	0.13	-0.01	-0.12	-0.41	-0.45	1.00	
Depression	0.15	0.22	-0.37	-0.02	0.51	-0.01	-0.12	-0.06	1.00

VITA AUCTORIS

Elmar Gardizi was born in Kabul, Afghanistan in 1983. He completed his undergraduate degree at the University of Toronto where he obtained his BSc (Hons) with a major in Neuroscience and minors in Psychology and Biology in 2007. From there, he was admitted into the Ph.D. program in Clinical Neuropsychology at the University of Windsor. He obtained his Master degree in 2010 and successfully defended his dissertation in January 2015. He is expected to graduate in the fall of 2015.