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Cognitive and emotional outcomes after traumatic brain injury in older adults

Kelly An
University of Windsor

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By

Kelly An

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Kelly An

APPROVED BY:

_____

Dr. S. Horton
Department of Kinesiology

_____

Dr. C. Abeare
Department of Psychology

_____

Dr. A. Baird, Advisor
Department of Psychology

September 4, 2014

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ABSTRACT

Older adults have a higher rate of mortality and complications after a traumatic brain injury (TBI) compared to other age groups, but little is known about their post-TBI cognitive and emotional outcomes. The present study aims to (1) elucidate the relation between age at time of injury with cognitive and emotional functioning post-TBI, and (2) examine whether age at time of injury moderates the relationship between post-TBI cognitive and emotional outcomes. Data from participants (n = 67) with mild-complicated to severe TBI who completed neuropsychological assessments for compensation purposes were retrospectively analyzed. Results revealed that age at time of injury was not related to cognitive and emotional functioning and did not moderate the relation between cognitive and emotional outcomes. These findings suggest that older adults who survive a TBI show cognitive and emotional outcomes similar to those of younger adults on a long-term basis.

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I. INTRODUCTION

Traumatic brain injury (TBI) poses a significant problem for both the individual and the larger health care and economic systems. As a leading cause of mortality worldwide, it is estimated that over 194,000 hospitalizations were due to TBI in Canada in the 2003-2004 fiscal year (Canadian Institute for Health Information (CIHI), 2006). In terms of societal costs, TBI presents a large toll on resources, time, and loss of work days each year. However, the effects on individuals are all the more devastating. For those who survive, many are left with persisting psychological and cognitive symptoms, thus affecting their ability to return to work and daily activities. Although these consequences are pertinent across the lifespan, one group of individuals in particular appears to be more affected by the consequences of TBI than any other group. Previous literature has suggested that older adults are particularly vulnerable to the negative effects of head injury, with higher rates of hospitalizations, medical complications, and deaths compared to younger adults (Goleburn & Golden, 2001). Furthermore, older adults are less likely to function independently in daily activities and employment after a TBI (Goleburn & Golden, 2001). It is estimated that 38% of all TBI hospital admissions between 2002 and 2008 were those age 65 and over (Depreitere, Meyfroidt, Roosen, Ceuppens, & Grandas, 2012). In Canada, older adults accounted for 29% of head injuries, second only to children (30%; CIHI, 2006). This high prevalence, combined with the high mortality and morbidity in older adults sustaining TBI, underlines the magnitude and significance of this issue in our society.

Despite the devastating consequences of TBI in this population, relatively little research on post-TBI outcomes has been conducted with older adults compared to

younger adults. Increased rates of mortality and morbidity in older adults have been well documented in the literature across severities and types of injuries (e.g., McIntyre, Mehta, Aubut, Dijkers, & Teasell, 2013). In terms of post-TBI cognitive outcomes and age, however, studies have produced inconsistent findings (e.g., Ashman et al., 2008; Mazzucchi, Cattelani, Missale, Gugliotta, Brianti, & Parma, 1992). Even less research has been conducted examining the emotional and psychosocial outcomes in older adults. For the studies that have been conducted, many have focused only on comparing outcomes of older adults sustaining TBI to age-matched healthy controls (e.g., Rapoport, Herrmann, Shammi, Kiss, Phillips, & Feinstein, 2006), but few have compared older adults to younger adults directly (e.g., Senathi-Raja, Ponsford, & Schönberger, 2010a). Hence, despite our general knowledge that age is a significant predictor of global outcome, much less is known about how older adults differ from younger adults in specific domains of cognitive and emotional functioning after a TBI. Such research is warranted, as differences exist between the brains of older and younger adults, and the impact of a head injury may interact with the aging process (Vollmer & Eisenberg, 1990). Therefore, many of the findings with younger adults may not be applicable to older adults, whose aging brains and different life circumstances introduce unique variables to take into account.

The current study aimed to narrow the gap of knowledge in this area, and particularly to elucidate the relationship between cognitive and emotional outcomes after a mild complicated to severe TBI for older and younger adults from about 3 months to 13 years post-injury. Specifically, cognitive and emotional outcomes in the context of the current study refer to performance on neuropsychological testing and self-reported

depression and anxiety symptoms on standardized questionnaires, respectively. It is important to note that difficulties in cognition and self-regulation after a TBI may go beyond those observed in the structured, one-on-one environment of neuropsychological testing. In addition, the emotional consequences post-TBI extend beyond just depression and anxiety symptoms, and may include, for instance, changes in personality and development of other psychological symptoms. While these other aspects are acknowledged as important consequences post-TBI, the scope of the current research focused on examining the specific cognitive and emotional outcomes described.

Although there is no universally agreed upon age at which to apply the term "older," for the purposes of this study and the following review, older adults will refer to individuals age 50 and over. This definition was chosen because, aside from the fact that a majority of the studies reviewed utilize this age range (Goldstein & Levin, 2001; Rapoport et al., 2006; Rapoport et al., 2008; Whelan-Goodinson, Ponsford, Schönberger, & Johnston, 2010), it is during the sixth decade of life when many physiological changes occur and the incidence of sustaining complications from TBI rises (Goldstein & Levin, 2001). However, there is some variability between studies in the definition, with some studies including those over 55 years old (e.g., Ashman et al., 2008), 60 years old (e.g., Rapoport et al., 2001), and 65 years old as older adults (e.g., Deb & Burns, 2007). This variability was taken into account in the review when inconsistent findings between studies arose.

There is also variability in how injury severity is measured and defined in the literature. While most studies determine TBI severity with the Glasgow Coma Scale (GCS), some studies include post-traumatic amnesia/post-traumatic confusion

(PTA/PTC), length of loss of consciousness (LOC), and evidence on neuroimaging in their criteria. The GCS is a brief scale used to measure levels of consciousness on 3 aspects of behavior (motor, verbal, and eye opening), and provides a score between 3 and 15 (Teasdale & Jennett, 1974). A score between 3 to 8 is indicative of severe TBI, 9 to 12 moderate TBI, and 13 to 15 mild TBI (Lezak, Howieson, Bigler, & Tranel, 2012). In addition, those who meet criteria for mild TBI but show evidence of intracranial brain abnormality on neuroimaging are classified as having a mild complicated TBI (Williams, Levin, & Eisenberg, 1990). Research has supported the idea that the cognitive and functional outcomes and recovery of mild complicated TBI are more consistent with TBI of moderate severity than mild uncomplicated TBI (Borgaro, Prigatano, Kwasnica, & Rexer, 2003; Kashluba, Hanks, Casey, & Millis, 2008; Williams et al., 1990). As such, mild complicated TBI was examined and grouped with moderate TBI for this study and the following literature review.

II. REVIEW OF THE LITERATURE

Traumatic brain injury etiology

Traumatic brain injuries may be a result of various events. In older adults, the most common cause of a TBI is from a fall, which accounts for 50-80% of all TBI cases in this population (Goleburn & Golden, 2001). Cognitive impairment, medication side effects, physical weakness, poor vision, and chronic and acute physical conditions all increase the risk of falls for this population (Goleburn & Golden, 2001). Following a fall, older adults are more likely to sustain secondary complications, such as intracranial hemorrhaging and hematomas, than younger adults (Goleburn & Golden, 2001). Hence, not only are falls the main culprit for sustaining a TBI in older adults, but also cause more complications in this group compared to younger adults. Aside from falls, older adults may sustain a TBI from motor vehicle accidents, which are the second most common cause of a TBI in this age group (Goleburn & Golden, 2001). While these incidents are not as frequent as falls, their consequences are often more devastating, as there is a higher chance that the brain injuries sustained in a motor vehicle accident will be severe or fatal (Goleburn & Golden, 2001). Other incidents, such as gunshot wounds or physical assaults, are less common etiologies of TBI in older adults than falls and motor vehicle accidents.

Neuropathology of TBI

After initial impact to the head, a cascade of cellular and biochemical events takes place that results in the symptoms frequently observed after a TBI. Some of these physiological changes in the brain occur immediately after impact, while others develop days to weeks after the injury. It is often the complications after the injury instead of the impact itself which create the disruption of brain functioning and corresponding

symptoms (Lezak et al., 2012). For example, individuals sustaining a TBI may suffer from an insufficient oxygen supply (hypoxia) or blood supply (ischemia) that further damages the brain (Lezak et al., 2012). Other processes, such as elevated intracranial pressure, blood clotting, and inflammation also may occur after the initial injury and interrupt normal brain activities (Lezak et al., 2012).

TBI may be classified as a closed head injury (CHI), in which the brain has not been contacted directly by an external source, or penetrating head injury (PHI), in which the brain has been penetrated by some object. While CHI and PHI share some similar physiological processes (e.g., both involve shearing-tearing and structural changes of axons), the nature and mechanisms of injury are largely different for the two types (Lezak et al., 2012). In addition, CHI and PHI may produce different symptomology and outcomes, which further emphasizes that the two should not be treated as equivalent. Because it is more pertinent to the current study, the neuropathology and sequence of events for CHI will be the focus of the following discussion.

CHI often involves the head hitting a stationary object or a moving object hitting the head. In addition, CHI may involve sudden movement of the head as a result of movement from the body. The impact from these events involves both acceleration and deceleration movement from inertial forces, which may result in contusions (Lezak et al., 2012). To illustrate these biomechanical processes, imagine an individual who has sustained a CHI from a motor vehicle accident. With the sudden halt of the vehicle, the individual's head moves forward with angular acceleration forces, which alone may result in shearing and strain on axons. The individual's head subsequently hits the dashboard. This direct blow of where the external force impacted the head is the coup,

where the brain is impacted on the bony ridges of the skull. Subsequently, the brain may bounce back to the opposite side of the skull, producing what is known as contrecoup lesions (Lezak et al., 2012). Both coup and contrecoup contusions account for many of the common patterns of damage. For example, the frontal and temporal lobes are often sites of injury and are reflected in the commonly observed difficulties in executive function and memory after a TBI (Lezak et al., 2012). Similarly, the corpus callosum is vulnerable to damage from the acceleration and rotational forces, which is reflected in the frequently observed symptom of slowed processing speed post-TBI (Lezak et al., 2012).

Aside from the focal damage to the brain upon impact, CHI also produces a sequence of events resulting in diffuse damage to the brain. There may be diffuse axonal injury to various regions of the brain in which there is minute damage to and atrophy in white matter (Lezak et al., 2012). In addition, there is also evidence that brain gray matter may be widely affected and increases in ventricular volume are commonly observed (Lezak et al., 2012). These widespread diffuse changes are related to the severity of injury such that more diffuse injury should be expected with more severe injuries (Lezak et al., 2012). Because of this widespread, diffuse damage in the brain, patients with severe injuries often exhibit a general decline of cognitive abilities (Lezak et al., 2012).

In addition to focal and diffuse neuronal damage, the impact of the CHI may disrupt the vasculature in the brain (Lezak et al., 2012). Intracranial hemorrhage is not an uncommon consequence of CHI, and may appear in different forms depending on the site and nature of the hemorrhage. Particularly relevant to the current study, hematomas are more likely to occur in older adults, and hence contribute to their poorer outcome (Vollmer & Eisenberg, 1990). Hematomas are blood clots formed in the coverings over

the brain, as in subdural and extradural hematomas, or within the brain and may compress the brain at the point where the blood is pooled. In the case of subdural and extradural hematomas, this compression often creates a crescent shaped depression on the surface of the brain and can cause substantial neuronal damage, elevate intracranial pressure, and reduce cerebral blood flow (Lezak et al., 2012). The acceleration and impact forces of CHI may also stretch and tear the brain's network of capillaries and cause intraparenchymal hemorrhages (Lezak et al., 2012). This damages the brain as neurons are starved of oxygen supply and the direct contact of blood with tissues can have a toxic effect. Finally, intraventricular hemorrhages could occur if there is damage to the lining of the ventricles or if blood makes its way into the ventricular spaces (Lezak et al., 2012).

After the initial mechanical events resulting in the primary injury described, a cascade of biomolecular processes then begins in the cells, which may take minutes to months to develop. As mentioned, it is often these secondary injuries that significantly affect long-term outcome. Recent literature has suggested that secondary injuries are most predictive of morbidity and functioning in those who survive the initial impact (Greve & Zink, 2009). While it is outside the scope of this review to discuss the complex cellular and molecular interactions involved in the secondary injury phase, major sources of damage include excitotoxicity from glutamate release, changes in calcium homeostasis, and oxidant damage, all which play a role in atrophy or neuronal death of the cells (Greve & Zink, 2009). There is also evidence that the blood brain barrier could be compromised from the TBI, thus further decreasing the brain's ability to maintain homeostasis and regulate the cerebral environment (Greve & Zink, 2009).

As evident from this brief review, the neuropathology and mechanisms underlying TBI are complex and multifaceted. The sequence of events occurring after a CHI produces focal coup and contrecoup as well as diffuse injury from the impact and mechanical forces. Moreover, a cascade of secondary physiological and biomolecular interactions develops over minutes to months after the injury. Such processes all have a role in determining the symptoms and outcomes, which will be subsequently discussed.

Mechanisms underlying age as a predictor of outcome

Aside from injury severity, age is the single most important predictor of outcome after a TBI (Goleburn & Golden, 2001). Study after study, it has been consistently found that older adults sustain poorer global outcomes than younger adults (McIntyre, Mehta, Aubut, et al., 2013; McIntyre, Mehta, Janzen, Aubut, & Teasell, 2013). Such a robust finding begs the question of why do older adults sustain worse outcomes? Many potential reasons exist. Most importantly, the characteristics of the aging brain make it more vulnerable to the effects of trauma (Vollmer & Eisenberg, 1990). With normal aging processes, the brain begins to become less plastic and has less ability for repair after injury (Kinsella, 2011; Stocchetti, Paternò, Citerio, Beretta, & Colombo, 2012). There is a loss of cerebral white matter integrity (Madden, Bennett, & Song, 2009) and gray matter reduction in the brain (Raz & Rodrigue, 2006), which makes older adults more vulnerable in developing complications after a TBI. For example, the cerebral atrophy causes the veins to stretch and increases the risk of rupture and subdural hematoma (Goleburn & Golden, 2001). As previously mentioned, older TBI patients are more vulnerable to sustaining a subdural hematoma post-TBI, with 56% of those older than 65 sustaining a subdural hematoma, compared to 33% in those between 20 to 35 years old (Depreitere et al., 2012). Moreover, they are three times more likely to sustain an

intracranial hematoma compared to younger adults (Goleburn & Golden, 2001). Contusions, subdural hematomas, and intracranial hematomas all decrease the chance of a good outcome. Thus, the normal aging process makes older adults particularly vulnerable to brain complications, which contributes to their poorer outcomes.

Aside from the natural aging process contributing to their increased susceptibility, older adults on average also have more pre-injury comorbid medical conditions that may contribute to their poorer outcomes (Kinsella, 2011; Stocchetti et al., 2012). It has been found that 72% of older patients with TBI have some kind of cardiovascular disease (Stocchetti et al., 2012). Older adults are also more likely to have diabetes, hypertension, and chronic renal failure, which may also worsen outcomes (Stocchetti et al., 2012). These comorbid conditions can worsen their TBI symptoms post-injury, although it is unclear how such conditions and TBI interact to produce poorer outcomes. One mechanism could be through increased use of medication, since some drugs, such as anticoagulants, may worsen cerebral damage after trauma by affecting the cerebrovascular response to injury necessary for the recovery process (Thompson, McCormick, & Kagan, 2006). Although differences in medical comorbidity may contribute to their greater mortality and morbidity, it does not account for all of the variance, as at least one study has reported that age is still a significant predictor of poor outcome after controlling for comorbid disorders as covariates (Stocchetti et al., 2012).

Finally, older adults' worse outcomes post-TBI may be due partly to poorer rehabilitation efforts for this group. Specifically, older adults are more likely to decide to withdraw from treatment, and thus may not reap the benefits of rehabilitation like younger adults (Depreitere et al., 2012). It is unclear what the reasons are for the poorer

adherence to treatment, but it may be due to negative, predetermined beliefs that older adults are less likely to have a good outcome or recovery after injury. This expectation may prompt less aggressive treatment by hospital settings as well as influence older adults' decision to withdraw from treatment (Stocchetti et al., 2012). Furthermore, those who do remain in rehabilitation may be less successful because of their pre-trauma changes in cognition associated with normal aging and their comorbid medical conditions, which may affect their recovery (Stocchetti et al., 2012). Thus, older adults' aging brains and bodies, coupled with less intensive treatment efforts, are some reasons explaining why age is one of the strongest predictors of global outcome after TBI.

Outcomes in older adults after TBI

Compared to the extant literature on younger adults with TBI, relatively little research has been conducted on post-TBI cognitive and emotional outcomes in older adults. Research on older adults with TBI has primarily focused on mortality and global outcomes. As discussed, it has been widely established that older adults have a significantly higher mortality rate post-TBI than their younger counterparts. A recent meta-analysis on mortality in older adults sustaining TBI found that overall, 38% of cases resulted in mortality (McIntyre, Mehta, Aubut, et al., 2013). However, this depended on the severity of injury, with those sustaining severe injuries having especially poor outcomes, with reported mortality rates ranging from 65% (McIntyre, Mehta, Aubut et al., 2013) to 76% (Brazinova et al., 2010) in this population. This is substantially higher than other age groups and represents the highest mortality rates post-injury (McIntyre, Mehta, Aubut et al., 2013). In terms of global functioning, the research that has been conducted suggests that older adults have poorer functional outcomes than other age groups, although this is also dependent on injury severity. In a meta-analysis on

functional outcomes in older adults, it was found that for severe TBI, only 7.9% of older adults had favorable outcomes on the Glasgow Outcome Scale (GOS), 32.3% had favorable outcomes for moderate TBI, and 80.5% had favorable outcomes for mild TBI (McIntyre, Mehta, Janzen, et al., 2013). Hence, the prognosis for older adults sustaining moderate to severe injuries is extremely poor, although recovery is possible. Because death is frequently the outcome for older adults sustaining severe TBIs, few studies have examined the cognitive and emotional outcomes for this population. Hence, the following review of cognitive and emotional outcomes in older adults mainly focuses on TBIs of mild and moderate severity. Table 1 summarizes the characteristics and findings of the studies reviewed.

Table 1

Summary of Reviewed Research on Older Adult Post-TBI Outcomes

Study	**Participants**	**Age**	***N*[c]**	**Injury Severity**	**Time post-injury**	**Control group**	**Exclusion**	**Findings**
				Cognitive Outcomes Post-TBI				
Ashman et al. (2008)	Community volunteers, recruited through outpatient rehabilitation clinics	>55	54	Mild (63%) Moderate (37%) Based on self-reported PTA and LOC	*M* = 13 years, *SD* = 16.5 years (range: 1 – 58 years)	Healthy age-matched community-dwelling controls	Non-traumatic acquired brain injury (e.g., vascular), pre-existing neurocognitive disorder and psychotic disorder	TBI group had significantly lower performance on verbal memory and attention tests. No significant difference in language, visual memory, processing speed, and executive function.
Bay et al. (2012)	Community volunteers, recruited from outpatient rehab clinics	18-39 40-61	81 78	Mild (*N* = 68) Moderate (*N* = 91)[b]	1 – 36 months	None	History of psychosis and neurological disorders, absence of severe TBI	Older adults had more subjective memory complaints compared to younger adults.
Goldstein et al. (2001)	Recruited from acute care neurosurgery services	>50	35	Mild (*N* = 18) Moderate (N = 17)[a]	1-2 months	Community-residing, age-matched healthy controls	Substance abuse, psychiatric disorders, neurological disorders, dementia	No significant difference between mild TBI and controls. Those with moderate TBI performed worse than controls on several cognitive measures.

Study	Participants	Age	*N*[c]	Injury Severity	Time post-injury	Control group	Exclusion	Findings
Green et al. (2008)	Recruited from hospital rehabilitation program	17-79	75	GCS & PTA determined; mixed severity	2, 5, 12 months	Longitudinal Repeated measures	Orthopedic injuries, pre-injury neurological and psychiatric disorders, PTA > 6 weeks post-injury, failure on SVTs	Age moderated cognitive recovery from 2 to 12 months for simple and complex processing speed. Older adults had poorer recovery in processing speed. No age effect for other cognitive domains.
Kinsella et al. (2014)	Recruited from hospital trauma services	>65	50	Mild Complicated[b]	3 months	Orthopedic & healthy age-matched controls	Pre-injury functional dependence & co-morbidity, insufficient English	Trauma groups (TBI & orthopedic) performed worse on prospective memory and attention measures than healthy group; no difference between trauma groups
Leblanc et al. (2006)	Retrospectively reviewed from registry at hospital	18-39 40-59 60-99	971 627 684	Mild (66%), Moderate (12%), Severe (22%)	Acutely post-injury (not specified)	None	Not specified	Older adults across all severities had significantly worse self-reported cognitive functioning compared to younger and middle aged. No significant difference between younger and middle aged.

Study	Participants	Age	*N*[c]	Injury Severity	Time post-injury	Control group	Exclusion	Findings
Rapoport et al. (2006)	Recruited from hospital TBI clinic	>50	69	Mild (*N* = 37) Moderate (*N* = 32)[b]	12 months	Community-residing, demographic matched	Neurologic disease, serious acute medical illness, schizophrenia, bipolar disorder, pre-existing cognitive decline	Moderate TBI: Significantly worse than control group on processing speed, verbal memory, language, executive function, subjective cognitive complaints. Mild TBI: No significant differences compared to control group.
Rapoport et al. (2008)	Recruited from hospital TBI clinic	>50	69	Mild (*N* = 37) Moderate (*N* = 32)[b]	2 years	Community-residing, demographic matched	Neurologic disease, serious medical illness, schizophrenia, bipolar disorder, pre-existing cognitive decline, significant alcohol consumption	No significant difference on general cognitive tests, attention, processing speed, language, memory, and executive function.
Senathi-Raja et al. (2010a)	Recruited from hospital database	16-34 35-54 55-81[d]	39 39 34	GCS: Mild (20.5%) Moderate (10%) Severe (43%)	5-22 years (*M* = 11.2, *SD* = 3.7 years)	Demographic and IQ matched healthy controls	Subsequent head injury, history of neurological disease, degenerative dementia, psychiatric illness	Older adults performed worse on all cognitive domains than younger adults when compared to healthy controls. Age x time-post injury interaction also observed.

Study	Participants	Age	*N*[c]	Injury Severity	Time post-injury	Control group	Exclusion	Findings
				Emotional Outcomes Post-TBI				
Deb & Burns (2007)	Recruited from hospital database	>65 18-65	45 120	Mixed (mostly mild complicated)[a]	1 year	None	None specified	Younger adults scored higher on measures of psychiatric outcome compared to older adults. Younger adults were more likely to be diagnosed with psychiatric disorder (32 vs 16%) and depression (16 vs11%) compared to older adults, (non-significant).
Goldstein, et al. (2001)	Recruited from acute care neurosurgery services	>50	35	Mild (*N* = 18) Moderate (*N* = 18)[a]	1-2 months	Community-residing, age-matched healthy controls	Substance abuse, psychiatric disorders, neurological disorders, dementia	Mild and moderate TBI groups had greater depression and anxiety than control group.
Levin et al. (2010)	Recruited from hospital trauma center	>16	69	Mild (*N* = 60) moderate (*N* = 9)[a]	3 months	Combined TBI and general trauma patients	Penetrating brain injury, schizophrenia, mental deficiency, previous TBI substance abuse, high blood alcohol	No age differences in depressed and non-depressed groups (based on SCID).

Study	Participants	Age	*N*[c]	Injury Severity	Time post-injury	Control group	Exclusion	Findings
Levin et al. (1997) cited from Menzel (2008)	Unknown	>65	41	Mild (*N* = 14) Moderate (*N* = 27)	*M* = 34, 218, and 411 days	Community-residing age-matched controls	Not specified	Mild TBI: 21% mild/moderate levels of depression, 79% no depression Moderate TBI: 6% mild/moderate depression, 11% severe levels depression, 63% no depression.
Rapoport & Feinstein (2001)	Recruited from hospital TBI clinic	>60 18-59	26 30	Mild[b]	1 month	None	History of focal brain disease, serious acute medical illness, major psychiatric illness	Older adults had higher scores on GOS and endorsed less psychological distress. Not significant when controlled for current employment.
Rapoport et al. (2003a)	Recruited from hospital TBI clinic	19-59 >60	146 64	Mild[b]	*M* = 49 days	None	Pre-injury history of brain disease, serious acute medical illness, schizophrenia, bipolar disorder, dementia, substance abuse	Older adults had lower rates of depression (assessed with SCID) than younger adults after accounting for history of pre-injury major depression, MMSE, and time post-injury.

Study	Participants	Age	*N*[c]	Injury Severity	Time post-injury	Control group	Exclusion	Findings
Rapoport, et al. (2003b)	Recruited from hospital TBI clinic	15-91	170	Mild[b]	*M* = 48 days *SD* = 34 days Range: 1-227 days	None	History of focal brain disease, serious medical illness, dementia, schizophrenia, bipolar disorder, major depression	15% of sample had major depression disorder based on SCID No significant difference in age between those with post-TBI major depression disorder and those without .
Rapoport et al. (2006)	Recruited from hospital TBI clinic	>50	69	Mild (*N* = 37) Moderate (*N* = 32)[b]	12 months	Community-residing, demographic matched	Neurologic disease, serious medical illness, schizophrenia, bipolar disorder, pre-existing cognitive decline	TBI group had worse self-reported distress, anxiety, depression and higher rates of meeting diagnosis for depression than control group.
Senathi-Raja et al. (2010b)	Recruited from hospital database	16-27 28-39 40-51 52-63 64-81[d]	29 19 23 20 21	Mixed (mostly moderate-severe)[b]	5-22 years	Healthy demographic matched controls	Subsequent head injury, neurologic disease, degenerative dementia, psychiatric illness	No significant age effect on HADS score. Age x time post-injury interaction was observed.
Whelan-Goodinson et al. (2010)	Recruited from hospital database	19-74[e]	100	GCS: Mild: 35%, moderate: 20%, severe: 45%	0.5 – 5.5 years (*M* = 3, *SD* = 1.5)	None	Previous history of TBI, serious neurological disorder	Age significantly related to post-TBI anxiety symptoms. Those 50-60 years old had highest frequency of anxiety disorders, with reduced frequency at other ages.

Study	Participants	Age	*N*[c]	Injury Severity	Time post-injury	Control group	Exclusion	Findings
Relation Between Post-TBI Cognitive & Emotional Outcomes								
Chamelian & Feinstein (2006)	Recruited from outpatient TBI clinic	18-60	63	Mild Moderate	6 months	None	Not specified	Those who had self-reported cognitive complaints had higher depression rates than those who did not.
Chaytor et al. (2007)	Originally recruited for a drug study (hospital setting)	14-87	216	Moderate-severe*	6 months	None	Age <14, drug exclusions, pregnant, prisoners, or live overseas	Significant but weak relation between depression and cognitive performance on several tests.
Deb & Burns (2007)	Recruited from hospital database, prospective	>65 18-65	45 120	Mixed (mostly mild complicated)[a]	1 year	None	None specified	MMSE scores related to ICD-10 diagnosis rates for younger but not older adults.
Himanen et al. (2009)	Recruited from hospital database	43-83	61	PTA determined; Mixed severity	27-48 years	Healthy demographic matched controls	Prior brain disease, no available medical records	No difference in attention between depressed and non-depressed patients in the long-term.
Rapoport et al. (2005)	Recruited from hospital TBI clinic, prospective	18-64	74	Mild Moderate	6 months	None	History of focal brain disease, serious medical illness, dementia, schizophrenia, bipolar disorder	Those with major depression had lower scores on working memory, processing speed, and verbal memory than those with no depression.

Study	Participants	Age	*N*[c]	Injury Severity	Time post-injury	Control group	Exclusion	Findings
Rohling et al. (2001)	Compensation-related evaluations, recruitment method unclear	*M* = 42.4, *SD* = 11	420	Mixed diagnoses including TBI	Unspecified	None	Suboptimal performance	No significant difference between depressed and non-depressed groups on cognitive performance. Significant difference found between depression groups on subjective cognitive complaints.
Satz et al. (1998)	Recruited from hospital neurotrama units	16-7777[e]	100	Moderate-Severe[a]	6 months	Other injury control group matched for age and gender	Non-fluency in English or Spanish, history of medial illness, neurological disease, pre-existing cognitive decline	No significant association between cognitive performance and depression. Self-reported depression and subjective cognitive complaints was significantly related.

Note. All studies with healthy controls are at least age-matched. Unless noted, all studies examining an age effect utilize a cross-sectional design. Unless noted, studies do not explicitly specify if age refers to age at time of injury or age at time of assessment. Demographic matched = Age, education, gender matched.

[a]Injury severity based on GCS and imaging; [b]Injury severity based on GCS, post-traumatic amnesia, and length of loss of consciousness; [c]Number of participants in the TBI group; [d]Age at time of injury; [e]Age at time of assessment

Cognitive outcomes. Cognitive functioning in older adults after TBI largely depends on both the severity of the injury and time post-injury. There is evidence that older adults sustaining a TBI of mild complicated to moderate severity perform worse on tests of attention, processing speed, memory, executive functioning, and language than healthy age-matched controls 2 months post-injury (Goldstein, Levin, Goldman, Clark, & Altonen, 2001) and 12 months post-injury (Rapoport, Herrmann, Shammi, Kiss, Phillips, & Feinstein, 2006). However, the literature suggests no differences in these domains exist at 2 years post-injury, implying that older adults sustaining moderate TBI and healthy older adults performed similarly in the long-term (Rapoport et al., 2008). A recent study on the cognitive outcomes after mild complicated TBI in older adults suggested that these post-injury differences in performance between older adults sustaining TBI and healthy controls may not be due to the actual brain injury, but reflect the effects of general trauma (Kinsella, Olver, Ong, Gruen & Hammersley, 2014). Specifically, Kinsella et al. (2014) reported that older adults sustaining a mild complicated TBI at 3 months post-injury performed worse on measures of prospective memory and attention than healthy age-matched controls, but no difference in performance was found between the TBI and orthopedic injury groups. Because none of the other studies included an orthopedic control group, it is unclear whether the differences found in those studies are indeed a result of the TBI or reflect general trauma effects. Whatever the underlying explanation, it appears that older adults sustaining a mild complicated to moderate TBI have poorer performance on neuropsychological tests than healthy older adults before 1 year post-injury (Goldstein et al., 2001; Rapoport et al., 2006) but perform similarly by 2 years post-injury (Rapoport et al., 2008).

Although much research exists comparing older adults with TBI with healthy older adults, little research has directly compared the neuropsychological performance of younger and older adults with mild complicated to severe TBI while taking into account the performance of healthy age-matched controls. In a cross-sectional study by Senathi-Raja, Ponsford, and Schönberger (2010a), older adults with TBI of mixed severity examined 5 to 22 years post-injury performed worse on all cognitive domains tested (processing speed, attention, verbal and visual memory, working memory, executive function) than younger adults when compared to age-matched controls. Senathi-Raja et al. (2010a) also found an unexpected age X time post-injury interaction. Specifically, there was a positive association between time post-injury and cognitive performance for the older age group (55 years or older), no association for the middle-aged group (35-54 years), and a negative association for the younger age group (16-34 years). Hence, it appears that for older adults, cognitive performance may be less impaired relative to age-matched healthy controls when those adults are assessed at a later time after injury compared to sooner after injury. While this finding might imply that older adults ultimately may recover to the same level as younger adults over time, the authors suggested that their finding may be due to selective enrollment of higher functioning TBI survivors in participants over 55 years old. This explanation was based on the observation that a higher percentage of potential participants in the older group either were deceased or refused participation when recruited for the study. Thus, there may be some sampling bias affecting the results.

One other study (Green et al., 2008) examined the effects of age on cognitive recovery using a longitudinal design. Such a study has an advantage over Senathi-Raja et

al. (2010a) in that cognitive functioning can be observed in individuals of different ages over time instead of examining time post-injury and age only at one point in time. Using a sample of participants with mild complicated to severe TBI, Green et al. (2008) found that age was related to the trajectory of recovery when measured at 2 to 12 months post-injury. Specifically, it was found that age moderated cognitive recovery such that older adults tended to make less recovery gains during this time span than younger adults. However, this was only found for tests of simple and complex processing speed, and was not found for tests of memory, attention, and executive function. While no healthy age-matched comparison group was included in the study, Green et al. (2008) created summary neuropsychological test scores adjusted for age-related changes in cognition. Although these results diverge from those of Senathi-Raja et al. (2010a), who found age differences across various cognitive domains post-TBI, these two studies differ in many other ways, including the time post-injury measured (e.g., Green et al. and Senathi-Raja et al. examined time post-injuries less than 12 months and greater than 5 years, respectively), and type of design (longitudinal versus cross-sectional). Thus, it is possible that age has a differential influence on cognitive functioning depending on time post-injury. Combining the findings of these two important studies, the effect of age on performance in various cognitive domains may be most pronounced at longer times post-injury (e.g., 5 to 10 years post-injury; Senathi-Raja et al. 2010a), but may have little influence on the post-TBI recovery trajectory acutely post-injury (e.g., before 12 months post-injury; Green et al., 2008).

In contrast to the literature on moderate TBI, research examining cognitive outcomes after mild uncomplicated TBI suggests that outcomes are similar (a) between

younger and older adults with mild uncomplicated TBI and (b) between older adults with mild uncomplicated TBI and healthy age-matched controls. That is, on average both younger and older adults seen 2 to 3 months or longer after a mild uncomplicated TBI show no significant differences in cognitive functioning from healthy age-matched controls across cognitive domains, including language, visual memory, processing speed, and executive functioning domains (Ashman et al., 2008; Goldstein, Levin, Goldman, et al., 2001; Rapoport et al., 2006; Rapoport et al., 2008). Such similarities were already found at one to two months post-injury (Goldstein & Levin, 2001), suggesting that no differences in cognitive functioning exist between older adults after TBI and age-matched controls from early in recovery. Similarly, it appears there is no difference in cognitive functioning between older adults who have sustained mild TBI and healthy older adults at one (Rapoport et al., 2006) and two years post-injury (Rapoport et al., 2008). This is similar to overall findings with mild uncomplicated TBI in the general population in that there is usually no neuropsychological impairment by 3 months post-injury (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005). Overall, the majority of studies on mild uncomplicated TBI suggest that few if any differences in cognitive sequelae exist between older adults and younger adults with mild uncomplicated TBI or between older adults with mild uncomplicated TBI and healthy age-matched controls at both short (i.e., one to two months; Goldstein & Levin, 2001) and long-term (i.e., one to two years; Rapoport et al., 2006; Rapoport et al., 2008) study points post-injury.

Despite similar *performance* relative to healthy age-matched controls on objective neuropsychological tests by 1 to 3 months after mild uncomplicated TBI, there is evidence that older adults *report* poorer cognitive functioning than younger adults after a

TBI regardless of severity and time after injury. In a large scale study, older adults reported poorer cognitive functioning on the Functional Independence Measure compared to younger adults across all injury severities (LeBlanc, De Guise, Gosselin, & Feyz, 2006). Likewise, there was a greater tendency for older adults to report more subjective memory complaints than younger adults 3 years post-injury (Bay, Kalpakjian, & Giordani, 2012). These findings are congruent with studies finding an age effect on metamemory in a healthy, non-TBI population, such that healthy older adults have greater subjective global concern about their memory than healthy younger adults (Dobbs & Rule, 1987). Hence, greater subjective memory concern is not unique to a TBI population of older adults, but appears to be the norm in the general healthy older population. Nevertheless, the findings of LeBlanc et al. (2006) and Bay et al. (2012) suggest that despite similar performance relative to age-matched controls on objective testing between older and younger adults after mild to moderate TBI 2-years post-injury, older adults are more likely to perceive themselves to have poorer cognitive functioning than younger adults.

Emotional outcomes. Emotional outcomes (i.e., depressive and anxiety symptoms) after TBI in older adults have been far less investigated than cognitive outcomes, with the majority of research in this area being conducted in the past decade. A recent literature review only found one study on depression and TBI in those over 65 years old (Menzel, 2008). There are also a few studies examining post-TBI depression in those under 65 years old but classified as older adults as well as a couple of studies conducted since the review. Despite the sparse literature, the extant research has suggested that while older adults sustaining TBI have more emotional symptoms than

healthy older adults, older adults have comparable, if not better, emotional outcomes than younger adults post-TBI, especially when assessed at longer times post-injury.

For healthy older adults in the community, the rate of depression ranges from 2 to 9%, although this is higher in acute and long-term care settings (10 to 25%; Menzel, 2008). Levin, Goldstein, and MacKenzie (1997) found that for older adults sustaining a mild TBI, 21% had mild to moderate levels of depression and 79% had no depression around 1 month post-injury. In terms of TBI of moderate severity, Levin et al. (1997) found that 6% of older adults had mild to moderate levels of depression, 11% had severe levels of depression, and 63% had no depressive symptoms in the short term. In addition, Deb and Burns (2007) reported that 11% of their older adult sample sustaining TBI of mixed severity was diagnosed with depression after one year. Hence, these results suggest that the prevalence of depression is higher in older adults sustaining TBI compared to community residing older adults, which parallels the findings of younger adults with and without TBI (Deb, Lyons, Koutzoukis, Ali, & McCarthy, 1999). These results are found at various time points post-injury. Specifically, it has been found that older adults sustaining TBI report more psychological distress, anxiety, and depression and meet diagnostic criteria for depression at higher rates than healthy older controls at both 2 months post-injury (Goldstein et al., 2001) and 6 to 12 months post-injury (Rapoport et al., 2006).

In terms of studies examining an age effect, it has typically been found that, surprisingly, older adults who sustain a TBI do not have worse emotional outcomes than younger adults with TBIs. In the general TBI population, the rates of depression range from 10 to 42% within the first 2 years of injury (Menzel, 2008). For older adults with

mild to moderate TBI, 24% had mild to moderate levels of depression and 7% had severe depression at approximately one month post-injury (Levin et al., 1997). This prevalence decreases approximately 7 months and one year later. Thus, the rates of depression after TBI appear to be similar for older adults and the general TBI population. Other studies using age as a continuous variable also have shown no differences in age between depressed and non-depressed groups post-TBI (Levin et al., 2001; Jorge et al., 2004; Rapoport, McCullagh, Streiner, & Feinstein, 2003b).

For studies directly comparing the rates of depression and psychiatric disorder between younger and older adults post-TBI, the overall consensus is that older adults actually have better emotional functioning than younger adults. At one year post-injury, Deb and Burns (2007) found no significant difference in depression and psychiatric disorder rates between the two groups. However, there was a trend toward poorer scores in younger adults than older adults on 3 different measures of emotional functioning and younger adults were more likely to be diagnosed with a psychiatric disorder (32% versus 16%) and depression (16 % versus 11%) with the ICD-10 than older adults. This is consistent with Rapoport, McCullagh and Streiner (2003a), who also directly compared prevalence rates of depression diagnosed with the Structured Clinical Interview for DSM-IV (SCID) in older and younger adults and found a lower rate of depression in older adults. Furthermore, Rapoport and Feinstein (2001) found that older adults in the acute phase after TBI reported less psychological distress when compared to younger adults with acute TBI. Thus, it appears that older adults sustaining TBI have a lower incidence of depression and better emotional functioning overall than their younger counterparts.

The results of the above studies (Deb & Burns, 2007; Rapoport & Feinstein, 2001; Rapoport et al., 2003a) cannot be assumed to be related to the effects of age at injury after a TBI because normal age-related differences in cognition were not controlled for using healthy age-matched comparison groups. However, at least one study with such comparison groups suggests that age at injury and time post-injury jointly determine the impact of TBI on emotional functioning. Senathi-Raja, Ponsford, & Schönberger (2010b) found that overall, there was no significant difference between age groups on the Hospital Anxiety and Depression Scale (HADS) for those sustaining a TBI compared to the healthy demographic matched control group. In addition, a significant three-way interaction was observed between age, time post-injury at assessment, and membership in the TBI versus healthy comparison group. Specifically, younger adults sustaining TBI in the 5 to 13 years post-injury group had less emotional distress than those in the 14 to 22 years post-injury group after the scores of the healthy control group was taken into account. In contrast, older adults sustaining TBI in the 5 to 13 years post-injury group had more emotional distress than those in the 14 to 22 years post-injury group after accounting for scores from the healthy control group.

These findings suggest that older adults may actually have better emotional functioning when assessed at a later time post-injury, while younger adults have increasingly worse emotional functioning longer after their injury, and is consistent with the findings of Deb and Burns (2007), Rapoport and Feinstein (2001), and Rapoport et al. (2003a) that younger adults scored worse on psychiatric measures and were more likely to receive psychiatric and depression diagnoses. Moreover, findings that older adults report less psychological distress and symptomology and lower rates of depression than

younger adults are found both shortly after TBI at one to two months post-injury (Rapoport & Feinstein, 2001; Rapoport et al., 2003a) and long-term at 1 year (Deb & Burns, 2007) and after 5 years post-injury (Senathi-Raja et al., 2010b). These results are found for both TBI of mild (Rapoport et al., 2003a; Deb & Burns, 2007) and moderate severity (Senathi-Raja et al., 2010b).

In terms of anxiety disorders, the extant literature suggests that while community-residing older adults have a lower rate of anxiety disorders than their younger counterparts, anxiety symptoms are still very common in this population (Flint, 1994; Henderson, Jorm, Korten, Jacomb, Christensen, & Rodgers, 1998; Wolitzky-Taylor, Castriotta, Lenze, Stanley, & Craske, 2010).The prevalence rate of anxiety disorders in community-residing older adults range from a 6-month prevalence rate of 10.2% (Beekman et al., 1998) to a 12 month prevalence of 11.6% (Byers, Yaffe, Covinsky, Friedman, & Bruce, 2010) . Generalized anxiety disorder and phobias account for most of the anxiety disorders in older adults (Beekman et al., 1998; Byers et al. 2010), and are highly comorbid with depression in this population (Lenze et al., 2000). These rates of anxiety disorders are lower compared to those of younger adults and the general population in the community (Henderson et al., 1998). In addition, within the population of older adults (>55 years old), there is a decline of anxiety disorders with age (Byers et al., 2010). However, older adults tend to have high rates of subclinical anxiety disorders, with subclinical rates reported to be around 18.5% in older adults (Heun, Papassotiropoulos, & Ptok, 2000). Thus, when one takes into account their rate of both subclinical and clinical levels of anxiety, anxiety symptoms appear to be very common amongst older adults.

Little research on post-TBI anxiety disorders in older adults has been investigated. However, a recent study examined the prevalence of post-TBI anxiety disorders in older and younger adults and found that older adults were more likely to have anxiety disorders than younger adults sustaining TBI (Whelan-Goodinson, Ponsford, Schonberger, & Johnston, 2010). Although this may appear to contradict previous studies on post-TBI depression and age (e.g., Deb & Burns, 2007; Rapoport et al., 2003a), the authors stated that their findings are not necessarily inconsistent with previous findings because of differences in how age groups were defined. Specifically, Whelan-Goodinson et al. (2010) found that those between 50 to 60 years old had the highest rates of anxiety disorders. This age range was defined as an older adult group in this study, but was part of the younger adult group in studies by Deb and Burns (2007) and Rapoport et al. (2003a) , who defined their older adult age groups as above 65 and above 60 years old, respectively. Consequently, the peak age for incidence of anxiety (50 to 60 years old) found in the study by Whelan-Goodinson et al. (2010) was included in the younger adult group in other studies.

The study by Whelan-Goodinson et al. (2010) raises an important consideration for the relation between age and emotional outcomes. Their finding that the peak incidence of anxiety disorders after TBI occurred in ages 50 to 60 with those younger and older than this age range endorsing fewer anxiety symptoms suggests that the relation may be a curvilinear one. A nonlinear (quadratic) relationship was also found between anxiety and depression scale scores and participants' ages in the study by Senathi-Raja et al. (2010b), although this relation was not significant, as described above. Because the studies by Deb and Burns (2007) and Rapoport et al. (2003a) clustered participants into

only two age groups, this potentially masked the relationship of age on depression and anxiety rates. Analyzing the data with two groups will only allow for detection of a linear relation (i.e., two points can only make a straight line). For studies examining age as a continuous variable (e.g., Levin et al., 2010), the true relationship between age and outcome also may be masked if the statistical method assumes a linear relationship (e.g., correlation or linear regression analyses). As Whelan-Goodinson et al. (2010) and Senathi-Raja et al. (2010b) found, the relation between age and outcome may not be linear and such a relation should not be automatically assumed.

Overall, the extant literature suggests that while older adults sustaining mild and moderate TBI do experience more depressive and anxiety symptoms than healthy older adults residing in the community, emotional functioning appears to be similar or even better in older adults than younger adults after mild and moderate TBI, especially with greater time post-injury. However, this may depend on how age groups are defined and the possibility of a non-linear relation between age and emotional outcomes should be further explored.

One limitation of the cognitive and emotional outcome studies reviewed is the lack of specificity of whether "age" refers to age at time of injury or age at time of assessment. In all but a few studies (e.g., Satz et al., 1998; Senathi-Raja et al., 2010a; Senathi-Raja et al., 2010b; Whelan-Goodinson et al., 2010), such pivotal information is unreported and thus, how participants are grouped into age groups becomes unclear. It may be assumed that for many of the studies, participants are grouped according to age at the time of assessment. However, it may well be that age at time of injury is being used and such an assumption of one or the other cannot be definitively made without further

specification. This distinction is important as it addresses two different research questions. One is comparing outcomes for individuals of different ages at the time when they sustained the TBI although all participants may be the same age at the time of assessment. The other is comparing outcomes for individuals who might have been the same age at the time of injury, but are different ages (because of variability in time post-injury) when assessed for the study. This issue is particularly concerning in studies examining a longer time post-injury (e.g., Ashman et al., 2008; Senathi-Raja et al., 2010b), as it means that there is a greater discrepancy between the age at time of injury and age at time of assessment. For example, a 55-year old at the time of assessment whose injury occurred 10 years ago and a 55-year old at the time of assessment at 2 years post-injury would have been 45 and 53 years old, respectively, at the time of injury. Thus, age at injury and age at assessment are clearly not equivalent, and in the current study, these two participants would have been categorized into separate age groups. Similarly, an 18-year old at the time of injury is not equivalent to an 18-year old at the time of assessment, who may have been an adolescent or child at the time of the TBI and experienced different factors on the developing brain. Hence, without further specification of the term "age", it is unclear in many of these outcome studies whether they are examining age at the time of injury or assessment and the two cannot be seen as equivalent. The current study is interested in age at time of injury, as the important question to examine is how the aging brain at the time of injury adapts and recovers.

Cognitive and emotional outcomes. Little research has been conducted on the relation between cognitive and emotional outcomes after TBI and the extant literature is inconclusive on this topic. Although an association between depression and

neuropsychological performance has been found in other populations, such as stroke, cancer, and renal disease, studies with TBI have led to divergent findings (Satz et al., 1998). Some studies have found an association between performance on neuropsychological measures and depression (e.g., Chaytor, Temkin, Machamer, & Dikmen, 2007; Jorge et al., 2004; Rapoport, McCullagh, Shammi, & Feinstein, 2005), whereas others found no relation between the two (e.g., Rohling, Green, Allen, & Iverson, 2002; Satz et al., 1998). Rapoport et al. (2005) found that in a sample of mild to moderate TBI patients under the age of 65 at the time of assessment, those diagnosed with depression had significantly lower scores on working memory, processing speed, and verbal memory measures, and more perseverative responses compared to those with no depression. Jorge et al. (2004) and Chaytor et al. (2007) also found that TBI patients who were depressed had significantly poorer performance on various neuropsychological tests, including areas of memory and executive functioning. The magnitude of this relation has been found to vary, and effect sizes are as large as $d = 0.87$ and 0.82 for some measures (Jorge et al., 2004) and as small as $r = .16$ to .22 for others (Chaytor et al., 2007). In contrast to this research, other studies have found no association between performance on neuropsychological tests and depression in TBI patients, regardless of whether self-report measures (e.g., BDI; Rohling et al., 2001; Himanen et al., 2009) or an interview tool (e.g., the SCID; Satz et al., 1998) was used.

Despite inconsistent findings regarding the relation between performance on *objective* neuropsychological tests and depression, it appears that *subjective* cognitive complaints are consistently associated with emotional outcomes. For example, Satz et al. (1998) examined the association between functional, emotional, and cognitive outcomes

in moderate to severe TBI at 6 months post-injury and found no significant relation between neuropsychological performance and depression after controlling for the effects of general trauma with an orthopedic control group. However, there was an association between subjective cognitive and memory complaints and self-reported depressive symptoms. In other words, Satz et al. (1998) found that TBI participants who complained of poor mood also complained of cognitive difficulties when compared to the control group, suggesting that there is a relation between subjective but not objective cognitive functioning and depressive symptoms. These results also have been found in compensation-related settings with a mixed sample of participants that included individuals with TBI (Rohling et al., 2001) as well as more recent TBI studies (e.g., Chamelian & Feinstein, 2006).

As illustrated here, although studies have been consistent on the relation between subjective cognitive complaints and depression, there are divergent findings in the literature on the relation between emotional outcomes and objective cognitive performance post-TBI. These differences are probably not accounted for by differences in time post-injury, as studies with divergent results have looked at similar time points. For example, Satz et al. (1998) and Rapoport et al. (2005) both studied outcomes at 6 months post-injury but had divergent results. Furthermore, these incongruent findings are likely not accounted for by differences in the method of assessing depression. Satz et al. (1998) and Rapoport et al. (2005) both used the SCID to measure depression rates but had divergent findings. Likewise, Himanen et al. (2009) and Chaytor et al. (2007) both used self-report measures of depression (e.g., BDI-II) but resulted in divergent findings.

However, these studies did not examine the effects of age at time of injury on the relation between cognitive and emotional outcomes, which may be one of the reasons for the discrepancies in the literature. It is possible, for example, that the insignificant relation between objective cognitive performance and self-reported depression in the study by Satz et al. (1998) is masked by the moderation of age such that the association between emotional and cognitive outcomes differs depending on age at injury. Rapoport et al. (2005) and Chamelian and Feinstein (2006) both used younger samples (younger than 65 and younger than 60 years old at time of assessment, respectively) and found significant associations between cognitive and emotional outcomes. Other studies used a mix of ages, although it appears that the average age at the time of assessment was quite young in most of these studies (e.g., the sample in the study by Chaytor et al. had a mean age of 29.5) and most studies did not examine the effect of age at time of injury on this relationship. Hence, most studies that found a relation between cognitive and emotional outcomes focused on adults who were young or middle aged at time of the assessment.

To date, there has only been one study to my knowledge that also examined the effects of age on the relation between post-TBI cognitive and emotional functioning, although it is unclear whether the age reported was age at time of injury or time of assessment. At one year post-injury, Deb and Burns (2007) found that cognitive functioning was related to psychiatric outcome in younger adults but not older adults. Specifically, scores from the Mini-Mental State Exam (MMSE) were significantly related to rates of diagnosis of depression in younger adults, but this relationship was not found in older adults at one year post-injury. However, their study was limited as they only assessed cognitive performance using the MMSE and did not assess participants on a

broader range of neuropsychological domains. The MMSE is a short measure primarily used for dementia screening and assesses a restricted set of cognitive abilities (Lezak et al., 2012). It is not equivalent to a comprehensive neuropsychological assessment and cannot provide details on patients' performance on various domains of cognitive functioning (e.g., memory, attention, executive functioning). Furthermore, recent research has suggested that the MMSE has low sensitivity to cognitive impairment and low predictive value and utility in older adults with TBI (Srivastava, Rapoport, Leach, Phillips, Shammi, & Feinstein, 2006).

In addition to this limitation, Deb and Burns (2007)'s study used the ICD-10 to diagnose depression, which may underestimate those with subclinical levels of depressive symptoms. As discussed, research has suggested that depression manifests differently in older compared to younger adults such that subclinical levels of depression may be more common in older adults, with rates for subsyndromal depressions ranging from 13% to 27% in community-dwelling older adults (Heun et al., 2000; Lebowitz et al., 1997). Thus, older adults may not qualify for a diagnosis for major depressive disorder based on the ICD-10 but may still have significant depressive symptoms that interfere with daily functioning. Further limitations of this study include the fact that their sample had significantly more males in the younger group, which was not controlled for and may have affected the results. As several studies have shown, gender is related to post-TBI cognitive and emotional outcomes (e.g., Lecours et al., 2012; Liossi & Wood, 2009). Lastly, psychiatric history was not accounted for in this study and there were no healthy age-matched comparison groups to allow one to take into account differences in manifestation of depression in younger versus older adults. Hence, while Deb and Burns

(2007) did find that the relation between cognitive and emotional outcomes differed for older and younger adults, their study was limited by their methodology in measuring cognitive and emotional functioning as well as various confounding variables that were not taken into account.

III. HYPOTHESES AND RATIONALE FOR THE CURRENT STUDY

The present study had two broad objectives. The first objective of this research was to describe post-TBI cognitive and emotional functioning across the adult lifespan and examine whether these outcomes differ between adults who were older (≥50 years) and younger (≤49 years) at time of the TBI. As discussed, one of the limitations of past outcome studies is that age at time of injury was not used or specified. In the current study, age at time of injury was used, and the term "age" refers to age at the time of injury unless otherwise specified.

As discussed, some studies have suggested that the relation between age and cognitive and emotional outcomes may not be a linear one. A nonlinear relation may potentially explain some of the discrepancy among studies and hence was further explored in this study. To my knowledge, this study was the first to specifically examine the form of the relationship between performance on neuropsychological tests and age, anxiety symptoms and age, and depression symptoms with age. Furthermore, while depression has been studied as an outcome in older adults, sparse research has been conducted on post-TBI anxiety symptoms in this population. This research contributes to our current knowledge as it is one of the few studies focusing on post-TBI anxiety symptoms in older adults.

The second objective of the present study was to examine whether age moderates the relationship between post-TBI cognitive performance and emotional symptoms. In other words, is there a difference in the relation between cognitive functioning and self-reported emotional functioning after TBI as a function of age? As discussed, only one study to date has examined the relation between cognitive and emotional outcome at

various points after TBI as a function of age (Deb & Burns, 2007), but is methodologically limited in several ways.

In correspondence with the objectives of this study, three research questions were investigated:

1. What kind of relation does age at time of injury have with cognitive functioning post-TBI after accounting for injury severity and time post-injury? While previous studies (e.g., Senathi-Raja et al., 2010a) have examined the relation between age and cognitive functioning post-TBI, no research has specifically investigated the form of this relationship. Cognitive functioning here, as discussed, is defined as overall performance on neuropsychological testing as well as performance on specific cognitive domains that have been shown to be most affected post-TBI (e.g., memory, executive functioning, and processing speed). As mentioned, the majority of studies divide participants into two age groups (i.e., older and younger), thus assuming a linear relationship. Of the studies reviewed, only Senathi-Raja et al. (2010a) used more than two age groups in comparing cognitive outcomes. They found no evidence of a nonlinear relationship between age and cognitive functioning in their sample. However, this relationship was examined at 5 to 22 years post-injury, whereas the current study examined the relation between cognitive functioning and age in the interval from 3 months to 13 years post-injury. Hence, to my knowledge, the form of the relationship between cognitive outcome and age at less than 13 years post-injury has not been previously investigated, and it is possible that a nonlinear relation exists between post-TBI cognitive performance and age at different times post-injury. For example, the relationship could be quadratic, which would be evident from one "bend" in the regression line. Without sufficient literature, it is unclear

what form this relationship may take, and neither a linear or nonlinear relationship can be assumed without further exploration of this topic.

2. What kind of relation does age at time of injury have with emotional functioning post-TBI after accounting for injury severity and time post-injury? To investigate the relation between age and emotional functioning, it was hypothesized that there will be a nonlinear (quadratic) relationship between self-reported depression and anxiety symptoms and age after controlling for injury severity and time post-injury. Specifically, it was predicted that there will be an inverted-U relationship in which middle-aged adults have the worst emotional outcomes and younger and older adults have better outcomes post-TBI. This would be consistent with previous studies that found that those between 50 to 60 years old (Whelan-Goodinson et al., 2010) and 28 to 51 years old (Senathi-Raja et al., 2010b) had the highest levels of anxiety and depression after TBI. Such a relation may be due to the possibility that older adults have fewer responsibilities (e.g., employment and caregiving) to return to after the TBI than middle-aged adults and may have developed better coping strategies to deal with the effects of the injury. Indeed, research has supported the idea of developmental changes in coping styles and life responsibilities, with older adults (>65 years old) reporting experiencing fewer hassles in life and using more passive, intrapersonal coping styles (e.g., positive appraisal, acceptance of responsibility) than adults between the ages of 35 to 45 years old (Folkman, Lazarus, Pimley, & Novacek, 1987). Furthermore, it could be that older adults have less insight into their difficulties caused by the TBI (Deb & Burns, 2007), and hence may not notice the changes in their cognition, which might otherwise cause emotional symptoms.

3. Does age at time of injury moderate the relation between post-TBI cognitive and emotional outcomes after accounting for injury severity and time post-injury? Finally, it was hypothesized that there would be an interaction effect such that there would be a significant relationship between cognitive and emotional outcome for adults who were younger at time of injury but no relationship for adults who were older at time of injury after controlling for severity and time post-injury (Figure 1). Specifically, it was predicted that younger adults with TBI who showed worse cognitive outcomes would also have worse emotional outcomes, but no relation would emerge for older adults when time since injury and injury severity are comparable across age groups. Given that past research has suggested that those who are older at time of injury are more likely to have worse post-TBI cognitive outcomes than those who are younger at time of injury but comparable or lower rates of depression and anxiety when compared to healthy controls, it would be rational to hypothesize that the relation between cognitive and emotional outcomes would be different depending on age. As mentioned, such a relation has only been reported in one study (Deb & Burns, 2007), but was limited by both the measures used and uncontrolled confounding variables. The present study not only addressed these limitations, but was also the first to examine the relation between post-TBI anxiety symptoms and cognitive outcomes as a function of age.

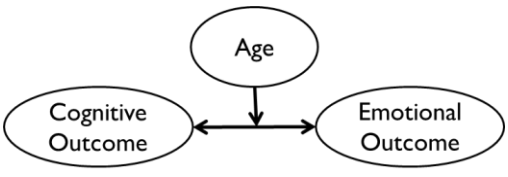

Figure 1: Hypothesized relation between age at time of injury, cognitive outcome and emotional outcome post-TBI.

IV. METHODOLOGY

Participants

This study consisted of data from individuals in an archival database of a board certified registered clinical neuropsychologist. These individuals originally were referred for a neuropsychological assessment in a private practice for compensation-related reasons and sustained a head injury by way of a motor vehicle accident. Because of the litigation context in which the evaluations took place, the effects of litigation on test performance must be considered. Much research has supported the idea that those with mild TBI in litigation are more likely to continue having cognitive symptoms three months post-injury and experience worsening of cognitive performance over time, even after taking into account test invalidity and malingering on symptom validity testing (Belanger et al., 2005). This is in contrast with individuals sustaining mild TBI who are not in litigation, in which most cognitive symptoms resolve by 3 months post-injury (Belanger et al., 2005). However, this does not appear to be the case for more severe TBIs, as research has suggested that there is no difference between litigating and non-litigating individuals with severe TBI at both early (4 months) and later (10 years) time points post-injury (Wood & Rutterford, 2006). As the participants in this study consisted of those with mild complicated to severe TBI, it is unlikely that the litigation context played a great role in their neuropsychological performance, but this context should still be considered when interpreting the results.

Participants over the age of 18 were included in the study if they sustained a closed-head TBI of mild complicated to severe severity. TBI severity was determined with the Glasgow Coma Scale (GCS) and neuroimaging findings. As mentioned, the GCS is a brief scale used to measure levels of consciousness on 3 aspects of behavior (motor,

verbal, and eye opening), and provides a score between 3 and 15 (Teasdale & Jennett, 1974). It has evolved to serve as a common classification tool for TBI severity. A score between 3 to 8 is indicative of severe, 9 to 12 moderate, and 13 to 15 mild TBI (Lezak et al., 2012). This widely accepted classification based on the GCS was the primary method used to define TBI severity in the present study. As discussed, mild complicated TBI was grouped with moderate TBI for this study as it has been shown in the literature that cognitive and functional outcomes and recovery after mild complicated TBI are similar to TBI of moderate severity rather than mild uncomplicated (Borgaro et al., 2003; Kashluba et al., 2008; Williams et al., 1990). Thus, this study categorized participants into one of two severity groups: mild complicated-moderate (GCS scores ranging from 9 to 12 or 13 to 15 plus evidence of intracranial abnormalities on neuroimaging) or severe (GCS scores ranging from 3 to 8).

Despite its widespread usage, determining severity based solely on the GCS has its limitations. GCS scores are affected by factors such as alcohol intoxication at the time of injury and medically induced coma at the time of the rating (Lezak et al., 2012). Furthermore, there is some debate as to which time point of GCS measurement is best to determine severity. After injury, patients' level of consciousness may fluctuate over time, with their ability to respond to commands and spontaneously open their eyes impaired initially, for example, with more alertness several minutes later. Hence, it is unsurprising that the GCS score may fluctuate with the point in time at which it is measured, underlining the important issue of considering the context and time of administration of the GCS (Lezak et al., 2012). Often, GCS scores are continually measured by medical personnel. Each GCS assessment offers a "snapshot" of the patient's consciousness level

at that moment (Lezak et al., 2012). In the literature, GCS at various time points has been used for research purposes. For example, the emergency GCS taken at the scene or ambulance, GCS at admission to the hospital, Best Day-1 GCS, and Worst Day-1 GCS have all been used in research. For the purposes of the current study, the GCS measured initially by EMS at the scene was primarily used as a measure of injury severity. GCS scores at the scene were obtained through medical charts of the participant. In cases where such information is not available, the lowest GCS score available was used to determine severity.

Because it was found that many participants did not have readily available GCS scores, findings on neuroimaging (beyond just determining presence or absence of intracranial abnormality for mild complicated TBI) were also used to classify severity. Previous research has shown that different types of lesions observed on CT scans are related to outcome. For example, it has been found that subdural hematoma, midline shift, and abnormalities of the third ventricle are associated with poorer prognosis and greater injury severity on the GCS (Wardlaw, Easton, & Statham, 2002). In contrast, epidural hematomas are less predictive of a severe TBI (Gennarelli et al., 1982; Wardlaw et al., 2002). Classification systems for TBI severity based on neuroimaging readily exist in the literature. For example, Marshall et al., (1991) developed a system based on observations from CT scans (e.g., cisterns, midline shift, lesion densities) and grouped individuals with diffuse injury severity on levels I to IV. The Marshall Classification system has been found to correlate with GCS ratings (Marshall et al., 1991). However, there is to my knowledge no literature on converting classifications based on CT scans to GCS-based severity categories (e.g., mild, moderate, severe), which would be necessary

for the current study. As such, a classification system was devised based on the literature discussed for the purposes of grouping participants in one of the two severity group for this study. Specifically, participants were classified as having mild complicated-moderate TBIs if there were positive neuroimaging findings but no evidence of a subdural or subarachnoid hematoma and no midline shift on CT scan, and, if coma data are available, the duration of the post-TBI coma is less than 24 hours. Intracranial abnormalities that were observed for mild complicated-moderate severity may include epidural hematomas, contusions, and diffuse axonal injury. Participants were classified into the severe TBI category if a subdural or subarachnoid hematoma or midline shift is present on CT scans or, if coma data is available, the coma duration is greater than 24 hours.

Participants were excluded if they had a previous history of TBIs, psychiatric or neurological disorders, serious medical illnesses, developmental disabilities, or pre-existing cognitive decline or disorders. Table 2 lists all excluded conditions for this study. Medical, neurologic, psychiatric, and developmental history was originally assessed by way of participants' self-report during the interview as well as their medical records. In addition, participants were excluded if they did not speak fluent English at the time of assessment, as this would affect test performance. Lack of fluency was determined by whether the record indicated the need for an interpreter and scores on the Wide Range Achievement Test 4 (WRAT-4) Reading and Sentence Comprehension subtests. Participants with scores lower than a Grade 5 equivalency level on these tests were excluded.

Table 2

Conditions Excluding Participation in the Study

Excluded conditions
Major psychiatric disorders
Bipolar Disorder
Schizophrenia
Substance Abuse/Dependence
Previous history of TBI
Serious medical illnesses
Developmental disabilities
Learning disorder
Autism
Neurological conditions
Dementia (any etiology aside from TBI, including Alzheimer's Disease, Vascular, Frontotemporal Dementia, Dementia with Lewy Bodies)
Pre-existing abnormal cognitive decline (e.g., Mild Cognitive Impairment)
Cerebrovascular disorders (e.g., stroke)
Movement disorders (including Parkinson's Disease, Huntington's Disease, Progressive Supranuclear Palsy)
Multiple Sclerosis
Normal Pressure Hydrocephalus
Brain tumors and cancers affecting the brain
Penetrating brain injury

Participants also were excluded if they were deemed to be exerting suboptimal effort during the assessment. Due to the nature of these referrals, participants were all administered various symptom validity tests (SVTs) to assess for suboptimal effort. For the purposes of this study, scores from multiple SVTs were used to exclude participants with suboptimal effort. This approach to determining suboptimal effort is supported by

recent literature showing that failure on just one SVT is not uncommon for examinees exerting adequate effort in real world clinical settings (Larrabee, 2014; Victor, Boone, Serpa, Buehler, & Ziegler, 2009). Although these tests strive for maximum accuracy in distinguishing between those exerting good and poor effort, none of the tests alone has 100% specificity and sensitivity. Victor et al. (2009) found that relying on one SVT failure provides good sensitivity (94.6%) but low specificity (53%), indicating a high false positive error rate. By examining the pattern of performance across multiple SVTs, one is able to attain greater specificity and sensitivity in determining the credibility of one's test scores. Victor et al. (2009) devised and recommended a "pairwise model" for this purpose. This method, which involves requiring failure on any two SVTs, has been found to provide high sensitivity (93.8%) and specificity (93.9%) and a good overall hit rate of 90.3%. Using any more than two failures (e.g., 3 or 4) resulted in high specificity but low sensitivity (Victor et al., 2009). Hence, pairwise failure on SVTs appears to provide the best sensitivity and specificity and was used to exclude participants exerting suboptimal effort in the current study. Specifically, participants were excluded if they scored below the cut-off of two SVTs for those with at least two SVTs administered (n = 43). For participants who were administered only one SVT or if only one was available for the current study (n = 24), participants were excluded if they failed this SVT.

All participants received at least the Test of Memory Malingering (TOMM), a 50-item forced-choice test measuring visual recognition memory and used to assess for suboptimal effort (Tombaugh, 1996). Several other SVTs were originally administered to clients on a case-by-case basis, including but not limited to the Dot Counting Test (DCT; Boone, Lu, & Herzberg, 2002), Rey 15-Item Memory Test (RMT; Rey, 1964), Victoria

Symptom Validity Test (VSVT; Slick, Hopp, & Strauss, 1997), and Word Memory Test (WMT; Green, 2005). Because there was variability in how many SVTs were administered for each participant, the percentage of SVTs passed was reported for this study instead of the absolute number of SVTs passed.

The recommended test manual cutoff scores were used to exclude for suboptimal effort for the TOMM, DCT, and the WMT. For the RMT, cutoff scores for the combined recall and recognition score (i.e., free recall + [recognition hits – false positives]) was used as it provides better sensitivity (71%) and specificity (>92%) than using just the recall cutoff alone (Boone, Salazar, Lu, Warner-Chacon, & Razani, 2002). For the VSVT, the recommended cutoff scores reported in the manual were not used as subsequent research found that these criteria are rather conservative and produce high numbers of false negatives (Macciocchi, Seel, Alderson, & Godsall, 2006). Macciocchi et al. (2006) developed a set of guidelines for a sample of severe TBI patients which produced no false positives and greatly improved the true positive rate from 5% in the manual guidelines to 75% using their criteria. Thus, in the current study, participants were excluded if their scores fall in the invalid range on the guidelines proposed by Macciocchi et al (2006). The Trail Making Test Part A (TMT-A) was also used as an embedded SVT measure. Time cutoff scores for mild complicated and moderate-severe TBI were used based on the guidelines reported by Iverson, Lange, Green, and Franzen (2002). These cut-off scores were based on those scores falling at or below the 5^{th} percentile in a TBI sample as this suggested a red flag for possible malingering. However, the authors note that the TMT has low sensitivity in detecting suboptimal effort

alone and should be used in conjunction with other tests (Iverson et al., 2002), which is what the current study has done.

It is acknowledged that many other factors (e.g., hypertension, pain) affect post-TBI functioning and performance on neuropsychological tests. However, these other factors were not a basis for exclusion in the current study. Although this may be a potential limitation as the sample may not be as "clean," there must be a balance between internal and external validity. In other words, some potentially confounding factors were not used as exclusion criteria in order to preserve generalizability and representativeness of the sample as well as to increase power. Furthermore, some factors potentially affecting performance (e.g., pain, fatigue) were not originally measured in participants. As discussed later, where possible confounding factors were not excluded, they were statistically controlled for if differences existed between age groups.

The number of participants required for this study was determined a priori with a power analysis. The G-Power application was used to this end (Faul, Erdfelder, Lang, & Buchner, 2007). Because the moderation analysis (described in the Statistical Analysis section) contained the greatest number of predictors (and thus, will require the largest sample size), the power analysis was completed based on this test. A fixed linear multiple regression model – R^2 increase was specified. Alpha level was set as 0.05, power as 0.80, and the total number of predictors as 3 (cognitive outcome, age, and the interaction term) and the number of tested predictors as one (interaction term). Cohen's f^2 was used as an effect size measure for multiple regression analysis to calculate sample sizes, and guidelines for determining the size of small, medium, and large effect were set at f^2 = 0.02, 0.15, and 0.35, respectively, as suggested by Cohen (1988). Because there has been

no previous research on the relation between cognitive and emotional outcomes moderated by age, the size of this effect could not be estimated. In other words, it cannot be assumed without further research whether a small, medium, or large moderation effect exists. As such, a sample size was calculated assuming all three effect size levels. If the actual effect of this relationship was small ($f^2 = 0.02$), then the total sample size required to see the effect is 395. If the actual relationship was a medium effect ($f^2 = 0.15$), then the total sample size required to see the effect is 55. Finally, if a large interaction effect existed ($f^2 = 0.35$), then the total sample size required is 25.

Measures

Cognitive outcome. Cognitive outcome was measured with the Neuropsychological Assessment Battery (NAB) – Screening Module (Stern & White, 2003). This screening module includes tests covering attention, language, memory, spatial, and executive functioning and is designed to provide a quick assessment of an individual's abilities in these domains. As part of the larger modular battery, it is validated as a stand-alone measure as well as an initial test to determine which subsequent modules of the NAB should be administered. Because the items on the NAB Screening Module are similar to those found in the other respective modules, the Screening Module has been shown to be a good predictor of performance on other NAB modules (Strauss, Sherman, & Spreen, 2006). The NAB is particularly suitable for this study because the domains within the screening module are co-normed and is appropriate to use for individuals ranging from 18 to 97 years old, thus covering a wide age range.

Research investigating the performance on the NAB for individuals with moderate to severe TBI suggests that overall, the NAB Screening Module is a reliable and valid measure for this population. Specifically, the Total Screening Index score (S-

NAB; a sum of the five screening domain scores) has been found to maintain both internal consistency and construct and convergent validity in those with moderate to severe TBI (Zgaljardic & Temple, 2010). In terms of the five index scores of the Screening Module (Attention, Memory, Language, Spatial, Executive Function), it appears that there is weak internal consistency for all of these index scores in this population, although Cronbach's Alpha was in the acceptable range ($\alpha = .60$) for the S-NAB (Zgaljardic & Temple, 2010). This suggests that the subtests contributing to the index scores are not reliably correlated with each other. However, all index scores except for Executive Function have adequate convergent and construct validity, as they were significantly correlated with various neuropsychological tests of their respective cognitive domain (e.g., Trail Making Test, Logical Memory, Boston Naming Test; Zgaljardic & Temple, 2010). It is not too surprising that the Executive Function index score of the NAB Screening Module is only weakly correlated with other established executive function measures (e.g., the Wisconsin Card Sorting Test), as many different cognitive abilities fall under the umbrella of "executive function" (e.g., inhibition, planning, organization, switching; Lezak et al., 2012). Hence, the Executive Function index may be tapping into different aspects of executive function than the tests it is being correlated with. Despite the poor reliability and validity of the Executive Function index score for a moderate to severe TBI population, this index score was used in this study as executive dysfunction is a common complaint for many individuals after a TBI. However, the limitations of including this index are recognized. Overall, the S-NAB and the Attention, Memory, Language, and Spatial index scores maintain good construct validity, but only the S-NAB shows adequate reliability in a TBI population.

For the present study, the S-NAB standard score was used to provide an overall level of cognitive functioning. In addition, the Attention, Memory, and Executive Function index standard scores from the screening module were used to assess these specific cognitive abilities as these cognitive abilities have been found to be the most sensitive to changes after TBI (McDonald, Flashman, & Saykin, 2002; Salmond, & Sahakian, 2005). In addition, research with the NAB suggests that the Attention, Memory, and Executive Functions scores on the NAB Screening Module and module index scores are most likely to be scored in the impaired range for individuals with mild to moderate TBI (Stern & White, 2003). The current study used standardized scores for the S-NAB, Attention, Memory, and Executive Function index scores and no raw scores were used for the analyses. These standardized scores were normed based on a demographically corrected sample (N = 1448) consisting of healthy community-dwelling individuals ranging from 18 to 97 years old (Stern & White, 2003). The standardized scores take into account age (ranges: 18-29, 30-39, 40-49, 50-59, 60-64, 65-69, 70-74, 75-79, 80-97), sex, and education level (<11 years, 12 years, 13-15 years, >16 years). Although the present study did not use an age-matched control group, the use of standardized scores based on demographically corrected normative data allows correction for normal age-related changes in cognition and education-based differences. In other words, because the standardized scores already account for age-related changes in NAB scores, the normative data serves as the age-matched control for the present study. Table 3 describes the specific tests in the S-NAB and its indices.

Table 3

Tests in the NAB Screening Module

Domain	Test
Attention	Orientation
	Digits Forward
	Digits Backward
	Numbers & Letters
Language	Auditory Comprehension
	Naming
Memory	Shape Learning
	Story Learning
Spatial	Visual Discrimination
	Design Construction
Executive function	Mazes
	Word Generation

Emotional outcome. The Beck Depression Inventory Second Edition (BDI-II) and Beck Anxiety Inventory (BAI) were used to assess emotional outcome. The BDI-II is a 21-item self-report inventory that measures severity of endorsed depressive symptoms (Beck, Steer, & Brown, 1996). As one of the most frequently used tests by psychologists, the BDI-II has good psychometric properties, including high internal reliability and strong convergent and divergent validity with other measures of depression, including the SCID (Strauss et al., 2006). As per instructions, participants were to rate the statement for each item that best fits their mood in the past two weeks (Beck et al., 1996). There are 4 choices for each item representing different levels of severity. Higher scores indicate more severe levels of depression. The highest possible score on the BDI-II is 63.

Similarly, the BAI is a 21-item self-report inventory measuring severity of endorsed anxiety symptoms (Beck & Steer, 1993). Studies have shown that the BAI also has high internal reliability and convergent and divergent validity (Fydrich, Dowdall, & Chambless, 1992). Participants were instructed to rate the statements on a 4-point scale,

with higher scores indicating more severe levels of anxiety. The highest possible score on the BAI is 63.

The BDI-II and BAI both use cut-off ranges for interpretation and no standard scores or age-norms are available. Hence, the raw scores from the BDI-II and BAI were used for this study. Details about the original normative sample used to create the interpretive ranges are lacking, and no consistent marked age effects have been reported. However, a recent, large-scale study reported no significant differences in mean BDI-II scores between healthy community-residing individuals 17-29 years old (M = 9.21, SD = 8.5) and those 55-90 years old (M = 7.63, SD = 6.24; Segal, Coolidge, Cahill, & O'Riley, 2008). This study did not include a middle-aged group, and thus it is unclear whether an age effect might have been evident if those 30 to 54 years old were included. As discussed, there are differences in anxiety, depression and subclinical depression rates between older and younger adults. Given that there are no age norms for the BDI-II or the BAI and there was no age-based comparison group recruited for this study, the effect of age-related changes in depression and anxiety would be difficult to separate from the effect of TBI on these variables in different age groups. However, this problem is mitigated by findings from at least one large-scale study (Segal et al., 2008) showing no difference in BDI-II scores across those 17-29 years old and 55-90 years old.

Both the BDI-II and BAI were designed to measure subjective levels of depressive and anxiety levels reported by the participant and alone are not diagnostic of any depressive or anxiety disorders. While it may be argued that this may be a potential limitation, these screening measures have an advantage over structured interviews (e.g., the SCID) in that they take into account subclinical levels of depression, which may still

be clinically significant in affecting an individual's everyday functioning. As discussed, subsyndromal levels of depression may be more common in older adults (Heun et al., 2000; Lebowitz et al., 1997). While depression rates in older adults may range from 1% to 2% in the community, rates for subsyndromal depressions are observed from 13% to 27% in older adults in the community (Lebowitz et al., 1997). This level of depression may not reach the threshold of DMS-V or ICD-10 diagnoses but will still be captured with the BDI-II.

Some research has suggested that the BDI is not a suitable measure of depression for a TBI population, as scores on the BDI are more related to non-depressive symptoms after TBI (e.g., cognitive disturbance, fatigue, somatic complaints) than to a diagnosis of depression (Sliwinski, Gordon, & Bogdany, 1998). However, this study (Sliwinski et al., 1998) used the first edition of the BDI and not the BDI-II. Only three of the original items were unmodified from the first to the second version (Strauss et al., 2006), and it is likely that the findings of this study are not applicable to the new version. Furthermore, the nature and severity of the TBI sample in this study is unclear, as the criteria were only that participants self-identified as receiving a TBI and having a disability (Sliwinski et al., 1998). More recent research using the BDI-II with a TBI population revealed that there is a 3-factor structure of the BDI-II for this population, suggesting that this tool has adequate construct validity and is suitable for use for individuals with TBI (Rowland, Lam, & Leahy, 2005). Research on the use of the BAI in a TBI population is deficient.

Furthermore, some concerns have been raised in the literature that the original BDI is not appropriate as a screening tool for use with older adults. This concern stems generally from the finding that many of the items on the BDI deals with somatic

symptoms, which many older adults may endorse due to medical conditions and not depression (Gallagher, 1986). As such, research on the original BDI had mixed support for the use of this measure with older adults (e.g., Gallagher, Nies, & Thompson, 1982; Olin, Schneider, Eaton, Zemansky, & Pollock, 1992). However, this may not be a valid concern for the BDI-II. As discussed, only three of the original items were unmodified from the first to second edition, and thus it cannot be assumed that the second edition also shares these flaws with its predecessor. While little research has been conducted with the BDI-II, the available studies suggest that the BDI-II has strong psychometric properties when used with older adults and is a good screening measure for depression in this population (Segal et al., 2008). Particularly, the BDI-II appears to maintain good internal reliability as well as convergent and discriminant validity with a sample of community-dwelling older adults (Segal et al., 2008). Although it may be argued that a screening measure specifically designed for older adults, such as the Geriatric Depression Scale (GDS; Yesavage et al., 1983) may be more appropriate for assessing depression in older adults, studies have shown that the BDI-II is highly correlated with the GDS in this population (Jefferson, Powers, & Pope, 2001). Furthermore, it would not be reasonable to use the GDS for the present study as the purpose is to evaluate emotional functioning across the lifespan, not just in older adults. In other words, the GDS was developed for measuring depression in those over the age of 55, but the present study assessed depression in both younger and older adults. Overall, the BDI-II, which has been shown to have good psychometric properties for both younger and older adults, is a more appropriate measure for the current study.

Procedure

The current study was retrospective and cross-sectional in design. Archival data from the period between 2010 and 2013 were analyzed. Originally, participants underwent an interview and neuropsychological evaluation of their cognitive abilities. Testing took place in a quiet room free of distractions. Testing was completed by one of four psychometrists with at least a bachelor level education. These psychometrists received training in neuropsychological and psychological testing prior to commencing the position and were supervised by the registered neuropsychologist of the practice. Training included observation of other examiners' administration of the tests, watching the NAB Screening Video Training Program DVD, undergraduate coursework in neuropsychological assessment, and practice administrating the tests. Although standardized instructions and procedures were followed as much as possible, measurement variability (i.e., variability in how participants were tested) may be inherent when using multiple examiners. As such, the four examiners were compared on their participants' cognitive performance in order to determine and rule out any examiner effect on performance.

Informed consent was obtained prior to completing the assessment. Before the NAB was given, participants were assessed for suboptimal effort by way of the SVTs. The NAB Screening Module took approximately 45 minutes to complete. Participants were administered all subtests of this module with standardized instructions. Similarly, the BDI-II and BAI were administered with standardized instructions. Other neuropsychological tests that are not used in this study were also administered to individuals during the session (e.g., Wechsler Abbreviated Scale of Intelligence). These tests were typically given after administration of the SVTs and NAB and before the self-

report questionnaires. Total testing time (including the interview) varied depending on the number of other tests administered and variability in participants' speed, but usually was in the range of approximately 3 to 5 hours.

Participants in the database were screened for eligibility criteria and a new database was created containing cases meeting the inclusion and exclusion requirements of the study. The University of Windsor Research Ethics Board approved the study protocols and use of human participants in this study.

Statistical analyses

All analyses were conducted using SPSS. Demographic and injury variables were first examined and analyzed for any differences between age groups. These comparisons were conducted using one-way ANOVA for non-categorical variables (e.g., education) and the Chi-square test for categorical variables (e.g., gender, occupation). Differences in any demographic or injury variables were controlled for by entering it into the model as a covariate. Before the hypotheses were explored, a missing data analysis of the dataset was completed. The mechanism and pattern underlying the missing data was examined and missing data was handled using the Markov Chain Monte Carlo (MCMC) multiple imputation technique. Multiple imputation is currently the "gold-standard" in dealing with missing data and has replaced many of older techniques of mean substitution, case deletion, and simple imputation as it reduces the issues, such as decreased variability, sample bias, and a loss of power, inherent in many of these techniques (Graham, 2009). Multiple imputation involves creating simulated values predicted from the participant's observed values on other variables with random noise added to preserve variability (Schafer & Graham, 2002). This is done several times and the imputed datasets are pooled together to correct for error in predicting the missing values. This method is

suitable for an arbitrary pattern of missing values (i.e., missing at random; Schafer & Graham, 2002), which is an additional advantage over using traditional listwise case deletion, which assumes and provides only valid inferences when the data is missing completely at random (MCAR; Allison, 2002). Although some concerns of this technique have been voiced, including that filling in the unknown spaces is just "making up data" and may be inaccurate, this claim is unsupported by the literature (Graham, 2009). While it may be true that any single imputation has the limitation that the estimated values contain substantial error, by creating and pooling multiple imputed datasets, such error should be reduced and the resulting pooled dataset should preserve important characteristics, such as the variances, means, and correlations (Graham, 2009).

For the first two hypotheses, the relation between age with cognitive and emotional outcomes was examined via hierarchical multiple regression analyses (MRA). Separate regression analyses were conducted with the S-NAB, Memory, Executive Function, and Attention index scores, BDI-II, and BAI as dependent variables. The assumptions of MRA were first assessed for each regression analyses as violations of the assumptions can potentially invalidate the findings and threaten the inferences that can be made. In addition, the data was cleaned for any outliers and influential cases. For all analyses, injury severity and time post-injury were entered first into the model, as past literature has suggested an association between these variables and post-TBI cognitive and emotional functioning. Age was entered subsequently. To examine the nature of the association between that cognitive and emotional outcomes and age, a trend analysis was conducted. This was done by firstly creating new variables to represent the quadratic term by raising the values of Age to the power of 2 and then entering the quadratic term into

the model after age. To assess whether adding a quadratic term significantly contributed to the model, the R^2 change from adding the quadratic variable was examined for significance. A significant R^2 change would indicate that the quadratic term accounted for a significant portion of variance over and above the linear model.

In addition to examining the contribution of the variables (severity, time post-injury, age, age^2) to the overall model, each predictor was examined individually for its contribution to the model. This is typically analyzed using β-weights in the literature. However, there are criticisms of using only this measure, as β-weights are context-dependent and influenced by what other variables are entered into the model (Courville & Thompson, 2001). Hence, adding or removing variables could change the weights, and interpretation depends on an exact correctly specified model. In addition, β-weights are not meant to measure the relationship between predictor and outcome but simply how much change in the outcome variable exists for every standard deviation change in the predictor variable. Given these limitations, Courville and Thompson (2001) recommend including structure coefficients (i.e., bivariate relationship between predictor and outcome; $r_{s)}$ in conjunction with β-weights in the interpretation. Thus, structure coefficients were also calculated to examine the contributions of each predictor variable to the model.

Finally, to investigate whether age moderates the relation between cognitive and emotional outcomes, a separate hierarchical MRA was conducted. For this analysis, emotional outcomes (BDI-II and BAI scores) were used as the outcome variables in two separate regression analyses. The predictor variables (Age and S-NAB) were centered to address issues of multicollinearity and a new interaction variable (NABxAge) was

created. Order of entry into the model was the following: NAB, age, and the NABxAge interaction term. The interaction variable was assessed for significance. In addition, graphs of the relationship between cognitive and emotional outcome for different age groups were visually examined for any moderating effect of age.

An interaction in MRA using the variable definitions above would imply that the regression line of the outcome variable (emotional outcomes) depends on both the predictor (cognitive outcome) and moderator (age) variables. However, it is important to note that MRA does not imply there is a causal relationship between the predictor and dependent variables; it reveals only if a relationship (causal or not) exists. Causality is determined only through a theoretical and logical basis. Hence, the labelling of cognitive outcomes as the predictor variable and emotional outcomes as the dependent variable does not necessarily mean that this is the direction of the relationship. It may be that cognitive functioning affects emotional functioning post-TBI, but it may well be that emotional functioning also affects cognitive performance. Alternatively, cognitive and emotional functioning may both be affected by a third variable. Such an assignment of cognitive and emotional outcomes to predictor and outcome variables are arbitrary for this specific analysis, as the goal is to assess the moderating effect of age on the relation between cognitive and emotional outcomes, regardless of the causal direction between them.

V. RESULTS

Description of data

A set of 174 cases with at least a mild complicated TBI and judged to reflect valid effort (based on the neuropsychologist's comments in the database and not the criteria of the current study) were initially selected from hundreds of cases in the database. Of these cases, 107 cases were excluded as per the exclusion criteria. Specifically, participants were excluded for being younger than 18 years old (n = 8), use of an interpreter (n = 17), mild uncomplicated severity or severity could not be determined (n = 28), history of major psychiatric disorders (n = 24), developmental disorders (n = 8), neurological disorders (n = 10), previous TBI (n = 7), or serious medical conditions (n = 7), and failure on SVTs (n = 9). In addition to the set exclusion criteria, 11 cases were also removed due to inadequate available data, one case removed as it was a repeat assessment for a participant already included, and one case removed for age as the NAB norms did not extend to that age. Several of these excluded cases fit more than one of the exclusion categories discussed.

A total of 67 participants were included in the analyses after removing those not fulfilling inclusion criteria. The participants on average were 40.5 years old (SD = 19.8) and the sample included more younger than older adults (Table 4). However, there was still an adequate number of older and middle aged participants to conduct the current study. Comparison of participants' NAB scores between psychometrists revealed no significant differences ($F(5, 60) = .84$, $p = .53$), suggesting that there were no systematic differences in how examiners administered the NAB. As can be observed in Table 4, the sample of participants was not equally distributed on all demographic variables. Specifically, males and single marital status made up 62.7% and 49.3% of the sample

respectively. However, this distribution mainly reflects the composition of the younger adult group and not the middle-aged and older adult groups. Education appeared to be equally distributed, with approximately half of participants completing more than a Grade 12 education. Although the sample included a substantial portion of participants who were born outside of Canada and whose first language is not English (ESL), these participants were fluent in English as evident on the WRAT-4 Reading and Sentence Comprehension subtests. Furthermore, none of the participants required the use of an interpreter. One participant had a WRAT-4 Reading Grade equivalent of 2.0 but was included in the study as an interpreter was not required.

Comparison amongst age groups revealed no differences in gender, education, number born outside Canada or ESL between younger, middle-aged, and older adults. Because some of the occupation and marital status categories had fewer than 5 participants, the Chi-square assumption for the minimum frequencies per cell (no less than 5) was violated. To correct for this, the Fisher's Exact Test was used, which gives a better estimate for smaller samples. The analysis revealed that there were differences in occupation ($\chi^2(10, N = 67) = 43.7, p < 0.01$) and marital status ($\chi^2(6, N = 67) = 52.7, p < 0.01$) between age groups. As can be observed in Table 4, younger adults were more likely to be unemployed or students and were mostly single while older adults tended to be retired and married. Such differences are not unexpected between age groups, and as an ANOVA revealed that these variables do not relate to the outcomes (e.g., S-NAB, BDI-II) of the current study, marital status and occupation were not controlled for as covariates.

Participants completed between one to seven SVTs (M = 2.9, SD = 1.1). After removing participants with pairwise failures, the mean pass rate was 94.9% (SD = 13.6%). The TOMM and the RMT were the most frequently administered SVTs, with all participants receiving at least one of these tests.

Table 4

Demographic Characteristics of the Sample

	Younger (18-29 years)	Middle-Aged (30-49 years)	Older (50-88 years)	Total
n	30	17	20	67
Age[a]	22.6 (4.2)	42.3 (5.9)	65.7 (11.6)	40.5 (19.8)
Gender (Male)[a]	21 (70%)	10 (59%)	11 (55%)	42 (62.7%)
Education[a]	12.7 (2.4)	15 (3.7)	13.9 (4.4)	13.6 (3.5)
> High School	36.7%	70.6%	60%	52.5%
Occupation*				
Unemployed	8 (26.7%)	1 (5.9%)	0	9 (13.5%)
Student	9 (30%)	0	1 (5%)	10 (15%)
Manual	6 (20%)	3 (17.6%)	0	9 (13.5%)
Intermediate	7 (23.3%)	11 (64.7%)	8 (40%)	26 (39%)
Professional	0	2 (11.8%)	2 (10%)	4 (6.0%)
Retired	0	0	9 (45%)	9 (13.4%)
Born outside Canada	4 (13%)	8 (47%)	7 (35%)	19 (28.4%)
English Second Language	4 (13%)	5 (29%)	4 (20%)	13 (19.4%)
Marital Status*				
Single	28 (93.3%)	5 (29.4%)	0	32 (49.3%)
Married	2 (6.7%)	10 (58.8%)	15 (75%)	27 (40.3%)
Divorced	0	1 (5.9%)	1 (5%)	2 (3%)
Widowed	0	1 (5.9%)	4 (20%)	5 (7.5%)

[a] M(SD)

*p < .01 for Age.

In terms of injury variables, participants had GCS scores ranging from 3 to 15, with an average GCS of 10 (Table 5). A total of 9 participants did not have GCS data available and were classified into severity groups based on their neuroimaging findings as discussed in the methods section. Using this classification, 6 participants were grouped under mild complicated-moderate and 3 under severe TBI. One participant with evidence of a small subdural hematoma was classified as mild complicated-moderate rather than severe as there was no evidence of any other positive imaging results. Overall, the sample tended to consist of more mild complicated-moderate (59.7%) than severe TBIs (40.3%). Furthermore, injury severity differed significantly between age groups (χ^2 (2, N = 67) = 8.67, p = .02), with less severe injuries with increasing age at time of injury (Table 5). Specifically, there were significantly more older adults with milder injuries (85%) with only 3 older participants (15%) having severe TBIs, while the younger group consists of more severe (56.7%) than mild (43.3%) injuries. Because of these differences in injury severity between age groups, the analyses were conducted with the two injury severity categories separately in addition to the whole sample. It is important to note, however, that because of the small number of older adults with severe injury, there was very low statistical power for analyses examining age effects of time of injury on cognitive and emotional outcome in those with severe TBI and for analyses of cognitive and emotional outcomes in those older adults who sustained severe injury.

Participants sustained their injuries on average 33.5 months before their assessment (Table 5). The majority of participants sustained their injuries through a motor vehicle accident. No differences in time post-injury and mechanism of injury between age groups were found.

Table 5

Injury Characteristics of the Sample

	Younger (18-29 years)	Middle (30-49 years)	Older (50-88 years)	Total
n	30	17	20	67
Glasgow Coma Scale[a]	8.6 (4.4)	10 (4.5)	12.5 (3)	10 (4.3)
Neuroimaging	All positive	1 Negative	All positive	1 Negative
Severity*				
Mild -Moderate	13 (43.3%)	10 (58.5%)	17 (85%)	40 (59.7%)
Severe	17 (56.7%)	7 (41.2%)	3 (15%)	27 (40.3%)
Time post-injury[ab]	34 (32.3)	35 (19.1)	31.6 (20.1)	33.5 (25.8)
Range	3 – 158	6 – 90	11 – 82	3 – 158
Mechanism of injury[c]				
MVA	19 (63.3%)	11 (64.7%)	9 (45%)	39 (58.2%)
Pedestrian	8 (26.7%)	4 (23.5%)	7 (35%)	19 (28.4%)
Motorcycle	1 (3.3%)	1 (5.9%)	2 (10%)	4 (6%)
Fall	1 (3.3%)	1 (5.9%)	1 (5%)	3 (4.5%)

[a]*M*(*SD*); [b]Months; [c]Missing data from 1 participant

*$p < .05$ for Age.

Assumption testing, data cleaning & missing data analysis

Missing data. All participants had complete demographic data (e.g., age, education). While NAB scores were available for all 67 participants, one participant did not complete certain subtests on the Attention, Spatial, and Executive Function modules, thus resulting in a missing S-NAB score. Because only one case was missing for NAB data, this was handled by simple pairwise case deletion for the analyses affected as it would not bias subsequent analyses and would be more reasonable than conducting multiple imputation. Hence, for the analyses of S-NAB, Attention and Executive Function modules, only 66 participants were included while 67 participants were included for the NAB Memory module analyses.

In terms of the BDI-II and BAI, data were missing for a substantial number of participants. Specifically, 23 (34%) participants did not have any BDI-II and BAI scores

(including the descriptive range) and 5 (7.5%) participants had only the descriptive range of their scores (e.g., mild, severe). An analysis of the missingness mechanisms revealed that the missing data was both missing completely at random (MCAR) and missing at random (MAR). MCAR implies that the missing data is not related to any observed variables in the study (Schlomer, Bauman, & Card, 2010). MAR implies that the missing data is related to another variable in the study but not the variable on which data were missing (Schlomer et al., 2010). Little's MCAR test (Little, 1988) revealed that the missing BDI-II and BAI data is MCAR (χ^2 (16, N = 67) = 15.96, p = .46). However, the missingness mechanism was also analyzed by dummy coding missingness and conducting separate *t*-tests, which revealed that participants who had completed BDI-II and BAI data differed from those with missing data on the age variable (t (65) = 2.4, p = .02). Specifically, the missing data group consisted of participants who were younger than the completed data group. This finding suggests that the data is MAR. While these findings appear to contradict themselves in that the data cannot be both MCAR and MAR, MCAR has been conceptualized as a special case of MAR (Schlomer et al., 2010). Indeed, it is recommended to view mechanisms for missingness along a continuum instead of being mutually exclusive categories (Graham, 2009). Furthermore, it is possible that Little's MCAR test may not have captured the difference for the age variable as it is an omnibus test that evaluates significance on all variables combined instead of separate *t*-test comparisons (Schlomer et al., 2010).

There is also a possibility that the current missing data is missing not at random (MNAR), meaning that the missing data is related to the variable for which data are missing, and is a non-ignorable mechanism of missingness in that it would bias further

analyses (Schlomer et al., 2010). For example, it is possible that the participants who were originally administered the BDI-II and BAI presented as more depressed and anxious during the interview. Of note, participants who were not administered the BDI-II and BAI were more likely to have been administered the Personality Assessment Inventory (PAI; Morey, 1991), a 344-item self-report measure of personality and psychopathology. However, it is unclear from this alone whether this relates to the mechanism of missingness. For example, participants may have been administered the PAI instead of the BDI-II and BAI if they were deemed to have more psychopathology (e.g., mood and anxiety symptoms) from the clinical interview, which would argue that the data is MNAR. On the other hand, participants may have been administered the PAI over the BDI-II and BAI based on any number of factors, such as the time available for test administration (the 344 item-PAI versus the 21 item-BDI-II/BAI) or the tolerance level for testing for a particular individual due to varying fatigue and pain issues amongst this population. In such cases, whether or not the PAI was administered instead of the BDI-II and BAI is not related to missingness on the BDI-II and BAI itself. The possibility of MNAR is not testable and there is no way to distinguish between MAR and MNAR aside from following-up with the original participants who had missing data (Schafer & Graham, 2002). Thus, the true mechanism for missingness can only be speculated. Even if the data is MNAR, some literature have suggested that it would not severely bias multiple imputation (Schafer & Graham, 2002). Schafer and Graham recommend performing MAR-based methods, such as multiple imputation, over methods specific to MNAR unless the researcher is sure that the true cause of missingness is because of the response variable itself. This is because for most situations of MNAR, the true cause of

missingness is due to a third unmeasured variable, which would only produce minor bias for multiple imputation (Schafer & Graham, 2002). Hence, the MCMC multiple imputation technique was deemed to be appropriate in handling the 23 cases of missing data for the current study.

The standard procedure for multiple imputation is to use 3 to 10 imputations as this is sufficient for balancing accuracy and efficiency of the procedure (Schafer & Graham, 2002). The exact number of imputations necessary depends on the amount of data missing and was calculated based on the formula $100/(1 + \lambda/m)^{-1}$, where λ is the rate of missing information and m is the number of imputations (Schafer & Graham, 2002). With 5 imputations ($m = 5$) and 32% as the rate of missing data ($\lambda = 0.32$), the multiple imputation procedure would be 93.6% efficient in predicting the estimates. With 10 imputations, the procedure would be 96.9% accurate, and with 20 imputations, 98.4%. Hence, it appears that 5 imputations would suffice to obtain accurate estimates, and additional imputations would increase the accuracy of the estimates very little. Thus, 5 imputations were performed and a maximum of 10 iterations was set. Linear regression was used as the model type to calculate the imputed variables. All of the variables were set as predictors in the model to compute the imputed values and the BDI-II and BAI were set as both predictors and to be imputed. For the BDI-II and BAI results, both the original and pooled results from the imputed datasets were reported. For the moderation analysis, the interaction term (NABxAge) was created before the data was imputed. Some literature has suggested that interaction analyses may not be accurate if the data is imputed before the interaction term is created (i.e., the two variables producing the interaction term have already been imputed before creating the new term), as multiple

imputation does not model higher-order computations (Allison, 2002). Hence, imputing after the interaction term has been created avoids this issue.

For the 5 participants who only had the descriptive range of their scores, ranges were converted to BDI-II and BAI scores by using the middle score of that range. For example, a participant with a "Moderate" descriptor on the BDI-II was converted into a score of 24. This was done so that the BDI-II and BAI could still be analyzed as a continuous variable, which would preserve greater variability than if the reverse was done (i.e., converting raw scores into categorical descriptive scores). However, a potential issue with this method is that the created scores, especially for the Severe range, may not represent true scores. Specifically, because it is expected that BDI-II and BAI scores do not follow a normal distribution in this population, but are skewed towards the lower end of the scale, the middle score for the severe ranges may be a more extreme estimate than most of the scores in these ranges. However, as this method was only conducted with 5 participants, the effect on the results should be minimal. Table 6 lists the conversions of descriptive to raw scores.

Table 6

Conversion of BDI-II and BAI Descriptive Ranges to Raw Scores

	BDI-II	BAI
Minimal	6 (0 – 13)	4 (0 – 7)
Mild	16 (14 – 19)	11 (8 – 15)
Moderate	24 (20 – 28)	20 (16 – 25)
Severe	46 (29 – 63)	44 (26 – 63)

Before discussing the specific results, it is important to address the assumptions of MRA and examine and clean the data for any influential and deviant cases. As there were separate analyses conducted for each NAB module, BDI-II, BAI, and the moderation analysis, the diagnostics and assumptions were tested for each analysis. Because the findings from these separate tests were generally similar, they will be discussed as a whole, with only violations of the assumptions discussed separately.

MRA assumes a linear relationship between predictor and outcome variables, an adequate sample size, absence of multicollinearity, normality of residuals, homoscedasticity of errors, independence of errors, and absence of outliers or influential observations. Outliers on the y-axis were identified by examining the standardized residual values. Any standardized residuals that were above or below 2.5 standard deviations were considered outliers. Outliers on the x-axis were identified with the leverage value using a cut-off point of 0.18. Influential observations were assessed based on Cook's Distance of greater than 1 and a standardized DFFIT value of 2. It was found that there were two outliers on the x-axis for all NAB modules, BDI-II, and BAI and one outlier on the y-axis for NAB Memory and Executive Function modules. However, none of these were also influential observations and overall, there were no influential observations. This implies that although there are points that are beyond the standard deviation of 2.5, they are not significantly influencing the model or "pulling" the regression line towards the deviant case. Hence, these outliers were kept in the subsequent analyses in order to preserve power.

Linearity and homoscedasticity of errors were assessed through visual inspection of the standardized predicted vs. standardized residual scatterplot. Linearity and

homoscedasticity are evident if the points are evenly dispersed around the central line and the distribution of points do not curve up or down or "fan out," and this was indeed found for all regression analyses. Normality of residuals was assessed by visually inspecting the histogram plotting the frequency of the residuals, which revealed that the residuals followed a normal distribution for all analyses. Multicollinearity was assessed by examining the tolerance and variance inflation factor (VIF) values as well as analyzing the correlation matrix. Values below 0.1 for tolerance and above 10 for VIF or correlations between variables greater than 0.9 are indicative of violations of this assumption. There was no evidence of multicollinearity for any analyses. Independence of residuals was assessed by the Durbin-Watson test, in which values that are close to 2 are considered to be normal. This test was within normal range for all analyses but was slightly elevated for S-NAB, possibly due to the relationship between age and age^2 (as age^2 is calculated from Age and thus not independent of each other). Finally, MRA requires an adequate sample size in order to obtain a reliable regression model, which has been recommended as approximately 15 cases for every predictor. For this study, there are 67 participants with 4 predictors (severity, time post-injury, age, age^2). Thus, the sample size was adequate for the determination of a reliable model.

Hypothesis 1: Cognitive outcome

Hierarchical regression for the S-NAB, Attention, Memory, and Executive Function modules revealed no significant change in R^2 with the addition of age to the model (Model 3 in Table 7), implying that there was no effect of age after accounting for injury severity and time post-injury. In other words, when age was entered to the model (Model 3), the proportion of variance explained was not above and beyond the proportion of variance accounted for by severity and time post-injury. In addition, age was not a

significant predictor of overall NAB score or module scores in Model 3. As can be observed in Table 8, scores on the NAB were similar across age groups. Even when age was examined without accounting for severity and time post-injury in the model, age did not make any significant contribution in predicting S-NAB (r_s = .11, p = .20), although it emerged as a significant predictor for NAB memory module (r_s = .29, p = .01; Table 7). Aside from age, injury severity and time post-injury did not account for a significant portion of variance for any of the NAB modules or the total score and were not significant predictors in Model 1 and 2 respectively. As can be observed in Table 9, participants with severe TBIs generally had poorer performance on the NAB than those with mild-moderate TBIs, although this was not significantly different. Together, severity, time post-injury, and age (Model 3 in Table 7) accounted for 1.6% of the variance of the NAB total score. The overall fit of all 4 regression models shown in Table 7 was poor, and these models were not better predictors of cognitive outcome than just using the mean to predict NAB scores.

My hypothesis of a nonlinear relationship between age and cognitive outcome was tested in Model 4, in which the quadratic term for age was added. When Age^2 was added to the model, no additional variance was accounted for in NAB total or module scores. Thus, the relationship between age and cognitive outcome does not appear to be nonlinear.

Table 7

Hierarchical Regression Results for NAB scores

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
NAB Total (S-NAB)									
Model 1						.01	.62		
Severity	-3.22	4.09	-.10	-.79					
Model 2						.01	.35	<.01	.08
Severity	-3.07	4.15	-.09	-.74					
Time Post-injury	-.02	.08	-.04	-.29					
Model 3						.02	.34	<.01	.34
Severity	-2.17	4.45	-.07	-.49					
Time Post-injury	-.02	.08	-.03	-.24					
Age	.07	.11	.08	.58					
Model 4									
Severity	-1.93	4.49	-.06	-.43	-.10	.02	.34	<.01	.36
Time Post-injury	-.03	.08	-.04	-.32	-.05				
Age	.38	.53	.44	.71	.11				
Age^2	<.01	.01	-.37	-.60	.09				
NAB Attention									
Model 1						<.01	.27		
Severity	2.24	4.29	.07	.52					
Model 2						.03	.80	.02	1.32
Severity	2.88	4.32	.08	.67					
Time Post-injury	-.10	.08	-.14	-1.15					
Model 3						.04	.89	.02	1.06
Severity	4.53	4.60	.13	.98					
Time Post-injury	-.09	.08	-.14	-1.08					
Age	.12	.12	.14	1.03					
Model 4						.05	.81	.01	.60
Severity	4.85	4.63	.14	1.05	.07				
Time Post-injury	-.10	.08	-.15	-1.17	-.13				
Age	.54	.55	.61	.98	.11				
Age^2	<.01	.01	-.48	-.77	.90				
NAB Memory									
Model 1						.04	2.87		
Severity	-6.62	3.90	-.21	-1.70					
Model 2						.04	1.44	<.01	.05
Severity	-6.74	3.97	-.21	-1.70					
Time Post-injury	.02	.08	.03	.23					
Model 3						.10	2.21	.05	3.62
Severity	-3.91	4.17	-.12	-.94					
Time Post-injury	.03	.07	.05	.40					
Age	.20	.10	.25	1.90					
Model 4						.10	1.70	<.01	.26
Severity	-3.72	4.21	-.12	-.88	-.21*				
Time Post-injury	.02	.08	.04	.31	<-.01				
Age	.44	.48	.55	.91	.29**				

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
Age^2	<.01	.01	-.30	-.51	.26*				
NAB Executive Function									
Model 1						.05	3.55		
Severity	-7.30	3.87	-.23	-1.88					
Model 2						.06	1.83	<.01	.16
Severity	-7.09	3.93	-.22	-1.80					
Time Post-injury	-.03	.08	-.05	-.40					
Model 3						.06	1.39	.01	.53
Severity	-6.03	4.21	-.19	-1.43					
Time Post-injury	-.03	.08	-.04	-.35					
Age	.08	.11	.10	.73					
Model 4						.07	1.15	.01	.48
Severity	-5.77	4.24	-.18	-1.36	-.23*				
Time Post-injury	-.03	.08	-.06	-.44	-.08				
Age	.42	.50	.51	.83	.17				
Age^2	<.01	.01	-.42	-.69	.15				

*p < .05, **p <.01

Table 8

Mean (SD) for Cognitive and Emotional Outcomes by Age Group

	Younger[a]	Middle[b]	Older[c]
S-NAB	91.8 (16.0)	89.4 (17.0)	98.2 (15.6)
NAB Attention	79.9 (17.7)	83.2 (19.0)	87.0 (13.7)
NAB Memory	94.6 (16.0)	92.8 (15.8)	104.9 (13.7)
NAB Executive	91.7 (15.2)	94.3 (19.9)	99.8 (11.5)
BDI-II	18.6 (10.7)	19.2 (15.5)	20.9 (14.2)
BAI	13.4 (13.1)	14.5 (12.8)	14.6 (9.7)

Note. NAB scores represent age- and education-adjusted normed scores. BDI-II and BAI scores are from the original (non-imputed) data.

[a]N = 30 for NAB scores, N = 15 for BDI-II and BAI; [b]N = 17 for NAB scores, N = 13 for BDI-II and BAI; [c]N = 19 for NAB scores, N = 16 for BDI-II and BAI

Table 9

Mean (SD) for Cognitive and Emotional Outcomes by Injury Severity Level

	Mild Complicated-Moderate[a]	Severe[b]	Total[c]
S-NAB	94.3 (17.4)	91.1 (14.6)	93.0 (16.3)
NAB Attention	81.9 (17.5)	84.1 (16.6)	82.8 (17.0)
NAB Memory	99.9 (14.2)	93.3 (17.6)	97.2 (15.9)
NAB Executive	97.7 (16.3)	90.4(14.2)	94.7 (15.8)
BDI-II	23.5 (13.3)	14.1 (11.4)	19.6 (13.2)
BAI	18.4 (12.9)	8.0 (5.4)	14.2 (11.6)

Note. NAB scores represent age- and education-adjusted normed scores. BDI-II and BAI scores are from the original (non-imputed) data.

[a]*N* = 39 for NAB scores, *N* = 26 for BDI-II and BAI; [b]*N* = 27 for NAB scores, *N* = 18 for BDI-II and BAI; [c]*N* = 66 for NAB scores, *N* = 44 for BDI-II and BAI

The same analyses were completed with severity split (Table 10, Table 11). Results were consistent with the above analyses. Specifically, hierarchical regression for S-NAB, Attention, Memory, and Executive Function modules revealed no additional contribution of age beyond time post-injury for both mild complicated-moderate TBI and severe TBI. Age and time post-injury were not significant predictors of the model for either mild complicated-moderate TBI or severe TBI. Of particular interest, the quadratic age term did not significantly account for any more variance in the model in either severity groups. Overall, analyses within groups of similar severity yield results congruent with analyses for the sample as a whole.

Table 10

Hierarchical Regression Results for NAB scores - Mild Complicated-Moderate Severity (N = 39)

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
NAB Total (S-NAB)									
Model 1						.01	.26		
Time Post-injury	.07	.14	.08	.51					
Model 2						.01	.14	<.01	.03
Time Post-injury	.07	.14	.09	.51					
Age	.02	.15	.03	.16					
Model 3						.01	.14	<.01	.15
Time Post-injury	.06	.14	.07	.39	.08				
Age	.32	.78	.36	.41	.02				
Age^2	.00	.01	-.34	-.38	.01				
NAB Attention									
Model 1						.01	.27		
Time Post-injury	.07	.14	.09	.52					
Model 2						.07	.125	.06	2.2
Time Post-injury	.08	.14	.10	.63					
Age	.21	.14	.24	1.49					
Model 3						.11	1.44	.05	1.77
Time Post-injury	.04	.14	.05	.27	.09				
Age	1.19	.74	1.34	1.59	.23				
Age^2	-.01	.01	-1.12	-1.33	.18				
NAB Memory									
Model 1						.02	.71		
Time Post-injury	-.09	.11	-.14	-.84					
Model 2						.06	1.19	.04	1.67
Time Post-injury	-.08	.11	-.11	-.71					
Age	.14	.11	.21	1.29					
Model 3						.06	.78	<.01	.02
Time Post-injury	-.08	.12	-.12	-.71	-.14				
Age	.23	.60	.32	.37	.22				
Age^2	.00	.01	-.12	-.14	.22				
NAB Executive									
Model 1						<.01	.10		
Time Post-injury	.04	.13	.05	.31					
Model 2						.01	.11	<.01	.13
Time Post-injury	.04	.13	.05	.33					
Age	.05	.14	.06	.37					
Model 3						.01	.16	.01	.26
Time Post-injury	.03	.14	.03	.19	.05				
Age	.42	.73	.51	.57	.06				
Age^2	.00	.01	-.45	-.51	.04				

* $p < .05$. ** $p < .01$.

Table 11

Hierarchical Regression Results for NAB scores – Severe Severity (N = 27)

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
NAB Total (S-NAB)									
Model 1						.03	.38		
Time Post-injury	-.08	.09	-.18	-.89					
Model 2						.06	.45	.03	.86
Time Post-injury	-.07	.09	-.16	-.81					
Age	.18	.19	.18	.93					
Model 3						.07	.63	.01	.18
Time Post-injury	-.07	.09	-.15	-.76	-.18				
Age	-.25	1.01	-.26	-.25	.20				
Age^2	.01	.01	.45	.43	.21				
NAB Attention									
Model 1						.15	4.33*		
Time Post-injury	-.20	.10	-.38	-2.08*					
Model 2						.15	2.12	.01	.20
Time Post-injury	-.20	.10	-.39	-2.08*					
Age	-.09	.20	-.08	-.44					
Model 3						.16	1.40	<.01	.01
Time Post-injury	-.20	.10	-.39	-2.02	-.38				
Age	-.18	1.09	-.17	-.16	-.05				
Age^2	.00	.01	.08	.08	-.04				
NAB Memory									
Model 1						.03	.64		
Time Post-injury	.09	.11	.16	.80					
Model 2						.10	1.4	.08	2.01
Time Post-injury	.10	.11	.18	.94					
Age	.32	.22	.28	1.44					
Model 3						.12	1.03	.02	.40
Time Post-injury	.10	.11	.17	.87	.16				
Age	1.06	1.19	.92	.89	.27				
Age^2	-.01	.01	-.66	-.63	.23				
NAB Executive									
Model 1						.03	.41		
Time Post-injury	-.07	.09	-.17	-.84					
Model 2						.06	.51	.03	.68
Time Post-injury	-.07	.09	-.15	-.77					
Age	.15	.18	.16	.82					
Model 3						.06	.72	<.01	.02
Time Post-injury	-.07	.09	-.15	-.74	-.17				
Age	.02	.99	.02	.02	.18				
Age^2	.00	.01	.15	.14	.18				

* $p < .05$. ** $p < .01$.

In contrast to the above analyses, results differed depending on time post-injury. For participants sustaining a TBI less than 12 months before the assessment, injury severity was a significant predictor for S-NAB, Memory, and Executive Function modules, accounting for 49%, 73%, and 55% of the total variance respectively (Table 12). Specifically, those with more severe injuries tended to have lower NAB scores. However, injury severity no longer significantly contributed to the model and in predicting NAB scores in those who sustained TBIs 12 or more months before the assessment (Table 13). In terms of age at time of injury, this variable was significantly related to the NAB Memory domain when participants were at least 12 months post-injury and uniquely contributed 8% of the variance to the model. Aside from this, no relationship (linear or nonlinear) between age and any NAB domain or the S-NAB was found regardless of whether the time post-injury was acute (<12 months) or longer term (≥12 months).

Table 12

Hierarchical Regression Results for NAB Scores at <12 Months Post-Injury (N =8)

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
NAB Total (S-NAB)									
Model 1						.49	5.74*		
Severity	-21.25	8.87	-.70	-2.40*					
Model 2						.53	2.85	.04	.46
Severity	-23.03	9.65	-.76	-2.39					
Age	-.16	.23	-.22	-.68					
Model 3						.53	1.53	<.01	.01
Severity	-22.78	11.05	-.75	-2.06	-.70*				
Age	-.32	1.65	-.44	-.20	-.01				
Age^2	.00	.02	.23	.10	.02				
NAB Attention									
Model 1						.07	.47		
Severity	10.25	14.91	.27	.69					
Model 2						.07	.20	<.01	<.01

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
Severity	10.51	16.96	.28	.62					
Age	.02	.41	.03	.06					
Model 3						.20	.32	.12	.60
Severity	13.60	18.12	.36	.75	.27				
Age	-2.05	2.71	-2.25	-.76	-.05				
Age^2	.02	.03	2.32	.78	-.01				
NAB Memory									
Model 1						.73	16.55**		
Severity	-31.50	7.76	-.86	-4.06**					
Model 2						.74	6.94*	<.01	.04
Severity	-31.97	8.80	-.87	-3.63*					
Age	-.04	.21	-.05	-.20					
Model 3						.85	7.44*	.11	2.97
Severity	-34.87	7.64	-.95	-4.57**	-.86**				
Age	1.90	1.14	2.15	1.67	.19				
Age^2	-.02	.01	-2.24	-1.72	.16				
NAB Executive									
Model 1						.55	7.34*		
Severity	-18.75	6.92	-.74	-2.71*					
Model 2						.58	3.34	.02	.29
Severity	-19.86	7.66	-.79	-2.59*					
Age	-.10	.18	-.16	-.53					
Model 3						.71	3.30	.14	1.91
Severity	-22.06	7.22	-.87	-3.05*	-.74*				
Age	1.37	1.08	2.26	1.27	.05				
Age^2	-.02	.01	-2.47	-1.38	.02				

*$p < .05$. **$p < .01$.

Table 13

Hierarchical Regression Results for NAB Scores at ≥12 Months Post-Injury (N = 58)

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
NAB Total (S-NAB)									
Model 1						<.01	.02		
Severity	-.57	4.44	-.02	-.13					
Model 2						.02	.42	.02	.82
Severity	1.00	4.78	.03	.21					
Age	.11	.13	.13	.90					
Model 3						.03	.48	.01	.62
Severity	1.41	4.82	.04	.29	-.02				
Age	.56	.58	.64	.96	.12				
Age^2	.00	.01	-.51	-.79	.10				
NAB Attention									
Model 1						<.01	.06		
Severity	1.14	4.53	.03	.25					
Model 2						.03	.70	.03	1.34
Severity	3.18	4.85	.09	.66					
Age	.15	.13	.17	1.16					
Model 3						.04	.76	.02	.88
Severity	3.68	4.89	.11	.75	.03				
Age	.69	.59	.77	1.17	.13				
Age^2	-.01	.01	-.61	-.94	.11				
NAB Memory									
Model 1						.01	.49		
Severity	-2.89	4.14	-.09	-.70					
Model 2						.09	2.61	.08	4.71*
Severity	.65	4.33	.02	.15					
Age	.24	.11	.30	2.17*					
Model 3						.09	1.80	<.01	.23
Severity	.87	4.39	.03	.20	-.09				
Age	.48	.51	.60	.94	.29*				
Age^2	.00	.01	-.30	-.48	.27*				
NAB Executive									
Model 1						.03	1.66		
Severity	-5.55	4.30	-.17	-1.29					
Model 2						.04	1.26	.02	.87
Severity	-3.98	4.63	-.12	-.86					
Age	.11	.12	.13	.93					
Model 3						.05	.90	<.01	.22
Severity	-3.74	4.69	-.11	-.80	-.17				
Age	.37	.57	.43	.66	.18				
Age^2	.00	.01	-.30	-.47	.16				

* $p < .05$. ** $p < .01$.

Hypothesis 2: Emotional outcome

The dataset was first analyzed with the original non-missing cases (N = 44) before multiple imputation. Overall, severity, time post-injury, and age (Model 3 in Table 14) did not explain for a significant proportion of the variance in BDI-II and BAI scores. Together, these variables only accounted for 16% of the variance of BDI-II scores and 28% of the variance of BAI scores. Hierarchical regression for both the BDI-II and BAI revealed that injury severity was a significant predictor of scores in Model 3. Specifically, participants with less severe injuries tended to have higher BDI-II and BAI scores. In contrast, time post-injury did not significantly predict outcomes for either BDI-II or BAI. In terms of age, it was found that there was no effect of age after accounting for injury severity and time post-injury for BDI-II nor did age make any significant contribution in predicting BDI-II scores (Model 3 in Table 14). In contrast, when age was entered into the model for the BAI, it accounted for a significant proportion of variance above and beyond severity and time post-injury. Specifically, age explained for 7% of the total variance of BAI scores and was found to be a significant predictor of BAI scores in Model 3, although this was non-significant when examining the structure coefficient, which is not influenced by the context of other variables entered and thus a better estimate. In terms of a nonlinear relationship between age and emotional outcomes, results revealed that for both BDI-II and BAI, adding the quadratic age variable did not account for a significant portion of variance nor was it a significant predictor of the overall model.

Table 14

Hierarchical Regression Results for BDI-II & BAI (Original Data, N = 44)

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
BDI-II									
Model 1						.13	6.04*		
Severity	-9.44	3.84	-.35	-2.46*					
Model 2						.13	2.96	<.01	.03
Severity	-9.59	4.00	-.36	-2.40*					
Time Post-injury	.01	.07	.02	.16					
Model 3						.16	2.58	.04	1.71
Severity	-11.64	4.26	-.44	-2.73**					
Time Post-injury	-.01	.07	-.01	-.08					
Age	-.14	.11	-.21	-1.31					
Model 4						.16	1.91	<.01	.09
Severity	-11.56	4.32	-.43	-2.68**	-.36**				
Time Post-injury	-.01	.07	-.01	-.07	-.06				
Age	.00	.46	.00	.00	-.03				
Age^2	.00	.00	-.21	-.30	-.05				
BAI									
Model 1						.20	10.45**		
Severity	-10.42	3.23	-.45	-3.23**					
Model 2						.21	5.33**	.01	.37
Severity	-9.95	3.34	-.43	-2.98**					
Time Post-injury	-.04	.06	-.09	-.61					
Model 3						.28	5.13**	.07	3.96*
Severity	-12.48	3.47	-.53	-3.60**					
Time Post-injury	-.06	.06	-.14	-.98					
Age	-.17	.09	-.30	-1.99*					
Model 4						.28	3.87**	<.01	.34
Severity	-12.36	3.50	-.53	-3.53**	-.45**				
Time Post-injury	-.06	.06	-.14	-.96	-.19				
Age	.04	.38	.07	.11	-.05				
Age^2	.00	.00	-.38	-.58	-.07				

* $p < .05$. ** $p < .01$.

Pooled results from the multiple imputed datasets (N = 67) differed from analyses with the original data (Table 15). Hierarchical regression revealed that severity was no longer a significant predictor of BDI-II and BAI scores, accounting for only 3% and 6% of the variance respectively. Neither time post-injury nor age significantly contributed to the variance explained or were predictors of the model. Specifically, age only explained an extra 4% and 2% of the variance for BDI-II and BAI when entered into the model. The quadratic age variable also did not significantly account for any variance when entered into the model (Model 4 in Table 15), suggesting that a non-linear relationship between age and emotional outcomes do not exist. Overall, severity, time post-injury, and age together did not account for a significant proportion of variance for either BDI-II (7%) or BAI (9%) scores.

Table 15

Hierarchical Regression Results for BDI-II & BAI (Multiple Imputation Pooled Data, N = 67)

	B	SE	*t*	$\boldsymbol{r}_s$	R^{2a}	F^b	ΔR^{2a}	ΔF^b
BDI-II								
Model 1					.03	NS		
Severity	-10.37	10.26	-1.01					
Model 2					.03	NS	<.01	NS
Severity	-10.51	10.31	-1.02					
Time Post-injury	.02	.21	.10					
Model 3					.07	1 Sig	.04	2 Sig
Severity	-14.39	13.27	-1.08					
Time Post-injury	.00	.22	.01					
Age	-.27	.42	-.65					
Model 4					.08	NS	.01	NS
Severity	-14.41	13.54	-1.06	-.148				
Time Post-injury	.00	.21	.02	<.01				
Age	-.30	1.41	-.21	-.07				
Age^2	.00	.01	.02	-.08				
BAI								
Model 1					.06	2 Sig	.06	

	B	SE	*t*	r_s	R^{2a}	F^b	ΔR^{2a}	ΔF^b
Severity	-13.16	10.26	-1.28					
Model 2					.07	1 Sig	.01	NS
Severity	-12.98	9.61	-1.35					
Time Post-injury	-.02	.20	-.12					
Model 3					.09	1 Sig	.02	NS
Severity	-13.58	12.28	-1.11					
Time Post-injury	-.03	.21	-.13					
Age	-.04	.31	-.14					
Model 4					.11	1 Sig	.02	NS
Severity	-13.00	12.49	-1.04	-.23				
Time Post-injury	-.05	.20	-.22	-.05				
Age	.71	1.10	.65	.07				
Age^2	-.01	.01	-.77	.04				

Note: β not available for pooled data

[a]Average R^2 value from the 5 imputed datasets; [b]Number of imputations (out of 5) with significant *F* values; Sig = significant; NS = non-significant

* $p < .05$. ** $p < .01$.

When the pooled imputed data was analyzed separately for the mild complicated-moderate TBI group and the severe TBI group, the results were consistent with the analyses above (Table 16, 17). Specifically, time post-injury and age did not account for a significant proportion of variance in either emotional outcome variable. Age explained only an extra 1% of the variance for BDI-II and BAI beyond time post-injury for mild complicated-moderate TBI and an extra 2% for severe TBI. No significant contribution was made by the quadratic age variable for both severity levels, suggesting no non-linear relationship between age and emotional outcome within each severity category.

Table 16

Hierarchical Regression Results for BDI-II & BAI - Mild Complicated-Moderate Severity (Multiple Imputation Pooled Data, N = 40)

	B	SE	*t*	r_s	R^{2a}	F^b	ΔR^{2a}	ΔF^b
BDI-II								
Model 1					.02	NS		
Time Post-injury	.08	.35	.24					
Model 2					.06	NS	.04	NS
Time Post-injury	.06	.37	.16					
Age	-.25	.47	-.54					
Model 3					.07	NS	.01	NS
Time Post-injury	.04	.37	.11	.06				
Age	.08	1.78	.04	-.15				
Age^2	.00	.02	-.20	-.15				
BAI								
Model 1					.05	1 Sig		NS
Time Post-injury	-.10	.39	-.25					
Model 2					.11	1 Sig	.06	2 Sig
Time Post-injury	-.11	.41	-.28					
Age	-.18	.37	-.48					
Model 3					.12	1 Sig	.01	NS
Time Post-injury	-.14	.40	-.35	-.07				
Age	.34	1.29	.26	-.12				
Age^2	-.01	.01	-.46	-.13				

Note: β not available for pooled data

[a]Average R^2 value from the 5 imputed datasets; [b]Number of imputations (out of 5) with significant *F* values; Sig = significant; NS = non-significant

* $p < .05$. ** $p < .01$.

Table 17

Hierarchical Regression Results for BDI-II & BAI - Severe Severity (Multiple Imputation Pooled Data, N = 27)

	B	SE	*t*	r_s	R^{2a}	F^b	ΔR^{2a}	ΔF^b
BDI-II								
Model 1					<.01	NS		
Time Post-injury	-.02	.22	-.09					
Model 2					.04	NS	.03	NS
Time Post-injury	-.03	.23	-.15					
Age	-.31	.54	-.58					
Model 3					.06	NS	.02	NS
Time Post-injury	-.02	.23	-.11	-.02				
Age	-1.49	2.90	-.52	-.14				
Age^2	.02	.03	.43	-.12				
BAI								
Model 1					.01	NS		
Time Post-injury	.02	.21	.10					
Model 2					.05	NS	.04	NS
Time Post-injury	.03	.21	.16					
Age	.30	.49	.61					
Model 3					.07	NS	.02	NS
Time Post-injury	.03	.21	.14	.03				
Age	.79	2.87	.28	.15				
Age^2	-.01	.03	-.18	.14				

Note: β not available for pooled data

[a]Average R^2 value from the 5 imputed datasets; [b]Number of imputations (out of 5) with significant *F* values; Sig = significant; NS = non-significant

* $p < .05$. ** $p < .01$.

Analyses conducted with groups separated by time post-injury revealed that Age and Age^2 did not significantly predict BDI-II or BAI scores or contribute to the model regardless of whether participants were assessed before 12 months post-injury (Table 18) and at least 12 months after injury (Table 19). Furthermore, no relationship between injury severity and BDI-II and BAI scores were found irrespective of time post-injury.

Table 18

Hierarchical Regression Results for BDI-II & BAI Scores at <12 Months Post-Injury (Multiple Imputation Pooled Data; N = 8)

	B	SE	*t*	r_s	R^{2a}	F^b	ΔR^2	ΔF^b
BDI-II								
Model 1					.02	NS		
Severity	3.34	23.78	.14					
Model 2					.26	NS	.23	NS
Severity	-3.60	24.02	-.15					
Age	-.62	.72	-.86					
Model 3					.34	NS	.01	NS
Severity	-2.74	27.42	-.10	.08				
Age	-1.19	4.04	-.30	-.45				
Age^2	.01	.04	.14	-.43				
BAI								
Model 1					.20	NS		
Severity	-22.40	23.50	-.95					
Model 2					.43	NS	.23	1 Sig
Severity	-28.28	24.51	-1.15					
Age	-.523	.506	-1.03					
Model 3					.51	NS	.08	NS
Severity	-30.8	27.42	-1.12	-.44				
Age	1.16	3.52	.33	-.32				
Age^2	-.02	.04	-.47	-.33				

Note: β not available for pooled data

[a]Average R^2 value from the 5 imputed datasets; [b]Number of imputations (out of 5) with significant *F* values; Sig = significant; NS = non-significant

* $p < .05$. ** $p < .01$.

Table 19

Hierarchical Regression Results for BDI-II & BAI scores at >12 Months Post-Injury (Multiple Imputation Pooled Data; N = 59)

	B	SE	*t*	$\boldsymbol{r}_s$	R^{2a}	$\boldsymbol{F}^b$	$\boldsymbol{\Delta R}^2$	$\boldsymbol{\Delta F}^b$
BDI-II								
Model 1					.04	NS		

		B	SE	*t*	r_s	R^{2a}	F^b	ΔR^2	ΔF^b
	Severity	-12.76	11.14	-1.15					
	Model 2					.07	1 Sig	.03	1 Sig
	Severity	-15.69	15.12	-1.04					
	Age	-.20	.49	-.41					
	Model 3					.08	NS	.01	NS
	Severity	-15.64	15.55	-1.01	-.18				
	Age	-.14	1.66	-.08	-.03				
	Age^2	<0.01	.02	-.04	-.03				
BAI									
	Model 1					.05	1 Sig		
	Severity	-12.16	10.69	-1.14					
	Model 2					.07	1 Sig	.02	NS
	Severity	-11.18	13.13	-.85					
	Age	.07	.31	.22					
	Model 3					.09	1 Sig	.02	1 Sig
	Severity	-10.38	13.57	-.77	-.21				
	Age	.92	1.29	.72	-.12				
	Age^2	-.01	.01	-.74	.09				

Note: β not available for pooled data

[a]Average R^2 value from the 5 imputed datasets; [b]Number of imputations (out of 5) with significant *F* values; Sig = significant; NS = non-significant

* $p < .05$. ** $p < .01$.

Hypothesis 3: Moderation of age between cognitive and emotional outcomes

The relation between cognitive and emotional outcomes was first examined. When analyzed with the original data (Table 20), a significant relation between the BAI and S-NAB was found (r_s = -.25, p = .05) while the relation between BDI-II and S-NAB was marginally significant (r_s = -.23, p = .07). Specifically, participants with higher NAB total scores had lower BAI and BDI-II scores (i.e., those with better cognitive outcomes had better emotional outcomes). In contrast, the pooled imputed data revealed no relation between BDI-II (r_s = -.18, p = .24) and BAI (r_s = .01, p = .47) with NAB total scores

(Table 21). Nevertheless, the moderation analysis was conducted as an interaction may be observed without the main effects.

Analyses with both the original and pooled imputed data revealed that age did not significantly moderate the relation between cognitive and emotional outcomes (Model 3 in Tables 20, 21). Specifically, no additional variance was explained by the NABxAge interaction term in predicting either the BDI-II or BAI. The final model explained only 6% of the variance for BDI-II and 7% for BAI when using the original data (Table 20). Hence, the results suggest that the relation between NAB total and BDI-II/BAI scores did not vary by age.

Table 20

Hierarchical Regression Results for Moderation of Age between Cognitive and Emotional Outcomes (Original data, N = 44)

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
BDI-II									
Model 1						.05	.14		
NAB Total	-.21	.14	-.23	-1.52					
Model 2						.05	1.13	<.01	<.01
NAB Total	-.21	.14	-.23	-1.50					
Age	.01	.11	.01	.06					
Model 3						.06	.81	.01	.22
NAB Total	-.22	.14	-.24	-1.54	-.23				
Age	.01	.11	.01	.09	.01				
NAB X Age	<.01	.01	.07	.47	.04				
BAI									
Model 1						.04	2.65		
NAB Total	-.19	.12	-.25	-1.63					
Model 2						.06	1.29	<.01	<.01
NAB Total	-.19	.12	-.25	-1.61					
Age	.01	.09	.01	.06					
Model 3						.07	.45	<.01	.16
NAB Total	-.18	.12	-.24	-1.52	-.25*				
Age	<.01	.09	.01	.04	.01				

	B	SE	β	*t*	r_s	R^2	*F*	ΔR^2	ΔF
NAB X Age	<.01	.01	-.06	-.40	-.10				

Note. NAB total, Age, and NAB X Age interaction are centered values

* $p < .05$. ** $p < .01$.

Table 21

Hierarchical Regression Results for Moderation of Age between Cognitive and Emotional Outcomes (Multiple Imputation Pooled Data, N = 67)

	B	SE	*t*	$\boldsymbol{r}_s$	R^2	*F*	ΔR^2	ΔF
BDI-II								
Model 1					.06	3 Sig, 2 NS		
NAB Total	-.35	.55	-.63					
Model 2					.08	2 Sig, 3 NS	.02	NS
NAB Total	-.33	.54	-.62					
Age	-.10	.33	-.31					
Model 3					.09	1 Sig, 4 NS	.01	NS
NAB Total	18.71	8.32	2.25	-.18				
Age	-.33	.53	-.62	-.07				
NAB X Age	-.10	.33	-.31	.06				
BAI								
Model 1					<.01	NS		
NAB Total	.03	.30	.09					
Model 2					.02	NS	.02	NS
NAB Total	.01	.29	.05					
Age	.11	.24	.47					
Model 3					.04	NS	.02	NS
NAB Total	<.01	.30	.01	.01				
Age	.11	.24	.47	.08				
NAB X Age	-.01	.01	-.64	-.10				

Note. NAB total, Age, and NAB X Age interaction are centered values; β not available for pooled data

[a]Average R^2 value from the 5 imputed datasets; [b]Number of imputations (out of 5) with significant *F* values; Sig = significant; NS = non-significant

* $p < .05$. ** $p < .01$.

While such a relation was not significant on statistical testing, graphs of the interaction revealed that there appears to be a trend towards an interaction when observed with both the original data (Figure 2, 3) and pooled imputed data (Figure 4, 5). Specifically, it appears that the relation between the BDI-II and S-NAB is stronger for younger than older adults, consistent with the hypothesis (Figure 2, 4). The opposite pattern is observed with the BAI, with the relation between S-NAB and BAI scores more prominent for older than younger adults (Figure 3). Furthermore, it appears that older adults tend to have higher BAI scores with increasing S-NAB scores whereas younger adults tend to have lower BAI scores with increasing S-NAB scores when the pooled imputed data are observed (Figure 5). Despite observation of these trends from the plots, it is important to keep in mind that none of these trends were statistically significant.

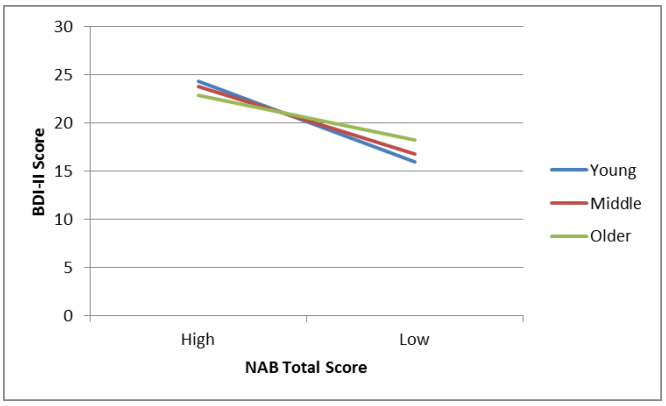

Figure 2: Relation between S-NAB and BDI-II score by age groups (Original data, *N* = 44).

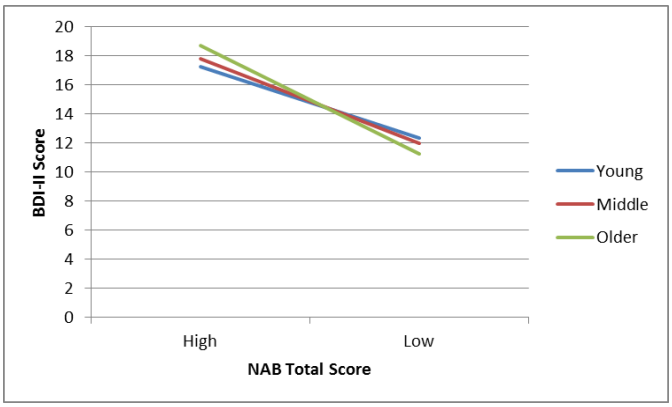

Figure 3: Relation between S-NAB and BAI score by age groups (Original data, *N* = 44).

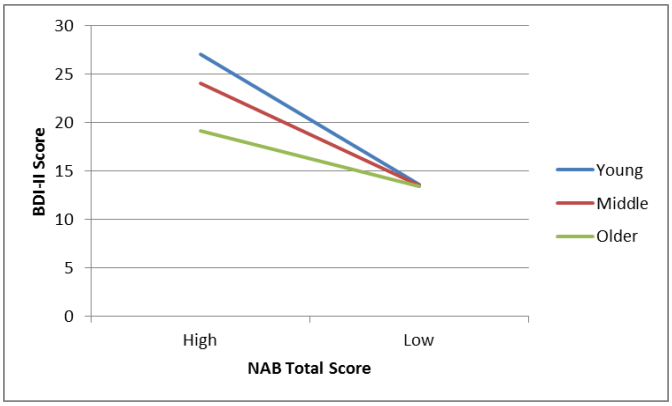

Figure 4: Relation between S-NAB and BDI-II score by age groups (Multiple Imputation Pooled Data, *N* = 67).

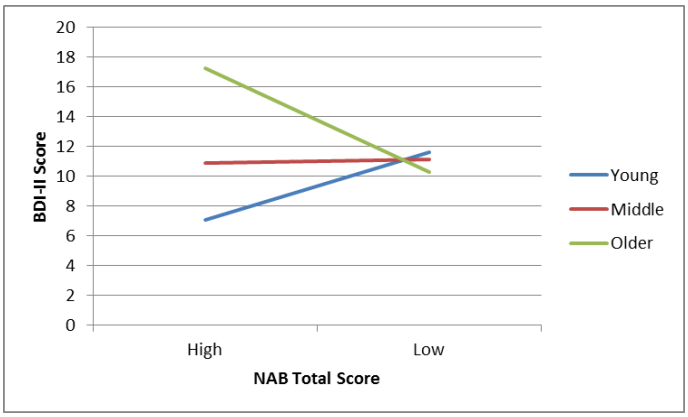

Figure 5: Relation between S-NAB and BAI score by age groups (Multiple Imputation Pooled Data, *N* = 67).

VI. DISCUSSION

Summary of findings

The relation between post-TBI cognitive and emotional outcomes and age at time of injury has been poorly described in the literature. The goal of the present study was firstly to examine post-TBI cognitive and emotional outcomes across the adult lifespan and subsequently to examine whether age moderates the relation between cognitive and emotional outcomes. It was predicted that there would be differences in outcomes between adults who were older and younger at the time of the injury and specifically that a nonlinear relation would emerge between age and outcomes. Moreover, it was hypothesized that the relation between cognitive and emotional outcome, if one existed, would vary according to age such that cognitive and emotional outcomes would be related for adults who were younger at time of injury but not for adults who were older at time of injury. Overall, the results of the analyses failed to support any of the study hypotheses. No relation (linear or nonlinear) was found between age and cognitive and emotional outcomes nor did age moderate the relation between cognitive and emotional outcomes. This was found regardless of whether injury severity and time post-injury were taken into account and regardless of whether the relation was examined within injury severity and time post-injury categories or across these categories.

In terms of emotional outcome, the results revealed some discrepancy between the analyses with the original and pooled multiple imputed datasets which warrants further discussion as it raises some uncertainty in determining which results represent the true population values. If analyzed with the original dataset with case deletion of missing data, age was related to anxiety symptoms. In contrast, analysis with the pooled datasets after multiple imputation revealed that age did not predict emotional outcomes. As

discussed, by pooling multiple imputed datasets, the multiple imputation technique should produce little error while retaining important parameter estimates of the sample (Graham, 2009). In contrast, the analyses with the original dataset excluding the missing cases are likely biased as the analyses are using only a subset of the sample. As discussed, listwise case deletion assumes and provides only valid inferences when the data is MCAR (Allison, 2002), which is untrue for the current study. The finding that the data is MAR for age implies that the participants with missing data and excluded in the analysis were younger at time of injury, and when discarded cases differ from the rest, estimates will be biased (Allison, 2002). Because of these reasons, the analyses with the pooled imputed data are more likely to represent the true population values more than the analyses with the original data. Hence, of the two conclusions suggested by the original versus the imputed data analyses, it is more likely that age at time of injury is not a significant predictor of anxiety symptoms for the current study.

Aside from the main hypotheses, injury severity and time post-injury were also examined in the model as they have been shown to be significant predictors of post-TBI outcome in previous research. When the data was analyzed as a whole, the current study did not find severity and time post-injury to be related to cognitive and emotional outcomes. However, severity was found to be significantly related to cognitive outcomes in those evaluated less than 12 months after injury. Specifically, severity predicted cognitive outcomes acutely after an injury (<12 months), with participants sustaining more severe injuries having poorer cognitive outcomes than other participants who also were assessed less than a year post-injury. This is not inconsistent with the literature. Green et al. (2008) reported that GCS scores are associated with cognitive outcomes

measured less than 12 months post-injury but were not predictive of outcomes in the long-term.

Potential explanations of the findings

The findings of the present study are consistent with some of the pertinent literature reviewed but discrepant with others. Like Green et al. (2008), Senathi-Raja et al. (2010b) and Levine et al. (2010), no age effect on cognitive and emotional outcome was found in the current analyses. In contrast, other studies examining the effect of age on cognitive (Senathi-Raja et al., 2010a) and emotional (Deb & Burns, 2007; Rapoport & Feinstein, 2001, Rapoport et al., 2003a, Whelan-Goodinson et al., 2010) outcomes found that adults who were older at the time of injury tended to have worse cognitive performance but better emotional functioning than adults who were younger at injury. Such findings warrant in-depth consideration of the differences between the current and previous research as well as the possible reasons underlying the current results.

The current findings are unlikely to be due to differences in demographic characteristics in the sample as education and gender were equally represented between age groups. While small differences between occupation and marital status existed between age groups, these differences were not related to cognitive and emotional outcomes. Similarly, differences in injury severity between age groups cannot account for the findings, as analyses were also conducted within severity groups. In addition, operationalization of age groups in this study (e.g., older adults defined as $\geq$ 50 years old) cannot account for the findings as the actual analyses involved regression analyses and used age as a continuous variable, thus eliminating any artificial division due to age. Furthermore, pre-injury characteristics (e.g., history of neurological disease, previous TBIs) were controlled for by excluding participants with these comorbid problems; thus

no differences existed between age groups. Similarly, excluded participants were equally distributed across age groups. While research has found that older adults sustaining a TBI have more comorbid medical problems than younger adults, which partly contribute to their worse functional outcomes and mortality (Kinsella, 2011), this was not the case in the present study. Older adults were not more likely to be excluded due to premorbid medical conditions than younger adults in the study, and therefore bias due to exclusion of less well older adults cannot explain the null findings.

When exploring possible explanations for the findings, it is important to not only consider the injury and neurological mechanisms that may be underlying the findings, but also other non-neurological factors that may be contributing. Indeed, researchers such as Suhr & Gunstad (2002) remind readers that "neuropsychological tests assess behavior and are not a direct measure of brain function" (p.448) and emphasize that psychological factors should also be considered. Such factors may include the effects of diagnosis threat (Suhr & Gunstad, 2002; Suhr & Gunstad, 2005), implicit beliefs and expectancies (Mittenberg, DiGiulio, Perrin, & Bass, 1992), and secondary gain (Binder & Rohling, 1996) on neuropsychological test performance. For example, Suhr & Gunstad (2002) found that participants for whom the diagnosis was made salient (i.e., the “diagnosis threat” group) performed worse than a control group on measures of attention, working memory, psychomotor speed, and memory, which was not accounted for by poorer effort or increased anxiety in the diagnosis threat group. While it is unknown whether such an effect played a role in determining the current findings, this example illustrates the complexity of brain-behavior relationships and demonstrates how other unmeasured variables may be contributing to participants’ performance. Neuropsychological

performance is not a direct mapping of brain integrity, but represents a complex behavior that involves a multitude of "organic" and psychological factors (Suhr & Gunstad, 2002).

Several potential explanations are plausible in elucidating the results and require further consideration. One of the major differences between the present study and previous studies cited is the type of population used and the original purpose of assessment. Specifically, the current study involved a retrospective examination of data collected from individuals assessed in a litigation or compensation setting to which they were originally referred for determination of post-injury benefits. In contrast, all but one (Rohling et al., 2001) of the studies reviewed utilized participants recruited from hospital databases, outpatient clinics, or the general community for research purposes. Although this does not necessarily preclude the possibility that these participants was also involved in litigation as a result of their injury (i.e., patients may be seen in an outpatient TBI clinic while also undergoing litigation assessments elsewhere), it is uncertain whether and how many of these samples included participants who were undergoing such processes at the time of their assessment. The present study is unique in that all participants were confirmed to be part of the process of acquiring benefits. Furthermore, even if participants in the studies with hospital or outpatient clinic recruitment were active in the litigation process, it is unclear whether their performance on neuropsychological testing would be consistent across testing situations (e.g., research versus compensation settings). A plethora of literature exists on the effects of litigation on post-TBI recovery and symptom maintenance that may assist in explaining the findings. It should be noted, however, that most of these studies involve mild uncomplicated TBI whereas the current study involved mild complicated to severe TBI.

Research has shown that individuals involved in litigation generally have a slower recovery and are more likely to report experiencing post-TBI symptoms such as headaches, fatigue, and attentional and memory difficulties (i.e., symptoms of post-concussive syndrome; Iverson, Lange, Brooks, & Rennison, 2010; McKinlay, Brooks, & Bond, 1983). Furthermore, not only do litigants report more post-concussive symptoms, but these symptoms tend to last longer, be more debilitating, and result in more psychological distress (Wood & Rutterford, 2006). Echoing this issue, the World Health Organization stated that the most consistent predictor of continued symptoms after a mild TBI is the presence of compensation (Sweet, Goldman, & Breting, 2013). Aside from the above cited research reporting increased subjectively endorsed post-TBI symptoms of litigants, there is also research conducted on the effects of litigation on neuropsychological testing, which has shown that financial incentives and litigation can have a profound effect on neuropsychological performance. In a widely cited meta-analysis, Binder & Rohling (1996) reported that the effect of litigation has a medium effect size on neuropsychological performance in TBI of mixed severity (d = .47). However, this meta-analysis did not exclude those who exerted suboptimal effort on SVTs, and thus has less relevance for the current study, which included only participants who were in litigation and were performing adequately on SVTs.

Unfortunately, few studies have examined the effects of a compensation-referral context in a population of litigating individuals who have not failed SVTs. However, the few studies conducted have found that non-malingering litigants perform similarly on neuropsychological testing to those who are not seeking compensation, despite reporting longer lasting and more severe symptoms. This was found for both severe TBI (Wood &

Rutterford, 2006; McKinlay et al., 1983) and mild TBI (Suhr, Tranel, Wefel, & Barrash, 1997). Hence, it is unlikely that litigation status alone can explain the current findings.

An alternative explanation of the findings also relates to the nature of the original referral. Specifically, it is possible that because the purpose of the original referrals was for determination of benefits, individuals with continuing cognitive or emotional difficulties were more likely to attend such assessments than those not requiring further treatment or benefits (i.e., those who have fully recovered and not experiencing any difficulties in everyday life). For example, an individual who sustained a TBI of mild complicated severity 3 years ago may attend the assessment due to continuing symptoms, even though most individuals with milder injuries recover within a few months to a year. Such issues of selection bias in TBI research have been previously voiced in the literature. The argument has been made that samples included in TBI research may be much different than the true TBI population (Luoto, Tenovuo, Kataja, Brander, Öhman, & Iverson, 2013). Thus, those individuals who have continuing symptoms, regardless of the severity or time post-injury, are more likely to receive and attend the assessment in the first place than those who no longer experience symptoms. In other words, it may be that the sample in this study may not represent the true population of all TBI patients, but instead is a subset of those who have continuing and are seeking compensation. This may contribute to the null findings as variability of the sample is possibly reduced and skewed to reflect those lower functioning individuals.

Aside from the context of the assessment, another potential explanation of the current results revolves around the literature reporting age differences in mortality rates. Research has suggested that there is a higher mortality rate in older adults sustaining

severe TBIs than in younger adults (McIntyre, Mehta, Aubut et al., 2013). This is reflected in a similar study, in which the deceased group not included in the study tended to be older than those included in the study (Senathi-Raja et al., 2010a). This disproportion in mortality rates between older and younger adults raises the possibility that the older adults who survived and are included in the study are different than those who succumbed to their injuries. For example, those older adults who survive after sustaining a severe injury may be a subsample of the population of older adults. This subgroup may be more resilient, have greater cognitive reserve, or have better recovery ultimately than the group who died from similar injuries had they lived. Similarly, it is possible that older adults with greater cognitive impairment did not receive the NAB during the original assessment but rather an easier measure (e.g., the Dementia Rating Scale; Mattis, 1988). This practice inadvertently would result in selection of higher functioning older adults for inclusion in the current study.

Definitions of the severity of injury may also contribute to the present findings. Injury severity in the current study was categorized based primarily from GCS data, with a few cases utilizing neuroimaging data in cases in which GCS scores were unavailable. Although older and younger adults may have equivalent GCS scores, this does not necessarily equate to similar force of the initial impact. In other words, injury severity in this study referred to depth of coma after the impact and was not equivalent to the actual severity of the impact itself. Perhaps older adults who sustain less severe blows to the head have GCS scores similar to younger persons with more severe blows. In other words, older adults may require less impact to the head to result in the same GCS score. This is possible given the age-related cerebral changes that contribute to their increased

susceptible to secondary complications and hemorrhaging (Kinsella, 2001). Without any way of measuring the initial mechanical impact to the head, it is unclear whether younger and older adults had similar initial injury severities.

Overall, the results of the present research diverge from some of the previous research on the effects of injury severity, time post-injury, and age on post-TBI outcome. However, several differences exist between present and past research with the current inquiry having some characteristics either not present or not made explicit in previous studies. One possible explanation of the null findings lies in the selection bias of the sample, such that those with continuing difficulties in everyday life are more likely to be referred and attend the original assessment. Furthermore, the effects of litigation status, age differences in mortality, and injury non-equivalence are unlikely to play a great role in explaining the results but cannot be completely ruled out.

Strengths & limitations

Methodologically, the present study improves upon previous relevant research in several ways. While the present study did not recruit an age-matched comparison group, the use of standardized norms accounting for age fulfills the requirement of controlling for normal age-related changes in cognition. Many variables thought to be related to cognitive and emotional outcomes and testing performance (e.g., psychiatric history, previous TBIs, poor English fluency, time post-injury) were controlled for by either entering the variable into the model or excluding it from the analyses. Another strength of the current study, as mentioned, is the specification of whether the *age* used in the analyses refers to age at the time of injury or at the time of assessment. The choice of specification has implications for the results as the two definitions of *age* cannot be used interchangeably. The current study is also unique in terms of the measures used. As

discussed, the NAB is an optimal measure for this population, as it covers a broad age range, contains TBI normative data and co-normed modules, and has strong psychometric properties in this population. Finally, the use of self-report questionnaires for depression and anxiety has the strength over using diagnostic categories as it allows one to capture subclinical levels of depression and anxiety, which may still be clinically significant. Since older adults are thought to have higher rates of subclinical depression and anxiety, the BAI and BDI are likely to be more sensitive than diagnostic categories determined by interview.

Despite these strengths, the present research also has limitations that must be considered when making inferences about the findings. The present study used a retrospective design instead of prospective. While there are no doubt benefits of using such a design (e.g., greater sample size, lack of experimenter bias as the outcome of interest was not related to the current purpose of the assessment), using such a design limits the amount of rigor and experimental control. Because the original purpose of the assessments was for forensic and not research purposes, unmeasured variables in the testing environment could not be controlled for and may not be consistent between participants, thus potentially confounding the results. For example, pain and fatigue are commonly observed after a TBI and have been shown to affect performance on neuropsychological testing (Hart, Martelli, & Zasler, 2000). Because of the nature of the assessments, examinees may have had differing levels of pain and fatigue, received different numbers and lengths of breaks, and been affected differently by these factors during testing. These variables were unmeasured and may have confounded the current findings if related to age. In addition, the collection of data was limited to what was

available, and in some cases in which more information was necessary or missing (e.g., BDI-II), the original participants could not be contacted for research purposes.

Another weakness of the study is that the effect of general trauma arising from physical injury could not be controlled. The focus of the current study was the effects of the TBI on cognitive and emotional outcomes, and not just the effect of sustaining any injury. Research has documented that individuals with orthopedic injuries score approximately 0.5 standard deviations lower on neuropsychological testing than healthy controls (Larrabee, Binder, Rohling, & Ploetz, 2013). This may be due to a multitude of factors after an injury, including general trauma effects and pre-morbid difficulties that made the individuals susceptible to sustaining an injury (Larrabee et al., 2013). Currently, the "gold-standard" for this type of research in the literature is to use an orthopedic control group (i.e., individuals with traumatic injuries excluding head injuries) in addition to a healthy comparison group in order to separate the effects of a TBI from general injury effects (Larrabee et al., 2013). However, the majority of previous research with TBI surprisingly does not follow these standards. Indeed, of the studies reviewed here, only one (Kinsella et al., 2014) used an orthopedic comparison group. This may be due to the issues inherent in obtaining such a group rather than ignorance of the problem. Much of the time, it may be difficult to obtain a "pure" orthopedic comparison group as head injury and other bodily injuries frequently co-exist in accidents. Thus, while the current study did not employ these methods, this is not better or worse than the majority of the relevant extant literature in which such a comparison group is lacking.

Finally, the measures used in this study, while having good psychometric properties, also are limited in some ways. The NAB may be limited in generalizing to

everyday cognitive functioning (i.e., ecological validity). Specifically, neuropsychological testing took place in a structured testing environment that may not reflect cognitive functioning in daily life. This is a common but important concern about neuropsychological testing in both clinical and research settings and it is not specific to the NAB. The BDI-II and BAI, while sensitive to subclinical levels of depression and anxiety to be measured, also have limitations. Specifically, no informant report for emotional outcomes was collected and emotional outcomes were based solely on single reports of the patient. As discussed, this may pose as an issue as blunted awareness and decreased insight of post-TBI symptoms are common problems after moderate to severe head injury (Flashman & McAllister, 2002).

Future directions & implications

The present research endeavored to elucidate the pattern and magnitude of the relation between age, cognitive outcome, and emotional outcome after a TBI. While some interesting findings were obtained, further research is necessary to increase our understanding of these relationships. Future studies should investigate these questions using a prospective sample in order to gain more control over extraneous variables. Furthermore, both orthopedic and healthy age-matched comparison groups should be recruited in order to make greater inferences about whether any differences observed are due to the effects of TBI and age or to normal age-related changes or general trauma effects. Studies conducted across a greater spectrum of settings (e.g., private practice, hospital) will help rule out whether the results found in the present investigation are unique to this compensation-based private practice setting or span across settings.

Despite the limitations of this study, the current findings carry implications for the greater health field. There is evidence that older adults sustaining TBIs are treated more

conservatively than younger adults in hospitals. Indeed, research has found that older adults receive lower intensity of care, fewer admissions to the ICU and transfers to appropriate trauma units, and less monitoring and specialty care (Lane, Sorondo, & Kelly, 2003; Thompson, Rivara, Jurkovich, Wang, Nathens, & MacKenzie, 2008). While it is uncertain why older adults are receiving this differential treatment, one likely factor involves beliefs regarding poorer recovery in older adults. Specifically, both health-care and family decision-makers may be influenced by age-related stereotypes that older adults inevitably will have poor outcomes after a TBI. Such beliefs may stem from the general pessimistic view portrayed of aging in society as well as research suggesting poorer functional outcomes and increased mortality in older adults. While it is true that older adults have a higher post-TBI mortality rate than younger adults, the current research shows that those who survive tend to recover cognitively and emotionally to a degree similar to that of younger adults, when cognitive changes associated with normal aging are taken into account. It should not be assumed that intervention efforts may not be as beneficial to older adults. Such age stereotypes, by preventing the appropriate level of care and intervention, only hinder further recovery and perpetuate the cycle of negative beliefs and conservative care. It is hoped that the findings of the present research help challenge these stereotypes and inform treatment efforts for older adults sustaining a TBI.

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VITA AUCTORIS

NAME:	Kelly An
PLACE OF BIRTH:	Beijing, China
YEAR OF BIRTH:	1989
EDUCATION:	University of Toronto, B.Sc. (Honors) in Psychology, Toronto, ON, 2011
	University of Windsor, M.A. in Clinical Psychology, Windsor, ON, Candidate, 2014