The Neuropsychological Mechanisms of Avoidant Coping Post Traumatic Brain Injury

by

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A thesis submitted in conformity with the requirements

for the degree of Doctor of Philosophy

Graduate Department of Psychology

University of Toronto

Your file Votre référence
ISBN: 978-0-494-67664-6
Our file Notre référence
ISBN: 978-0-494-67664-6

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ABSTRACT

Many people who sustain traumatic brain injuries (TBI) have poor psychosocial outcomes. Previous research has indicated that poor outcomes are related to the use of avoidant coping following TBI, although the mechanisms of this relationship are not clear. The major pathological consequence of TBI is damage to the frontal lobes and/or their connections, resulting for most people in executive and/or affective dysfunction. The purpose of this dissertation study was to delineate the neuropsychological, psychiatric, personality and physiological mechanisms of avoidant coping following TBI. Controls and people with TBI completed the *Baycrest Psychosocial Stress Test (BPST)*, where coping behaviour was observed directly, and physiological measures were recorded. Participants also completed a neuropsychological test battery, and a series of questionnaires assessing coping, psychiatric status, personality and outcomes. There were no significant differences between groups in self reported coping, However, the control and mild TBI group engaged in more planful than avoidant behaviour on the BPST. As a group, individuals with moderate-to-severe injury, in contrast, engaged in more avoidant than planful behaviour. However, analysis of individual differences in coping behaviour within the moderate-to-severe group revealed a bimodal distribution, allowing classification of people in this group as ‘planners’, or ‘avoiders’ (this distribution was not evident in the mild TBI group). Within the moderate-to-severe group, planners had better executive function, were more reactive to stress (psychologically and physiologically), performed better on the speech task during the BPST, and had greater return to productivity. However, planners also had worse psychosocial outcomes as compared to the avoiders. This was the first study, to the author’s knowledge, to examine coping behaviour during a simulated real-world stress test. Results indicate that behavioural measures of coping, such as the BPST, are more sensitive to changes in coping post TBI than are self and significant other reported questionnaires. Results also demonstrate that executive function and psychological and physiological reactivity are important factors that contribute to coping following moderate-to-severe TBI. These data raise important questions about the challenges of targeting coping through rehabilitation.

ACKOWLEDGEMENTS

I would like to acknowledge and thank the people who have made the completion of this thesis possible;

… the people who took part in this study, for their willingness to donate their time and effort in the name of science

… my supervisors, Dr. Nicole Anderson and Dr. Donald Stuss, for being willing to give me the opportunity to study at this level, and for their support, mentorship, and encouragement

… my mentors, Dr. Alison Fleming, who introduced me to science, and Dr. Deirdre Dawson who inspired my interest in TBI and coping

… my thesis committee members, Dr. Adam Anderson and Dr. Jordan Peterson, for their insight and encouragement

… my colleagues at Baycrest, especially Dr. Malcolm Binns, for his statistical advice, and Scott Blackwood, Anoosh Chaudhry, Susan Gillingham, Christina Gojmerac, Roshan Guna, Candace Ikeda-Douglas, Constance Nguyen, Sandra Priselac, Jimmy Shen, and Sarah Waters-Schulte for helping with the execution and analysis of this project

… my colleagues at the University of Toronto Mississauga, Dr. Munmun Chatterjee and Dr. Diptendu Chatterjee, for helping me with the cortisol assays

… my parents, Michael and Eugenia Krpan, and my brother, Peter Krpan, for believing in me when I didn't, and sharing with me the highs and lows of this endeavour over the last several years

… and my best friend and husband, Vedran Lovic, who has inspired, encouraged and guided me from the very beginning of this process, through to the completion of this thesis

I would also like to thank the organizations that provided me with financial support throughout my doctoral work;

The Canadian Institute of Health Research
The Ontario Research Coalition
Toronto Rehabilitation Institute
Max and Ruth Wiseman, KLARU at Baycrest
Mary Gertrude I'Anson, The School of Graduate Studies, University of Toronto

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LIST OF ABBREVIATIONS

BPST	Baycrest Psychosocial Stress Test
BFAS	Big Five Aspects Scales
BDI	Beck Depression Inventory
DEX	Dysexecutive Questionnaire
DLPFC	Dorsolateral Prefrontal Cortex
ED	Extradimensional Shift
EDR	Extradimensional Shift Reversal
GCS	Glasgow Coma Scale
ID	Intradimensional Shift
IDR	Intradimensional Shift Reversal
IGT	Iowa Gambling Task
PCD	Post-Concussional Disorder
SART	Sustained Attention to Response Task
SIP	Sickness Impact Profile
SO	Significant Other
TBI	Traumatic Brain Injury
TMAS	Taylor Manifest Anxiety Scale
TMT	Trail Making Test
TSST	Trier Social Stress Test
VMPFC	Ventromedial Prefrontal Cortex
WCST	Wisconsin Card Sorting Test
WOC	Ways of Coping Questionnaire – Revised

STATEMENT OF PURPOSE

Many people who sustain traumatic brain injuries (TBI) have poor psychosocial outcomes. Previous research has indicated that poor outcomes are related to the use of avoidant coping following TBI, although the mechanisms of this relationship are not clear. The major pathological consequence of TBI is damage to the frontal lobes and/or their connections, resulting for most people in executive and/or affective dysfunction. I propose that executive and affective dysfunctions are the mechanisms by which avoidant coping relates to negative outcomes post TBI. In addition, I propose that there are important factors that moderate these relationships (e.g., psychiatric status, personality and responsivity to stress). The primary objective of this dissertation is to further investigate the mechanisms of avoidant coping following TBI, and relate them to objective outcomes. The primary hypothesis is that executive and affective dysfunction drive a common relation between avoidant coping and long-term negative outcomes following TBI. The thesis is divided into the following sections. In the introduction, a review of the relevant literature on TBI outcomes and coping is followed by a discussion of how the pathology and functional consequences of TBI may be important moderators of avoidant coping. The core of this thesis is the description of a series of three studies designed to elucidate the neuropsychological mechanism of outcomes following TBI, and variables that moderate theses relationships. The final section of this thesis is a general discussion of the data presented and its potential implications for rehabilitation.

CHAPTER ONE

General Introduction

Prevalence and Incidence of Traumatic Brain Injury

Recent medical and technological advances have dramatically increased the survival rate of people suffering brain injuries secondary to serious accidents. As a consequence, more individuals are living with permanent disabilities that affect their cognitive function, productivity and quality of life (e.g., Christensen, Colella, Inness, Hebert, Monette, Bayley, & Green, 2008; Crepeau & Scherzer, 1993; Dawson, Levine, Schwartz & Stuss, 2004; Dikmen, Ross, Machamer & Temkin, 1995; Jennett, Snoek, Bond & Brooks 1981; Rappaport, Herrero-Backe, Rappaport, & Winterfield, 1989; Ruttan, Martin, Liu, Colella, & Green, 2008; Till, Colella, Verwegen, & Green, 2008). In Canada, 48 people are admitted to hospital with traumatic head injuries every day, at an estimated annual total of 16, 811 people (Canadian Institute for Health Information, 2006). The majority (+90%) of hospitalized patients are later diagnosed with severe traumatic brain injuries (Canadian Institute for Health Information, 2006). The annual cost of treating TBI patients is estimated to exceed 3 billion dollars (Statistics Canada, 1984). In the United States, every year there are approximately 1.5 million new cases of traumatic brain injury (TBI) (National Center for Injury Prevention and Control, 1999). It is estimated that 5.3 million Americans are currently living with disabilities resulting from TBI, at an estimated annual cost of almost 50 billion dollars (Kraus & McArthur, 1996). The long-term economic impact is most striking because TBI occurs commonly in young adults (Canadian Institute for

Health Information, 2006; Thurman, Alverson, Dunn, Cuerrero & Sniezek, 1999). These prevalence and incidence data present clinical and experimental challenges to understand and to treat the effects of TBI.

Psychosocial Outcomes following Traumatic Brain Injury

The following review of outcomes following TBI places emphasis on moderate-to-severe injuries (consistent with the focus of this thesis). It is fully acknowledged that there is a growing literature on the consequences and predictors of mild traumatic brain injury, particularly given that is it is estimated that 75 - 80% of TBI's are 'mild' (Bernstein, 1999; Gerberding & Binder, 2003 as cited in Moore, Terryberry-Spohr & Hope). According to the Center for Disease Control, mild TBI is characterized the following symptoms following head trauma; self reported transient confusion, disorientation, impaired consciousness, dysfunction of memory around the time of injury, or loss of consciousness of less than 30 minutes; and observed signs such as seizures acutely following a head injury, irritability, lethargy or vomiting following head injury, headaches, dizziness, irritability, fatigue, or poor concentration (Centres for Disease Control and Prevention, 2004; see also Moore, Terryberry-Spohr & Hope, 2006 for review). Collectively, these symptoms are often referred to as 'post-concussional disorder' (PCD) in the literature, and they have been linked to the presence of affective disorders such as post traumatic stress disorder (e.g., Bryant, Creamer, O'Donnell et al., 2009; Vanderploeg, Belanger, & Curtiss, 2009), and other affective disorders (see Moore, Terryberry-Spohr & Hope, 2006 for review). Recent research has suggested, however, that factors other than head injury can account for the presentation of PCD. Being female, being widowed, divorced or separated, having a higher estimated IQ, slower response speed, stress, pain, noise sensitivity, and most consistently, premorbid history of affective or anxiety disorders, and *not* head injury, predict PCD (De Leon, Kirsch, Maio et al., 2009; Dischinger, Ryb, Kufera & Auman, 2009; McLean, Kirsch, Tan-Shriner et al., 2009; Meares, Shores, Taylor et al., 2008; Tanner & Beer, 2009). As such, it has been suggested that PCD is not the direct consequence of brain injury (e.g., McLean et al., 2009; Meares et al., 2008), but rather that mild TBI triggers pathophysiological changes and co-morbid affective disorders in a subset of high-risk individuals (Busch & Alpem, 1998). Based on these data, it appears that mild TBI constitutes a unique subset of people with brain injury that is distinct from those with moderate-to-severe injuries. As such, and considering that the majority of the data presented in this thesis is relevant to moderate-to-severe injury, the remainder of the literature review will place emphasis on the consequences and predictors of moderate-to-severe TBI.

There is a rich literature documenting negative psychosocial outcomes following moderate-to-severe TBI, the conclusion of which has been that the social and emotional consequences are more detrimental to the individual than are the neuropsychological consequences (e.g., Antonak, Livneh, & Antonak, 1993; Jennett, Snoek, Bond, & Brooks, 1981). Psychosocial outcomes are broadly defined as changes in a person's psychiatric status, personality, interpersonal function, level of independence and productivity. Each of these constructs will be discussed in turn below, followed by a discussion of the predictors of psychosocial outcomes. The reader should note, however, that although there is a considerable literature documenting negative outcomes following TBI, there are several obstacles to understanding what moderates these outcomes. One significant challenge is the heterogeneity of pathology and resulting variability in the presentation of TBI (Dikmen, Reitan, & Temkin, 1983; Stuss & Gow, 1992). While some patients present with significant psychosocial and emotional deficits, others present with more prominent residual neuropsychological deficits. The following section describes psychosocial outcomes following TBI. A detailed description of the neuropsychological sequelae of TBI is discussed later in this chapter. A second significant challenge to understanding the moderators of outcomes relates to methodology. There is considerable variability in design, the definition of outcomes, and inclusion/exclusion criteria between studies. These methodological differences may account for some of the variability in the data described below.

Psychiatric Outcomes: Compared to non-brain injured trauma survivors, people with TBI report more mental health problems and a need for mental health services (Ouellet, Sirois, & Lavoie, 2009). TBI is a risk factor for the development of Axis I disorders, in particular anxiety and depression (Hibbard, Uysal, Kepler, Bogdany & Siler, 1998; see Hiott & Labbate, 2002 for review of generalized anxiety disorders): anxiety incidence - from 18 to 28% (Andelic, Hammergren, Bautz-Holter, Sveen, Brunborg, & Roe, 2008; Dikmen, McLean & Temkin, 1986; Hibbard et al., 1998; Hiott & Labbate, 2002; Labbate & Warden, 2000; McCauley, Boake, Levin et al., 2001); depression incidence - 25 to 77% (Anson & Ponsford, 2006; Antonak et al., 1993; Glenn, O'Neil-Pirozzi, Glodstein, Burke, & Jacob, 2001; Ownsworth & Oei, 1998). These risks are greater if accompanied by a premorbid psychiatric condition (Corrigan & Deutschle, 2008), and are often comorbid with other Axis I disorders, for example substance abuse (Hibbard et al., 1998; Hiott & Labbate for review), and bipolar and personality disorders (van Reekum, Bolago, Finlayson, Garner, & Links, 1996). Of these psychiatric conditions, depression is most common (Hawthorne, Kaye, & Gruen, 2009; Hibbard et al., 1998), but the presence of both depression and anxiety has been related to poor functional outcome (Pagulayan, Hoffman, Temkin, Machamer, & Dikmen, 2008; Whelan-Goodinson, Ponsford, & Schonberger, 2008), and interpersonal conflict (Whelan-Goodinson, Ponsford, & Schonberger, 2008) post TBI. The prevalence of psychiatric conditions post TBI suggests that they should be targeted through rehabilitation (e.g., Fleminger & Ponsford, 2005; Morton & Wehman, 1995).

Personality and Interpersonal Outcomes: In addition to the emergence of psychiatric conditions, people with TBI often display personality changes including: increased irritability, frustration, moodiness, impulsivity, and inappropriate outbursts (see case studies reported in: Eslinger & Damasio, 1985; Harlow, 1868), utilization behaviour (Lehermite, 1983), deficits in humour appreciation, changes in locus of control, feelings of optimism, and changes in general disposition (Malia, Powell & Torode, 1995), changes in self concept

(Tyerman & Humphrey, 1984), reduced vitality (Ouellet, Sirois, & Lavoie, 2009), increased neuroticism and decreased extraversion (Kurtz, Putnam, & Stone, 1998; Tate, 2003), and impaired emotional processing (Allerdings & Alfano, 2006; Green, Turner, & Thompson, 2004; McDonald, Flanagan, Rollins, & Kinch, 2003; for reviews see Bornhofen & McDonald, 2008; Radice-Neumann, Zupan, Babbage, & Willer, 2007; Wood & Williams, 2008). Undoubtedly related to personality change and impoverished social and emotional functioning, interpersonal relationships are also affected by TBI. At 2-years post injury, 33% of people with TBI report having significant interpersonal conflicts (Finset, Dyrnes, Krogstad, & Berstad, 1995), and at 5-years-post injury that number increases to 48% (Kaplan, 1993). By 17-years-post injury, 60% are married or cohabitating (Wood & Rutterford, 2006). Astonishingly, however, in a 10-20 year follow up, 61% of a sample of people with TBI had no friends, and relied only on family members for social affiliation (Thomsen, 1992). Consequently, social isolation is a serious problem following TBI (e.g., Hawthrone, Kaye & Gruen, 2009).

Independence and Productivity: Maintaining independence in daily life activities is also an important factor relating to outcomes following TBI. Estimated rates of independence range from 52% (Tate et al., 1989) to 76% (Dikmen et al., 1995) at different times post injury. For example, at 1-year post injury, 76% of individuals with TBI are able to live independently, compared to 90% of trauma controls (Dikmen et al., 1995). At 5-10 years post injury, between 52% and 75% of people with TBI live at home, but with care and significant emotional support (Tate et al., 1989; Rappaport et al., 1989, respectively). By 17-years-post injury 72% of those with severe injuries are able to live without assistance, 60% are married or cohabitating, but only 28.7% are able to work full-time (Wood & Rutterford, 2006).

Returning to work also represents a major challenge for many people following TBI, and the inability to return to work has serious psychological and financial consequences. Estimates of the proportion of people returning to work following TBI are variable, and range from 12% (Ruffolo, Friedland, Dawson, Colantonio, & Lindsay, 1999) to 87.5% (Haboubi, Long, Koshy, & Ward, 2001; see Shames, Treger, Ring, & Giaquinto, 2007 for review of return to work following TBI). Notably, these studies include various levels of injury severity, at various times post injury. It is clear, however, that despite the variability of reported outcomes following TBI, the consequences of TBI can be pervasive and long term. A remaining question is, what predicts these outcomes, and how might we manipulate these variables to improve quality of life for people with TBI?

Predictors of Outcomes: A series of investigations examining long-term outcomes following TBI have demonstrated that injury severity is predictive of outcomes. For example, injury severity predicts greater reliance on family members, reduced income and unemployment (Dikmen et al., 1995; also see Ponsford, Olver, Curran, & Ng, 1995), emotional problems (Dikmen et al., 2003), global life satisfaction (Wood, & Rutterford, 2006), and general psychosocial outcomes (Draper, Ponsford & Schonberger, 2007). Some predictive models have demonstrated that injury severity, in conjunction with other variables, are the best predictors of outcomes. For example, together with injury severity, education, duration of post traumatic amnesia, fatigue, aggression, anxiety and depression are all predictive of psychosocial outcome (Draper et al., 2007), and injury severity together with level of disability, are predictive of employment status (Ponsford et al., 1995).

However, it has also been demonstrated that negative outcomes can be, to some degree, independent of injury severity. Even in a subset of people with good outcomes (e.g., return to work/school), over half report significant difficulty and psychological stress independent of injury severity (Dawson et al., 2004). Younger age at time of injury, higher estimated premorbid IQ (Green, Colella, Christensen, Johns, Frasca, Bayley, & Monette, 2008), global neruopsychological performance at 5 months post injury and length of hospital stay (Green, Colella, Hebert, Bayley, Kang, Till, & Monette, 2008) and self-efficacy have also been shown to be a strong predictors of outcomes (Cicerone & Azulay, 2007). Another predictor of outcomes that has gained more attention recently is coping. In fact, escape avoidant coping accounts for variance in outcomes above and beyond neurological status, pain, or depression (Dawson, Schwartz, Winocur & Stuss, 2007). Understanding the mechanisms by which coping moderates these outcomes is an important step before rehabilitative measures can be taken.

Coping as a Moderator of Outcomes

Clearly, the long term outcomes for TBI are not good; what moderates these negative outcomes is uncertain. Coping has been hypothesized to be the final common pathway leading to negative outcome following TBI (Moore & Stambrook, 1995; also see Kendall & Terry, 1996). In a general sense, coping can be thought of as a person's cognitive and behavioural strategies for managing situations that have been appraised as stressful or taxing. The coping literature has broadly divided coping into 'problem-focused' and 'emotion-focused' coping styles. Problem-focused coping involves actively seeking a solution to the problem by defining the problem, generating alternative solutions, weighing the alternatives, choosing among the alternatives and acting upon them (Lazarus & Folkman, 1984). These processes are likely dependent on intact executive processes (e.g., foresight, planning, and putting problems in perspective). Emotion-focused coping involves managing stress through emotion, and could include avoidance, minimization, distancing, positive comparisons, and gaining positive value from otherwise negative events (Lazarus & Folkman, 1984). These processes likely depend, in part, on intact affective processing (e.g., emotional control, inhibition, behavioural self-regulation).

The terminology used to describe different sub-types of problem-focused and emotion-focused coping varies from study to study. Problem-focused coping has been described as approach coping (Dahlquist, Czyzewski, Copeland et al., 1993; Finset & Andersson, 2000), planful problem solving coping (Krpan, Levine, Stuss, & Dawson, 2007), and problem solving coping (Curran, Ponsford, & Crowe, 2000). Emotion-focused coping has been described as maladaptive/escape avoidant coping (Dawson et al., 2007), escape avoidant coping (Krpan et al., 2007), avoidance (Malia et al., 1995), defensive coping (Barger, Kircher & Croyle, 1997; Shane & Peterson, 2004), repressive coping (Weinberger, Schwartz & Davidson, 1979), defensive-repressive coping (Brown, Tomarken, Orth et al., 1996), and disengagement-type coping (Martz, Livneh, Priebe et al., 2005). One reason for these discrepancies in terminology may be that different instruments are being used to assess coping. The term 'problem focused coping', for this thesis, refers to strategy that necessitates defining the problem, generating alternative solutions, weighing the alternatives, choosing among the alternatives and acting upon them. The term 'avoidant coping', for this thesis, refers to a sub-component of emotion-focused coping that involves evading the stressor. The focus of this thesis is on avoidant coping because of its relation to outcomes post-TBI (as described next).

Coping Post-TBI: There are consistent patterns of relations between coping and outcome post-TBI. People with TBI who engage in less self-reported avoidant coping have better psychosocial outcome (Anson & Ponsford, 2006; Leach, Frank, Bouman, & Farmer, 1995; Malia et al., 1995; McMillan et al., 2003; Tomberge, Toomela, Pulver, & Tikk, 2005), whereas those engaging in more avoidant coping experience more anxiety, depression and increased psychosocial distress (Anson & Ponsford, 2006; Curran et al., 2000; Dawson et al., 2006; Finset & Anderson, 2000; Kendall et al., 2001; Lubosko, Moore, Stambrook & Gill, 1994). These relationships are present despite the lack of measurable differences in the types of coping style reported by people with TBI and matched control groups (e.g., orthopaedic controls, friends and family members; Curran et al., 2000; Dawson et al., 2006; Krpan et al., 2007).

Previous attempts may have been unsuccessful in measuring group differences in coping style post-TBI for a number of reasons. One may be that the instruments used to assess coping style may not been sufficiently sensitive to detect differences. Another possibility is that people with TBI may not accurately report their coping style in self-report questionnaires, or they are not aware of their true coping behaviour. Alternatively, there may be no differences in the coping styles used between groups, but rather in how contextually appropriate and strategically implemented those strategies are executed. For example, people with TBI may use the same types of strategies as healthy controls, but they may be using them in inappropriate situations (e.g., using an avoidant strategy when a problem-focused strategy is more advantageous), or the sequencing and execution of the strategy may be disorganized (Krpan et al., 2007). The question remains -- what are the mechanisms of the relationship between avoidant coping and poor outcomes post-TBI? The pathology of TBI might help to explain the mechanisms of this relationship and be the key to overcoming the limitations described above.

The Pathology and Neuropsychological Consequences of TBI

There are two primary mechanisms of damage following a TBI (for review of the mechanism and functional consequences, see Cicerone, Levin, Malec, Stuss, & White, 2006; Stuss and Levine, 2002; Levine, Turner & Stuss, 2008). The first mechanism of damage is diffuse axonal injury (DAI) (Povlishock, 1993). DAI occurs when rapid acceleration and deceleration forces (e.g., in a motor vehicle accident) cause the brain to shift within the skull causing microscopic stretching and tearing of axons and small blood vessels throughout the brain. This may occur with or without impact. The second mechanism of damage is focal cortical contusion. This may occur due to a direct blow to the head, or due to coupe contra coup forces causing the brain to abrade against ridges of the interior skull. In either case, the prefrontal regions of the brain are particularly vulnerable to TBI. The acceleration-deceleration forces arising from DAI may affect many brain structures and tracts, but disproportionately affects the prefrontal cortex (for review: Greenberg, Mikulis, Ng, DeSouza, & Green, 2008; Levine, Kovacevic, Nica, Cheung, Gao, Schwartz, & Black, 2008; Levine et al., 2002; Levin & Eisenberg, 1991; Nevin, 1967; Povlishock, 1993; Stuss & Gow, 1992). DAI may also affect subcortical structures with critical projections to the prefrontal cortex, thus interfering with complex frontal-subcortical networks that support complex behaviour (Adair, Williamson, Schwartz, & Heilman, 1996). Even in the case of mild TBI, recent neuroimaging technology has helped to demonstrate white matter damage (Kraus, Susmaras, Caughlin, Walker, Sweeney, & Little, 2007; Lo, Shifteh, Gold, Bellow, & Lipton, 2009). The ventral prefrontal regions of the brain are also particularly vulnerable to focal cortical contusions due to tissue strains against the ridges of the anterior and middle fossa (Courville, 1937; Ommaya & Gennarelli, 1974; Gentry, Godersky & Tompsen, 1988). Even beyond the acute stages of neurological recovery, there is evidence of a subacute progression of atrophy as late as 2.5 – 4.5 years post injury (Ng, Mikulis, Glazer et al., 2008).

There is strong evidence that the prefrontal cortex may be both functionally and anatomically fractionated into at least four distinct regions; dorsolateral prefrontal cortex (DLPFC); ventromedial prefrontal cortex (VMPFC), superior medial prefrontal cortex, and polar regions (e.g., Stuss, Alexander, Floden, et al., 2002; Stuss, 2007). Each of these regions has been associated with distinct functions, with implications for how rehabilitation may be best targeted for patients with varying presentations (see Cicerone et al., 2006; Stuss & Levine, 2002 for review). The focus of this thesis is the dissociation between the DLPFC and VMPFC as they relate to outcomes following TBI.

The Dorsolateral & Ventromedial Prefrontal Cortices: The dorsolateral prefrontal cortex (DLPFC) is both anatomically (Pandya & Yeterian, 1996) and functionally (Stuss & Levine, 2002) distinct from the ventromedial prefrontal cortex (VMPFC). The DLPFC is part of the archicortical trend that originates in the hippocampus. The VMPFC is part of the paleocortical trend that emerges from the caudal orbitofrontal cortex, which is intimately connected with limbic structures (Pandya & Barnes, 1987). Based on a rich literature, executive functions are defined as cognitive functions that have typically been associated with the DLPFC. Under this definition, executive functions would include initiation, planning, hypothesis generation, cognitive flexibility, decision-making, regulation, judgment, and feedback utilization (Stuss & Benson, 1986; Anderson & Tranel, 2002; Cicerone et al., 2006; Stuss & Levine, 2002).

In contrast to executive functions, affective functions are defined as those functions typically associated with the VMPFC. The VMPFC is thought to mediate inhibition, emotion, reward processing (including the acquisition and reversal of stimulus reward contingencies), and general behavioural self-regulation (Eslinger & Damasio, 1985; Harlow, 1868; Rolls, 2000; Stuss & Levine, 2002; Cicerone et al., 2006). Because the prefrontal cortex is particularly sensitive to the effects of TBI, most people with TBI experience some sort of executive and/or affective dysfunction. Diffuse brain damage can also impact on executive and affective functioning through reduction in the efficiency of information processing, even when the prefrontal cortex is not affected, with consequences for outcome following TBI (e.g., Stuss & Gow, 1992; Stuss, Gow, & Hetherington, 1992; Stuss & Levine, 2002).

Executive Dysfunction: Executive dysfunction following TBI may include reduced speed of attention, poor mental flexibility and abstract thinking, difficulty maintaining and shifting attention, utilizing feedback, inhibiting responses and working memory (e.g., Ashman, Gordon, Cantor & Hibbard, 2006; Burgess & Robertson, 2002; Shallice & Burgess, 1991; Stuss & Levine, 2002). These deficits are evident on a number of standard neuropsychological tests measuring cognitive functions. Some examples of tests that are designed to be sensitive to dorsolateral damage, in particular, are the Wisconsin Card Sorting Test (WCST), the Trail Making Test (TMT), and the Stroop Test (Stuss & Alexander, 2000; Stuss, Bisschop, Alexander, et al. 2001; Stuss, Ely, Hugenholtz et al., 1985; Stuss, Floden, Alexander, Levine & Katz, 2001).

Affective Dysfunction: People with TBI may also experience affective deficits associated with ventromedial prefrontal cortex damage. Affective dysfunction may include dramatic changes to personality, and difficulty with making decisions, regulating behaviour, and processing emotions and reward, as well as social disinhibition (Bechara, Damasio, Damasio & Anderson, 1994; Bornhofen & McDonald, 2008; Eslinger & Damasio, 1985; Harlow 1868; see Stuss & Levine, 2002 for review). In addition to the social and emotional sequelae described above, patients with right hemisphere frontal lobe damage, particularly involving right ventral and medial PFC, show attenuated skin conductance response (SCR) and heart rate to psychologically relevant or emotional stimuli (Andersson & Finset, 1998; Angrilli, Palomba, Cantagallo et al., 1999; O'Keeffe, Dockree, & Robertson; 2004, Sanchez-Navarro, Martinex-Selva, Roman et al., 2005; Zahn, Grafman & Tranel, 1999).

Linking Coping to TBI Pathology: A body of work has demonstrated quite clearly the relationships between reported coping and outcomes following TBI (e.g., Anson & Ponsford, 2006; Curran et al., 2000; Dawson et al., 2006; Finset & Anderson, 2000; Kendall et al., 2001; Lubosko, et al., 1994; Malia et al., 1995; McMillan et al., 2003; Leach et al., 1995; Tomberge et al., 2005). A remaining question concerns the mechanisms by which coping moderates these outcomes. Moore and Stambrook (1995) put forth a model where the behavioural, cognitive, emotional and interpersonal sequelae of TBI result in a cascade of changes in cognitive belief structure (e.g., external locus of control, internal attributional style), ultimately resulting in the selection of palliative emotion focused coping that leads to negative outcomes. The model, however, did not account for the pathology of TBI.

More recent work has illuminated some of the potential neuropsychological mechanisms by which avoidant coping might moderate outcomes post TBI. Krpan et al. (2007) found a relationship between executive function and coping 1-year-post TBI, where patients who had better executive function engaged in more problem-focused coping and those with lower executive function engaged in more avoidant coping. Importantly, these relationships were not evident in a matched sociodemographic control group, despite the fact that there were no group differences in self-reported coping style. The authors hypothesized that problem-focused coping requires executive processes, such as foresight, planning, and putting problems in perspective. It was further hypothesized that executive deficits in people with TBI interfere with their ability to successfully execute problem-focused coping strategies. As a result, people with TBI engage in avoidant coping, by default, under inappropriate circumstances. This hypothesis would help to account for the relationships between avoidant coping and negative psychosocial outcome following TBI. However, affective measures were not included in the Krpan et al. study. Given that there are at least two main functional consequences of TBI (executive and affective dysfunction), this story is incomplete. It is uncertain whether both executive and affective function are related to avoidant coping.

Summary

Coping has been hypothesized to be the final common pathway leading to negative outcomes following TBI (Moore & Stambrook, 1995; Kendall & Terry, 1996). Avoidant coping is related to negative outcomes following TBI (Curran et al., 2000; Dawson et al., 2006; Finset & Anderson, 2000; Kendall et al., 2001; Lubosko, et al., 1994), and there is evidence that neuropsychological performance mediates this relationship (Krpan et al., 2007). Variability in the context of coping and the presentation of TBI (Stuss & Binns, 2008; Stuss, Murphy, Binns & Alexander, 2003) make further delineating this relationship a challenge. Ultimately, it will be necessary to characterize sub-types of TBI patients based on their neuropsychological and coping profile so that rehabilitative intervention can be targeted (see Levine, Turner & Stuss, 2008). The purpose of the studies described below is to help delineate the mechanisms of avoidant coping following TBI.

Research Overview, Objectives and Hypotheses

The following is one large study designed to elucidate the mechanisms of the relationship between avoidant coping and poor outcomes post TBI. People with TBI and controls completed 1) a neuropsychological test battery assessing both executive and affective function; 2) a series of questionnaires evaluating self-reported coping, anxiety, depression and psychosocial outcomes; 3) the 'Baycrest Psychosocial Stress Test' (BPST), where coping was observed directly; and 4) physiological measures (heart rate, galvanic skin response and cortisol reactivity) were gathered during completion of the BPST. The hypotheses of the study are listed below.

The **HYPOTHESES** are as follows:

1) People with TBI will engage in more avoidant coping strategies on behavioural measures (Baycrest Psychosocial Stress Test [BPST]), but will under-report the use of such strategies on self-report measures (Ways of Coping Questionnaire - Revised, [WOC]).
2) Within the TBI group, both executive and affective function will be positively related to planful coping, and negatively related to avoidant coping on the WOC and BPST.
3) Variables such as psychiatric status, personality and physiological responsivity to stress will moderate the relationship between neuropsychological function and coping.

4) Planful coping will relate to positive outcomes, and avoidant coping to negative outcomes following TBI.

For the purposes of clarity, these hypotheses will be addressed in three sections. Chapter Three, entitled *The effects of psychosocial stress on coping behaviour post traumatic brain injury* addresses hypothesis one, and investigates observed coping behaviour during a simulated real-world stressful situation, and compares it to self and SO reported coping. Chapter Four, entitled *The moderators of altered patterns of coping behaviour following moderate-to-severe traumatic brain injury*, addresses hypotheses two and three, and investigates the factors that contribute to the use of avoidant coping in a subset of TBI patients who displayed unusual coping behaviour (i.e., the moderate-to-severe group). Chapter Five, entitled *The relationships between coping and self and other reported outcomes following moderate-to-severe traumatic brain injury* addresses hypothesis three, and investigates the relations between reported and observed coping and outcomes.

CHAPTER TWO

General Methods

The research protocol was approved by the Joint Baycrest/University of Toronto Research Ethics Board and all participants provided written informed consent.

Recruitment and Inclusion/Exclusion Criteria

Inclusion/Exclusion criteria were established based on previous published work (e.g., Krpan et al., 2007). TBI participants were recruited from the community by means of advertisements in the local newspaper and acquired brain injury newsletters, flyers, and word of mouth. Recruitment took place over a 19-month period (September 2007 – March 2009). Prior to being admitted to the study, participants answered a series of questions to ensure eligibility. Inclusion criteria were: English speaking, closed head injury, Glasgow Coma Scale (GCS) rating of 15 or less and/or amnesia of the event (at minimum), and at least 18 years of age. Patients with a premorbid history of neurological disease, systemic disease, psychiatric disorder, substance abuse or developmental disorder were excluded. Patients were also excluded if there were co-morbid spinal cord injuries, serious burns, serious facial disfigurement and/or amputations. Significant Other (SO) participants were recruited through the TBI participant. The SO group consisted of family members, friends, or support workers who knew the person with TBI well, and felt competent enough to complete the WOC, SIP and DEX on their behalf. Control participants were drawn from friends and family member of TBI participants (where possible) to control for sociodemographic factors specific to the TBI participants (see Dikmen, Ross, Machamer, & Temkin, 1995). When this was not possible, additional control participants were drawn from a subject pool at the Rotman Research Institute at Baycrest, Toronto, Canada, with an effort made to match the patients in terms of age, education, and sex. Control participants were English speaking and did not have any serious medical, psychiatric or substance abuse problems, and had no history of sustaining a brain injury or any neurological disease. Demographic and health related data were obtained from structured interviews, chart extractions (where possible), and questionnaires.

General Procedure

All testing was completed by the author (KMK) at the Rotman Research Institute, Baycrest, Canada. The participants described in this thesis participated in one large study over the course of two days. The data are presented in three chapters for the purposes of clarity.

On both days of testing, participants arrived between 9 and 10am. The BPST was always administered on the morning of the second test day to control for circadian rhythms influencing cortisol.

Day one of testing consisted of a series of neuropsychological tests and questionnaires. On day two of testing, the BPST was administered first, followed by another series of neuropsychological tests and questionnaires (described later in the methods section). Prior to task instructions for the BPST, participants completed the State component of the State-Trait-Anxiety-Inventory so that subjective anxiety could be measured prior to engaging in the task. Participants then provided a salivary cortisol sample (see below for details), and were then fitted with the equipment to measure heart-rate, heart-rate-variability and skin conductance response (also see below for details). Immediately prior to task instructions, participants sat quietly and were instructed to rest and reduce speaking and movement for a five minute period during which time heart-rate, heart-rate-variability and skin conductance levels were continuously recorded. This provided a baseline measure of physiological arousal. Following the rest period, task instructions were administered, and heart-rate and skin conductance level were continuously recorded throughout the task. Immediately following task completion, the participant completed a second State-STAI to index the level of subjective anxiety induced by the task. Then a second cortisol sample was taken, followed by a second 5-minute rest period to re-establish baseline physiological measures. Finally, 20 minutes following task completion, a third cortisol sample was taken. A detailed description of the BPST and the physiological measures can be found below.

Baycrest Psychosocial Stress Test

The ***Baycrest Psychosocial Stress Test (BPST)*** was adapted from two separate tests: the Trier Psychosocial Stress Test (TPST; Kirschbaum, Pirke & Hellhammer, 1993) and Barger, Kircher and Croyle's (1997) social speech task. The BPST consists of an anticipation period where coping can be directly observed (10 minutes) and a test period (10 minutes) in which the participant has to deliver a speech and perform mental arithmetic while being video recorded (see Figure 1). The author elected to use these aspects of the TPST because the test has been shown to reliably induce adrenocortical stress responses (increased adrenocorticotropin, cortisol, growth hormone, prolactin, as well as increases in heart rate), and has been used extensively in non-TBI populations, and a precise protocol has been published (see Kirschbaum et al., 1993). The author elected to include components of Barger et al., (1997) social speech task because it requires less staff and lab space to conduct the test relative to the TPST, and because their protocol includes providing the participant with auditory cues (during speech delivery) about what they should be discussing at a given time point. This feature of the Barger et al. (1997) social speech task was particularly beneficial for a group of people with TBI, who often had difficulty independently producing a speech on a specific topic for a 5 minute time period. It also allowed the experimenter more control of the content of the speech.

Upon arrival for day two of testing, participants were taken to a room where audio-video equipment was installed. Task instructions can be found in appendix A, and also see Figure 1 for a schematic of BPST protocol.

Briefly, participants were asked to give a 5-minute speech about crime in Toronto. They were told that they were going to be left alone in the examination room for 10-minutes, during which time they were free to use any of the materials they found in the room (barring they did not disturb wires measuring their heart rate, heart rate variability and skin conductance), but did not have to do so. They were told that following the 10 minute delay, a colleague of the examiner (e.g., Dr. Blackwood) would enter the room, and that they were to give their speech to him/her. The participant was told that Dr. Blackwood was an expert in communications, and was there to evaluate the content of their speech, as well as their non-verbal behaviour. In fact, Dr. Blackwood was a trained research assistant. In addition, participants were told that the speech would be recorded, and that later a panel of judges would analyze the content of the speech, non-verbal language, and analyze voice frequency. Participants were then provided the opportunity to ask questions, and clarify task demands. Following the oral instructions, the participant was left alone for 10 minutes to prepare the speech (or do as he or she pleased) and behaviour was video-recorded. In front of them on the table were materials that hypothetically could facilitate either problem-focused coping or avoidant coping. The problem-focused materials were a pencil and paper, a package labelled ‘speech requirements’ containing the required outline of the speech, and a newspaper. The avoidant materials were magazines (geared to both male and female audiences), crossword puzzles and Sudoku puzzles. Behaviour during the preparation period was video-recorded and coded by three raters at a later date.

Following the 10 minute preparation period, the confederate (e.g., Dr. Blackwood) entered the room and the participant was prompted to introduce himself, and begin his speech. If the participant finished the speech in less than 5 minutes, the confederate prompted by saying ‘You still have some time left. Please continue!’ If the participant finished again before 5 minutes, the confederate asked a set of prepared questions (see appendix A). Following completion of the 5-minute speech, the confederate required that the participant serially subtract the number 13 from 1,022 as quickly and as accurately as possible. On every failure, the subject was asked to restart at 1,022 with the confederate interfering saying ‘Stop. 1,022’. After 20 minutes (preparation + speech + mental arithmetic), the task was terminated and the experimenter returned to conduct an interview regarding strategy, and debrief.

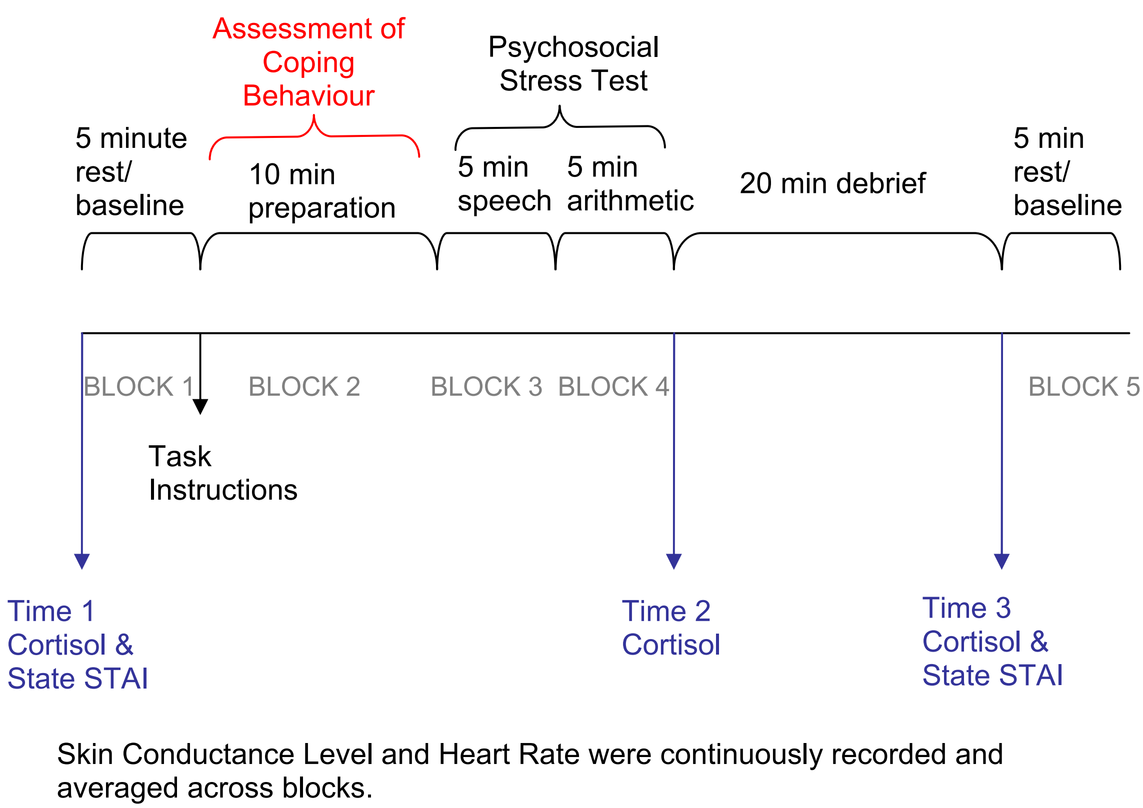

Figure 1: Schematic representation of the Baycrest Psychosocial Stress Test

Analysis of Behaviour

Coping behaviour during the preparation period was video recorded, and later coded by three independent blind viewers. During the preparation period, the behaviours observed included speech writing, reviewing the speech, reading speech requirements, thinking and writing, reading the magazine, playing crosswords, texting, staring into space, reading the newspaper, looking through materials and 'other' behaviours. To reduce these behaviours into meaningful categories, correlation analyses were performed. Behaviours that were highly correlated to one another were then summed to create total scores (see Appendix A for correlation matrices for behaviour). These analyses produced two types of coping scores; planful and avoidant. The total planful score was the summed durations of writing, reviewing the speech, and thinking and writing. The total avoidant score was the summed durations of reading magazines, playing puzzles, staring into space and 'other' avoidant behaviours. In addition to recording behaviours, raters also evaluated the quality of speech performance on a scale of one to ten. Two-way random absolute ICC analysis with 95% confidence intervals was then performed to assess reliability between the three raters' speech quality scores. Reliability was excellent; ICC for planful = 0.985; ICC for avoidant = 0.947; ICC for speech score = 0.888. The behavioural data presented in this thesis are the average across all three raters.

Perceived Level of Stress

The ***State Component of the State Trait Anxiety Inventory (State STAI)*** (Speilberger, 1983) is a 20-item inventory assessing state anxiety. The state STAI was used to assess how participants felt immediately prior to and immediately following the BPST. This allowed for an estimate of the level of anxiety that was induced by the task.

Physiological Correlates of Stress

Electrodermal Activity: Skin conductance response was recorded continuously using the BioPac GSR 100 amplifier with a constant voltage method (0.5V). The participant was asked to gently exfoliate the distal phalanges of the non-dominant index and middle finger with alcohol to ensure proper electrode contact. Two 8 mm Ag/AgCl finger electrodes were filled with electrode gel and affixed to the participant using self-adhesive tape and Velcro (see Dawson, Schell, & Filion, 1990; O'Keeffe, Dockree, & Robertson, 2004). Skin conductance was continuously recorded throughout all five blocks of the BPST (i.e., baseline, preparation, speech, arithmetic, and baseline 2).

Heart-Rate and Heart-Rate-Variability*:* Heart-rate and heart rate variability were also continuously recorded using the BioPac ECG 100 amplifier throughout the five blocks of the BPST. Participants were asked to gently exfoliate three places on their thorax with alcohol to ensure proper electrode contact (on the right below the clavicle, on the left below the clavicle, and on the left ribcage below the heart). Electrode gel was then applied to the surface of three self adhesive electrodes and affixed to the participant at the above named locations, and leads were attached. Heart-rate was calculated by averaging the mean beats per minute across each of the 5 blocks of the BPST. Heart-rate variability was calculated in the time domain by averaging the standard deviation of the R-R interval for each block of the BPST.

Cortisol: Salivary cortisol samples were taken using a salivette (Salimetrics, State College, PA). Participants removed a cylindrical cotton swab from a test tube and chewed or sucked on it for 30-45 seconds or until it was fully saturated. They then returned it to the test tube. Following the session samples were then frozen in a -40°C freezer until analyzed. Samples were later centrifuged for 10 minutes, at 3000 rpm at 4°C, before being analyzed in duplicate using a expanded range high sensitivity salivary cortisol enzyme immunoassay kit (Salimetrics, State College, PA) for quantitative measurement of salivary cortisol.

Cortisol was measured at Time 1, before task instructions; at Time 2, immediately following the BPST; and Time 3; 20 minutes after completing the BPST. Measurement of hormones at 20-minute intervals allowed for analysis of changing free (biologically active) concentrations of cortisol in response to the tasks (e.g., Fleming, Steiner, & Corter, 1997; Krpan, Coombs, Zinga, Steiner & Fleming; 2005).

Questionnaire to Assess Coping

The *Ways of Coping Questionnaire-Revised* *(WOC)* (Folkman & Lazarus, 1988) was designed to assess strategies that people use to cope with stressful life events. Participants are asked to recollect a stressful event that has occurred within the last week and to respond using a 4-point Likert-type scale (0 indicating ‘not applicable or not used’, to 3 indicating ‘used a great deal’) to a series of 66 statements as to how they dealt with the stressful occurrence. The WOC has been used with TBI populations (e.g., Krpan et al., 2007; Moore & Stambrook, 1994; Moore, Stambrook, & Peters, 1989; Moore & Stambrook, 1992) and has been shown to have acceptable reliability and validity. The WOC generates scores relating to eight coping styles: Confrontive coping, distancing, self-controlling, seeking social support, accepting responsibility, escape-avoidant, planful problem solving, and positive reappraisal. The current study was concerned with two coping styles in particular, ‘planful problem solving coping’, ‘escape avoidant coping’. The escape avoidant score was used because it has been related to negative outcomes in earlier research (e.g., Dawson et al., 2004; Dawson et al., 2006), and because it has been inversely related to executive function (Krpan et al., 2007). The planful problem solving score was used because the items on this scale are most reflective of deliberate, instrumental coping behaviours that likely involve a number of executive functions in order to be executed effectively. The WOC was also completed by an SO, who evaluated the person with the TBI on the same scale.

Neuropsychological Test Battery

A neuropsychological test battery assessing premorbid intelligence, working memory, and executive and affective functions was also administered across the two days of testing. The tests outlined below were selected to allow for 1) characterization of the sample, and 2) careful documentation of executive and affective function.

Intelligence

The vocabulary subtest of the ***Shipley Institute of Living Scale*** (Zachary, 1982) was used to assess pre-morbid intellectual functioning. The vocabulary subtest is thought to reflect crystallized (versus fluid) intelligence, and has been shown to be a good estimate of premorbid intelligence (Zachary, 1982).

The Raven Standard Progressive Matrices (Raven, 1960; Raven, Court, & Raven, 1976) was used to assess the ability to form perceptual relations and to reason by analogy independent of language. The test was used as a proxy of Spearman's g.

Memory

The ***alpha span*** (see Craik, 1990) is a measure of working memory that places emphasis on the central executive (Baddeley, 1986). Participants are initially presented verbally with two words and asked to verbally alphabetize them according to the first letter in each word. The number of words in the lists increases every second trial to a maximum of eight words. Scoring and administration was done as in Krpan et al. (2007), where poor working memory was related to avoidant coping style.

The ***Brown-Peterson Procedure*** (Brown, 1958; Peterson & Peterson, 1959) was also administered to assess components of working memory and control over interference, and is highly sensitive to TBI (Stuss et al., 1985). Initially, participants are required to recall a verbally presented three consonant trigram with no delay period. Following five zero-delay trials, participants are presented with 20 more trigrams and required to count backwards by threes for 3, 9, or 18 seconds before recalling the trigram. Administration and scoring was in concordance with Brown (1958) and Peterson and Peterson (1959). Higher performance on the Brown Peterson Procedure has been correlated with planful coping, and lower performance with avoidant coping (Krpan et al., 2007).

Executive Function

The ***Stroop Test*** was used to assess the ease of shifting perceptual set to meet changing task demands and the ability to suppress automatic responses in favour of unusual ones (Spreen & Strauss, 1998). Stimuli, administration and scoring were done in concordance with the Comalli, Wapner & Werner (1962) version (see also Stuss, Floden, Alexander, Levine & Katz, 2001). Lower performance in the Stroop Task has been correlated with the use of avoidant coping following TBI (Krpan et al., 2007).

The ***Trail Making Test*** (TMT) parts A and B (Army Individual Test Battery, 1944) was used to assess speed of attention, visual search and motor function, and mental flexibility (Spreen & Strauss, 1998). The primary scores on the TMT are time to completion on parts A, B, B-A (difference score) and (B-A)/A (proportional score) (see also Stuss et al., 2001). Lower performance in the TMT has been correlated with avoidant coping following TBI (Krpan et al., 2007).

The ***Wisconsin Card Sorting Test*** *(WCST)* was administered to assess the ability of individuals to think abstractly, maintain and shift attention appropriately and to utilize feedback in problem solving, and is sensitive to focal frontal lesions (see Stuss, Levine, Alexander, Hong, Palumbo et al., 2000). The WCST was administered according to the Grant and Berg (1948) criteria, and scored as specified by Stuss et al. (2000). Higher performance in the WCST has been correlated with planful coping post TBI, where lower performance on the WCST has been correlated to avoidant coping (Krpan et al., 2007).

The ***Zoo Map*** component of the Behavioural Assessment of the Dysexecutive Syndrome (Wilson, Alderman, Burgess, Emslie & Evans, 1996) was used to assess planning and the ability to modify behaviour based on feedback. The Zoo Map also allows for a comparison to be made between abilities under high demand low structure situations, versus concrete structured situations. Scoring and administration was done in concordance with Wilson et al. (1996).

The ***Controlled Oral Word Association TEST*** (aka FAS test) was used to assess the ability to spontaneously produce words beginning with a given letter. Performance on the FAS is contingent on cognitive activation, sustained output, and category switching and clustering that are reflective of frontal lobe function (Stuss, Alexander, Hamer et al., 1998; Troyer, Moscovitch, Winocur, Alexander & Stuss, 1998). Scoring and administration was in concordance with Stuss et al. (1998).

The **Sustained Attention to Response Task (SART)** was used to assess sustained attention, and is not only sensitive to TBI, but also correlates to everyday attentional failures (Robertson, Manly, Andrade, Baddeley, & Yiend, 1997). Two-hundred-and-twenty-five single digits (25 of each of the nine digits) were presented visually over a 4.3-min period. Each digit was presented for 250 ms, followed by a 900 ms mask. Participants responded with a key press to each digit, except 25 occasions when the target (e.g., 3) appeared, when they were asked to withhold a response. The target digit was distributed throughout the 225 trials in a pre-fixed quasi-random fashion (see Robertson, Manly, Andrade, Baddeley & Yiend, 1997 for detailed description). Each participant completed four blocks of the SART; two standard blocks, and an awareness and feedback condition. In the awareness condition, participants were required to make a verbal indication of an error of commission. In the feedback condition, participants were required to learn, through trial and error, what the two target stimuli were.

VMPFC Function as an Estimate of Affective Function

The neuropsychological tests used to assess executive functions clearly do just that; a test indexing planning (e.g., Zoo Map) requires a participant to plan; a test assessing feedback utilization (e.g., WCST) requires a participant to utilize feedback. The neuropsychological tests available to assess VMPFC, however, are not necessarily *affective* in nature. For example, the smell identification test (described below) maybe be sensitive to ventral prefrontal damage, but it does not necessarily require emotional functioning. As such, the neuropsychological tests described below will be referred to as VMPFC tests (rather than affective function tests), with the expectation that they will be highly correlated with affective function given their common neurological bases. Moreover, any task that contains a motivational component (e.g., Gambling Task, Object Alternation Task) is also considered to be affective, because motivation is an affective process.

The ***Brief Smell Identification Test*** (Doty, Shaman & Dann, 1984) was used to assess orbitofrontal integrity. Although patients with damage to medial temporal structures and generalized frontal lobe damage show deficits on this test, patients with damage including the orbitofrontal cortex perform significantly worse (Jones-Gotman & Zatorre, 1988). The test is recognized as being sensitive to TBI severity and ventral frontal pathology (Doty, Yosem, Pham et al., 1997; Fjuiwara, Schwartz, Gao, Black, & Levine, 2008; Yousem, Geckle, Bilker et al., 1999), and has also been linked to poor neuropsychological and functional outcome post TBI (Callahan & Hinkenbein, 1999).

The ***Gambling Task*** (Bechara et al., 1994) was used to assess decision making following TBI, and has been shown to be sensitive to ventral prefrontal damage (Bechara et al., 1994; Bechara et al., 1997), the effects of TBI (Levine, Black, Cheung et al., 2005; Fujiwara et al., 2008), and has correlated with outcome (real-life memory, executive and emotional problems, Levine et al., 2005).

The ***Object Alternation Task*** (Freedman, 1990) was also used to assess ventral prefrontal cortex integrity. This task has been shown to reflect ventrolateral-orbitofrontal and medial frontal dysfunction in humans (Freedman, Black, Ebert & Binns, 1994), and is sensitive to TBI (Fujiwara et al., 2008). The task requires participants to locate a penny hidden under one of two objects. Participants must learn that the object under which the penny was located is being alternated following each correct response. Administration and scoring was in concordance with Freedman (1990).

An ***ID/Reversal Shifting Task*** was used to assess visual discrimination, attentional set formation, maintenance, shifting and flexibility of attention. Stimuli consisted of colour-filled shapes and white lines. Simple stimuli were composed of one dimension (e.g., a shape), whereas compound stimuli was made up of both stimuli (white lines overlying colour-filled shapes). The participant must learn which stimulus is correct (using feedback), and be able to switch criteria as the task progresses. This task is thought to be sensitive to frontal lobe damage (Owen, Roberts, Polkey, Sahakian and Robbins, 1991), with deficits in reversal learning (interpreted as affective; see Stuss & Levine, 2002) relating to orbitofrontal cortex damage (e.g., Fuster, 1997; Mishkin, 1964; Rolls, 2000).

The Awareness of Social Inference Test (TASIT) (McDonald, Flanagan, Rollins, & Kinch, 2003) was used to assess social perception. There is evidence that people with TBI are impaired on tests of social perception and inference (e.g., McDonald & Flannagan, 2004), and social perception may be an important moderator of coping. The TASIT involves participants viewing vignettes of everyday social interactions and involves three parts. In the Emotion Evaluation Test participants are required to recognize emotional expressions (e.g., happy, surprised, sad, anxious, angry, disgusted and neutral). The Social Inference – Minimal test assesses comprehension of sincere versus sarcastic exchanges. The Social Inference – Enriched test assesses comprehension of lies versus sarcasm. Participants with TBI have been shown to have difficulty recognizing neutral emotions, fear and disgust (McDonald et al., 2003). They are also impaired at judging speaker beliefs when information is not explicitly provided (e.g., difficulty comprehending sarcasm, McDonald et al., 2003; McDonald &Flanagan 2004; McDonald & Saunders, 2005). It should be noted, however, that there are no studies reporting on the regional specifity of lesion effects on this test. It was selected because of its sensitivity to TBI, and its affective content.

Questionnaires

A series of questionnaires was also administered over the two day testing period. The measures were selected allowed for further characterization of the sample.

Big Five Aspect Scales (BFAS) (DeYoung, Quilty, & Peterson, 2007) is a 100-item scale that was used to characterize participants on the Big Five

Personality traits (openness, conscientiousness, extraversion, agreeableness, and neuroticism). This measure was included to help determine how personality moderates outcomes post TBI.

Taylor Manifest Anxiety Scale (Bendig, 1956) is a 20-item scale that is used to measure trait anxiety and is highly correlated with negative affect. This measure was included to help determine if anxiety is an important moderator of outcomes post TBI.

Beck Depression Inventory-II *(BDI)* (Beck, Steer, & Brown, 1996) is a 21-item self-report measure assessing somatic, cognitive and affective components of depression. It has been used to assess depression among people with TBI (e.g., Cantor, Ashman, Schwartz et al., 2005; Glenn, O'Neil-Pirozzi, Goldstein, & Burke, 2001; Seel, Rosenthal, Hammond, Corrigan, & Black, 2003). It was included in this study to help determine the role of depression in coping post TBI.

Sickness Impact Profile *(SIP)* is a 136-item self-report questionnaire assessing the impact of health on everyday life function. The SIP provides both psychosocial and physical domain scores, and has been used in TBI populations (Dawson et al., 2004; Dikmen et al., 1995; Klonoff, Costa, & Snow 1986), and the psychosocial dimension score has been demonstrated to accurately distinguish between head injury and control groups (Dikmen et al., 1993; McLean, Dikmen, & Temkin, 1993; Ownsworth, McFarland & Young, 2000; Temkin, McLean et al., 1988). Both TBI and SO participants completed the SIP.

The Dysexecutive Questionnaire *(DEX)* (Wilson et al., 1997; Burgess, Alderman, Evans et al., 1998) is a 20-item self-report scale measuring real-life executive deficits. DEX subscales include inhibition, intentionality, executive memory, and positive and negative affect. The DEX was completed by both TBI and SO participants.

Reporting of the Data: Estimates of Effect Sizes

A detailed description of each analysis can be found in Chapters 1-3. Importantly, all data are reported using not only traditional *p*-values, but also using estimates of effect sizes (partial eta squared, η_p^2). Partial eta squared is the proportion of the effect plus the error variance that is attributable to the effect, $\eta_p^2 = SS_{effect} / (SS_{effect} + SS_{error})$. Generally speaking, effects between .01 and .06 are considered small, between .06 and .14 moderate, and above .14 large (Cohen, 1988; also see Cohen, 1973). These estimates are of particular importance in clinical studies were sample sizes are often limited.

Descriptives

Demographic Characterization

Data were collected for 24 controls (8 sociodemographic and 16 from the Rotman Research Institute database), 10 people with mild TBI, and 18 people with moderate to severe TBI. Injury severity was determined using Glasgow Coma Scale Scores (GCS) that were obtained by chart review and or neuropsychological reports. Injury severity was characterized by standard criteria (Teasdale & Jennet, 1974), where a GCS of 13-15 was mild, and GCS < 13 was moderate-to-severe. Sample sizes vary from analysis to analysis for various reasons: Four participants chose not to complete all components of the study; four participants were colour blind and were therefore not able to participate in the neuropsychological tests where colour perception was necessary; one participant was severely allergic and refused the smell identification test; and finally, there were technical difficulties with BioPac such that components of the physiological recordings were not useable for 6 of the 52 study participants.

As seen in Table 1, there were no group differences in years of education, fluid intelligence, or years since injury. The mild TBI group was older, $F(2, 49) = 4.0$, $p<.05$, $\eta_p^2 = .139$, than both the control and moderate-to-severe groups. The moderate-to-severe group had lower estimated premorbid verbal intelligence, $F(2, 48) = 3.4$, $p<.05$, $\eta_p^2 = .123$, than both the controls and the mild group. For the results that follow in subsequent chapter, these factors have been co-varied from the analysis when there were theoretical reasons to suspect these group differences could drive effects.

Table 1. Demographic and injury severity characteristics. Effect sizes represent the differences between control and TBI groups.

	Control (24)	**Mild (10) GCS 13-15**	**Mod-Sev (18) GCS < 13**
Sex			
Male	10	3	12
Female	14	7	6
Mean Age in years (SD)	38.7 (17.4)	54.1 (13.3) η_p^2 = .162	38.8 (13.6) η_p^2 = 0
Mean Years of Education (SD)	15.8 (3.4)	16.0 (4.0) η_p^2 =.001	15.3 (3.0) η_p^2 = .007
Mean verbal score on the Shipley (SD)	32.6 (4.3)	32.5 (6.3) η_p^2 = 0	28.3 (6.8) η_p^2 = .139
Mean score on the Raven Progressive Matrices (SD)	18.2 (7.3)	14.0 (5.8) η_p^2 = .078	15.2 (7.8) η_p^2 =.038
Mean Time since Injury in Months (SD)	na	76 (45.7)	153 (117)
		η_p^2 = .132	

Neuropsychological Function

One-way ANOVA's were used to assess the differences between controls and people with mild TBI, and moderate-to-severe TBI in their working memory, executive function, and affective function. Means, standard deviations and estimates of effect sizes are displayed in Appendix B. There was considerable variability in performance, particularly among the TBI participants. Group differences were most prominent between the control and moderate-to-severe group, with the latter group generally displaying lower neuropsychological functioning.

Personality

One way ANOVA's were used to assess differences between controls and people with mild TBI, and then controls and moderate-to-severe TBI in their personality. Means, standard deviations and estimates of effect sizes are displayed in Appendix C. There were differences between the control and mild groups on a number of variables, but no differences between controls and the moderate-to-severe group.

Psychiatric Status

One way ANOVA's were used to assess differences between controls and people with mild TBI, and then controls and people with moderate-to-severe TBI in their psychiatric status. Means, standard deviations, and estimates of effect sizes are displayed in Appendix D. For these analyses, group differences were driven by the mild group, who reported experiencing more depression and anxiety than the control group.

Physiological Reactivity

One way ANOVA's were used to assess differences between controls and people with mild TBI, and then controls and people with moderate-to-severe TBI in their physiological response to stress (i.e., heart rate, heart rate variability, skin conductance level and cortisol). Means, standard deviations, and estimates of effect sizes are displayed in Appendix E. There were no differences between groups.

Data Reduction

The neuropsychological tests, questionnaires, and physiological recordings yielded a very large dataset. To best take advantage of this rich data set, variables were collapsed together to form meaningful composite scores for further statistical analysis. To do this, correlation analyses were first performed between scores for which there was a theoretical reason to suspect a relation. For example, in order to create a composite measure of executive function, correlation analyses were performed on the neuropsychological tests that index executive function. The scores that were most highly correlated with each other, and were *not* correlated with scores on tests thought to be sensitive to VMPFC functioning, were then transformed to z scores (flipping the sign of the z score where appropriate so that for all measures, higher scores reflect better performance), and averaged to form a composite executive function score fore each participant. This procedure was repeated for measures of ventral function, questionnaires, and physiological measures. As depicted in Appendix F, these analyses yielded five composites; executive function, VMPFC function, reactivity, openness/agreeableness, and sociodemographic. The components of each of these scores are listed below, along with a brief description of their meaning.

1. Executive Function: (Stroop IN-CN)/CN, Alpha Score, Brown Peterson 18 second delay, Zoo Map Sequence Score, and Wisconsin Card Sorting Test set loss. This score reflects an individual's overall executive functioning, as these tests are sensitive to DLPFC damage. These measures were selected based on theory (see Stuss & Levine, 2002 for review), and because they were not related to VMPFC tests.

2. VMPFC Function: Smell identification test, Object Alternation Errors, Intradimensional Shifting Reversal Errors. This score reflects an individual's overall affective functioning, as these tests are sensitive (if not specific) to VMPFC damage. These measures were included in the VMPFC composite because they have been demonstrated to reflect ventral integrity; they were correlated to one another; and they were *not* correlated with measures of executive function.

3. Reactivity: Beck Depression Inventory, Taylor Manifest Anxiety Scale, State – Anxiety Inventory post BPST, Neuroticism, and Conscientiousness. This score reflects an individual's overall sensitivity to stress. The tests composing this composite score reflect emotional sensitivity and a tendency to experience negative emotions, but also a tendency to show self-discipline and regulate impulses.

4. *Agree/Open:* Opennness and Agreeableness. This score reflects an appreciation for emotion, adventure and unusual ideas, and also a tendency to be compassionate and cooperative.

5. *Sociodemographic*[1]*:* Shipley, GCS, and Age. This score was calculated for TBI participants only and reflects an individual's overall socoidemographic status (e.g., higher means better premorbid intelligence, younger age, and higher GCS score).

The physiological measures did not correlate with each other, making it impossible to create composite scores in the same way as other measures. As described above, physiological measures were collected across five blocks of the BPST (baseline, preparation period, during the speech, during the mental arithmetic, and baseline 2). In order to condense these data, difference scores between block 3 (speech) and block 1 (baseline) were calculated. This single score provided an index of physiological responsivity (i.e., the difference between

[1] The author acknowledges that collapsing these variables is unconventional, as each of these factors may independently relate to coping, and their relation to each other may be an artefact of this particular sample. When this composite variable was found to be related to coping behaviour, the composite was deconstructed (see Chapter Four).

resting baseline and responses during the actual task). Cortisol was collected at three times; Time 1: prior to the BPST, Time 2: immediately following the BPST, and Time 3: 20 minutes following the BPST. A difference score between Times 3 and 1 was calculated for each participant. This score reflects a change in cortisol from baseline (Time 1), to the height of the stress response (Time 3). The Time 3 cortisol measure was selected because there is a 20 minute delay before changing free (biologically active) concentrations of cortisol are present in the saliva (see Fleming, Steiner & Corter, 1997; Krpan, Coombs, Zinga et al., 2005; Stallings, Fleming, Corter et al., 2001).

CHAPTER THREE

The Effects of Psychosocial Stress on Coping Behaviour Post Traumatic Brain Injury

Rationale

Many people who sustain traumatic brain injuries have poor outcomes (e.g., Christensen et al., 2008; Crepeau & Scherzer, 1993; Dawson et al., 2004; Dikmen et al., 1995; Jennett et al., 1981; Rappaport et al., 1989; Ruttan et al., 2008; Stuss et al., 1995; Till et al., 2008), which have been related to the use of avoidant coping (Anson & Ponsford, 2006; Malia et al., 1995; McMillan et al., 2003; Leach, Frank, Bouman, & Farmer, 1995; Tomberge, Toomela, Pulver, & Tikk, 2005). This relationship exists despite the fact that there are no measurable differences in self-reported coping style between matched controls and people with TBI (Curran et al., 2000; Dawson et al., 2006; Krpan et al., 2007). The purposes of this study were first to re-examine differences between control and TBI participants in self-reported and significant other (SO) reported coping, and second to directly observe coping behaviour during a simulated real-world psychosocial stress test. It was hypothesized that, consistent with earlier research, there would be no differences between controls and people with TBI in self-reported and significant other reported coping. Second, it was hypothesized that people with TBI would engage in more avoidant coping behaviour than the control group during a simulated real-world stress test.

Methods

The precise methods employed in this study are described in the General Methods section of this thesis. Briefly, people with TBI, their SO's, and matched controls completed the Ways of Coping Questionnaire to assess reported coping style. The BPST was administered to the control and TBI groups as a means of directly assessing coping behaviour (i.e., planful and avoidant). The State-Anxiety Inventory was administered immediately prior to and following the BPST to index subjective stress throughout the task.

Results

Self and Significant-Other Reported Coping

As depicted in Table 2 [2], there were no significant differences between controls and people with TBI in their self-reported coping on the WOC. This finding is consistent with earlier studies that have also demonstrated no measureable differences in self-reported coping following TBI (e.g., Curran et al., 2000; Dawson et al., 2006; Krpan et al., 2007). Estimates of effect sizes also revealed very few differences between groups, with the exception that the mild group reported employing moderately more self-controlling, seeking social support, and escape avoidant coping. There were significant differences in mild SO reported coping, where SO's reported that their loved one engaged in less planful problem solving and positive reappraisal than did the control group.

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[2] Two SO's (one in the mild TBI group and the other in the mod-sev TBI group) refused to complete the WOC, and one person in the mild TBI group withdrew from the study before completing the WOC.

Moderate-to-severe SO's reported their loved ones seeking more social support than the control group.

Table 2: Mean (SD) group differences on the WOC.
η_p^2 are relative to the control group
Bolded items represent $p < .05$

	Control (n=24)	Mild SO (n= 8)	Mild TBI (n=9)	Mod/Sev SO (n = 17)	Mod/Sev TBI (n=18)
Confrontive	3.5 (3.3)	2.8 (2.5) η_p^2 =.009	5.0 (4.4) η_p^2 =.032	5.8 (4.6) η_p^2 =.075	4.6 (2.5) η_p^2 =.031
Distancing	6.0 (3.9)	3.6 (2.2) η_p^2 =.084	7.2 (3.3) η_p^2 =.018	5.8 (4.1) η_p^2 =.002	6.1 (3.2) η_p^2 =.000
Self-Controlling	8.7 (4.7)	6.5 (3.6) η_p^2 =.047	11.6 (2.9) η_p^2 =.086	7.9 (4.7) η_p^2 =.007	8.6 (4.6) η_p^2 =.000
Seeking Social Support	5.7 (3.4)	4.9 (4.7) η_p^2 =.010	9.0 (6.6) η_p^2 =.098	**8.8 (3.4) η_p^2 =.160**	7.1 (2.8) η_p^2 =.044
Accepting Responsibility	3.5 (3.1)	3 (3.2) η_p^2 =.006	4.2 (3.4) η_p^2 =.010	3.9 (3.5) η_p^2 =.003	4.1 (2.3) η_p^2 =.010
Escape-Avoidant	5.6 (5.2)	5.1 (5.0) η_p^2 =.002	9.1 (3.8) η_p^2 =.095	7.2 (5.3) η_p^2 =.021	4.9 (3.2) η_p^2 =.006
Planful Problem-Solving	7.7 (4.2)	**4.2 (3.2) η_p^2 =.127**	6.5 (4.8) η_p^2 =.014	7.0 (4.0) η_p^2 =.007	7.9 (3.2) η_p^2 =.001
Positive Reappraisals	7.0 (5.5)	**2.5 (3.7) η_p^2 =.129**	8.3 (7.7) η_p^2 =.010	6.2 (5.7) η_p^2 =.005	6.1 (5.2) η_p^2 =.006
Total Score	6.0 (2.9)	4.1 (2.1) η_p^2 =.085	7.1 (3.1) η_p^2 =.057	6.6 (3.0) η_p^2 =.010	6.2 (2.6) η_p^2 =.002

Coping Behaviour on the Baycrest Psychosocial Stress Test

Perceived Level of Stress

As depicted in Figure 2, repeated measures analyses of the pre and post State-STAI scores revealed an effect of time, F(1, 46) = 45.04, $p< .001$, $\eta_p^2 = .495$ and severity, F(2, 46) = 7.11, $p< .05$, $\eta_p^2 = .236$, but no significant interaction. Univariate analyses of the pre and post STAI anxiety scores revealed that the mild TBI reported experiencing more anxiety than both the control and moderate-to-severe group prior to the BPST, F(2, 46) = 9.60, $p<.05$, $\eta_p^2 = .294$. Following the BPST, the mild group still reported experiencing more anxiety than either of the other groups, F(2, 47) = 2.83, $p=.069$, although the effect size was smaller, $\eta_p^2 = .108$. When age, depression and anxiety were entered as co-variants into the univariate analyses, these effects were still present.

Summary: All groups reported experiencing greater subjective anxiety after the BPST than before it. In comparison to the control and moderate-to-severe group, the mild group reported more anxiety both prior to the BPST, and following the test.

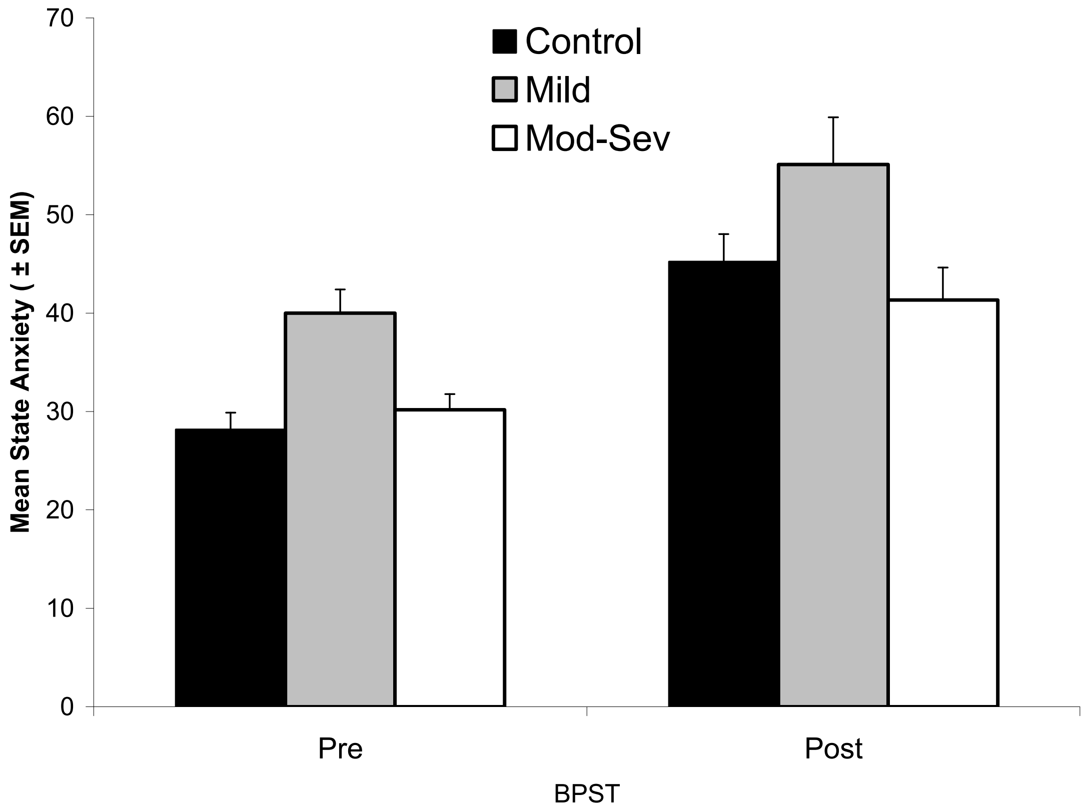

Figure 2: Mean state anxiety on the STAI (+SEM) before and after the BPST.

Analysis of Behaviour

The following analyses were conducted by injury severity. The data from two TBI participants (1 mild, 1 moderate-to-severe), and two controls are not included in these analyses because the video recording for those participants was of too poor quality to code. All post-hoc analyses were done using Tukey's HSD, *p* values were set at .05, and estimates of effect sizes are reported. The total scores for planful and avoidant behaviour were first analyzed, followed by an analysis of each of the subcategories composing the total scores. The latter analysis was performed for 1) descriptive purposes, and 2) to ensure that total scores were not being driven by one particular behaviour.

Planful Behaviour: Figure 3 shows the distribution of planful behaviours for each group. There was a significant effect of group, F(2, 45) = 5.30, *p* <.05, η_p^2 = .191, reflecting significantly greater total planful behaviour in the control than moderate-to-severe group (*p* = .026). This effect remained when co-varying for premorbid intelligence.

To determine if there was one single behaviour that was driving the effects of the total planful score, each of the components of the planful score were analyzed separately. There were significant differences between groups on Writing Total F(2, 45) = 3.801, *p* <.05, η_p^2 = .145; and Thinking and writing F(2, 45) = 3.71, *p* <.05; η_p^2 = .142). Post-hoc analyses indicated that these differences were mainly carried by the moderate-to-severe group, who engaged in less writing and less thinking and writing than did the control group (p = .026; p=.024, respectively), and these effects remained when co-varying premorbid intelligence.

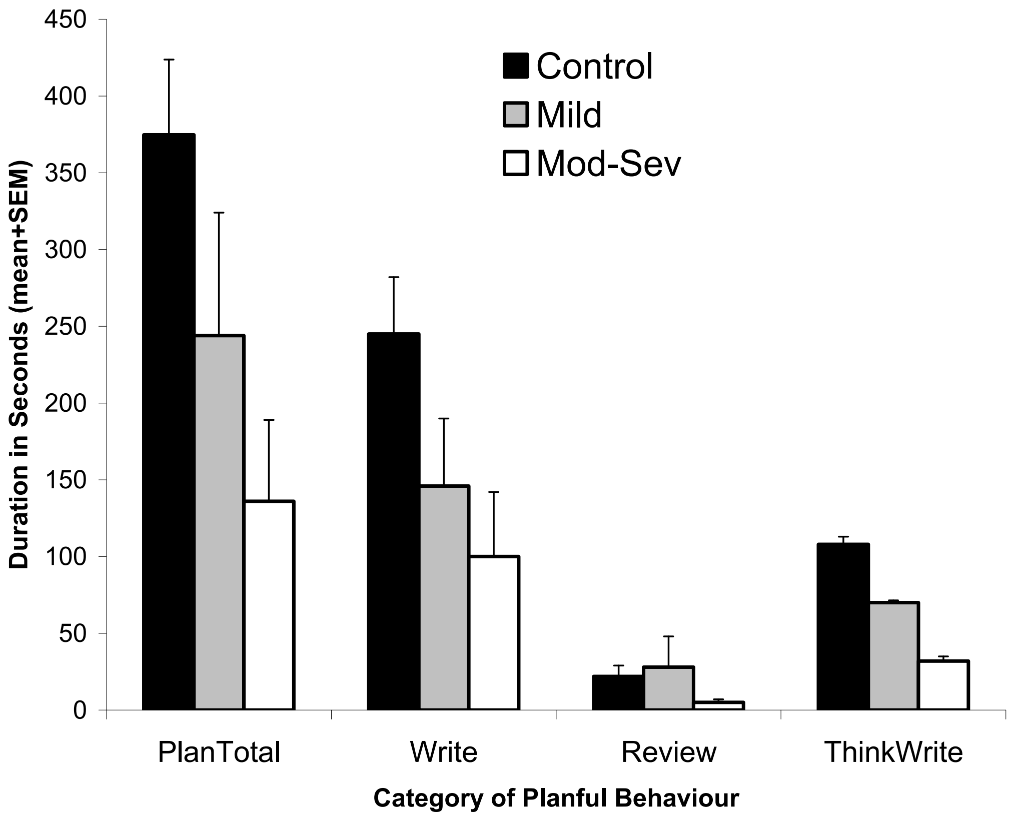

Figure 3: Mean differences (+SEM) in planful behaviour.

Avoidant Behaviour: Figure 4 shows the distribution of avoidant behaviours for each group. There was a significant group difference for total avoidant behaviour, F(2, 45) = 4.93, *p* <.05, η_p^2 = .18. Post-hoc analyses indicated differences between control and moderate-to-severe groups, where patients in the latter group engaged in more avoidant strategies (*p* =.02). When premorbid intelligence was entered as a covariate, this effect remained significant.

To determine if there was one single behaviour that was driving the effects of the total avoidant score, each of the components of the avoidant score were analyzed separately. There were significant differences between groups on time spent reading magazines, F(2, 45) = 4.03, *p* <.05, η_p^2 = .152. Post-hoc analyses indicated that this difference was also carried by the moderate-to-severe group, who spent significantly more time than the control group looking through magazines (*p* =.02), and this effect remained when controlling for premorbid intelligence (*p* = .089, but η_p^2 = .106). There were no significant differences on any of the other sub-category scores based on *p* values alone, however, estimates of effect sizes revealed small differences in playing with puzzles, F(2, 45) = 1.21, *p* = .318, η_p^2 = .051, other avoidant behaviours, F(2, 45) = 1.01, *p* = .372, η_p^2 = .043, and staring into space, F(2, 45) = 1.24, *p* = .298, η_p^2 = .052. In all cases, the moderate-to-severe group displayed more avoidant behaviour than did either the control or mild groups.

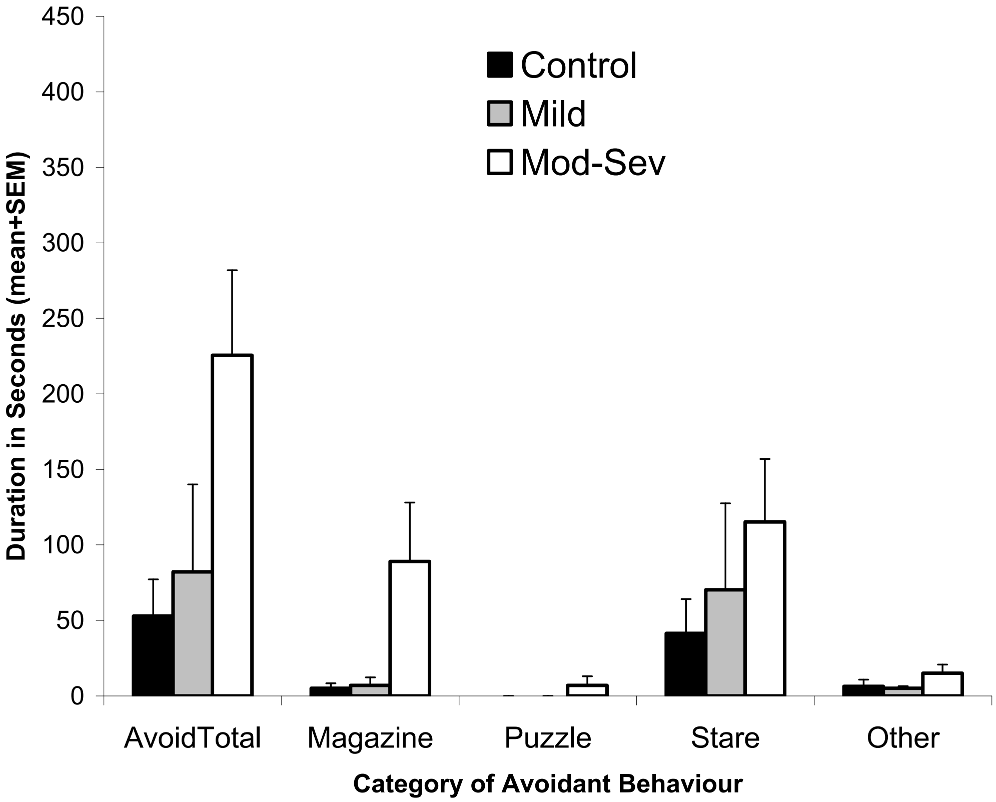

Figure 4: Mean difference (+ SEM) in avoidant behaviour.

Group X Coping Interaction: As depicted in Figure 5, repeated measures analysis examining total time engaging in planful strategies and total time engaging in avoidant strategies indicated that there was a significant effect of coping behaviour, $F(1, 45) = 6.13$, $p < .05$, $\eta_p^2 = .120$, and a significant group X coping interaction, $F(2, 45) = 6.884$, $p < .05$, $\eta_p^2 = .234$, but no significant group effect ($\eta_p^2 = .036$).

Repeated measures analyses were then performed within group. Both the control and mild TBI groups showed an effect of coping behaviour, indicating they spent more time engaging in planful than avoidant behaviour (control group: $F(1, 21) = 25.92$, $p<.001$, $\eta_p^2 = .552$; mild group: $F(1, 9) = 1.982$, $p = .197$, but $\eta_p^2 = .199$). The moderate-to-severe group, however, did not display an effect of coping behaviour $F(1,16) = .864$ $p = .367$, but estimates of effect sizes suggested that they actually engaged in more avoidant than planful coping, $\eta_p^2 = .051$.

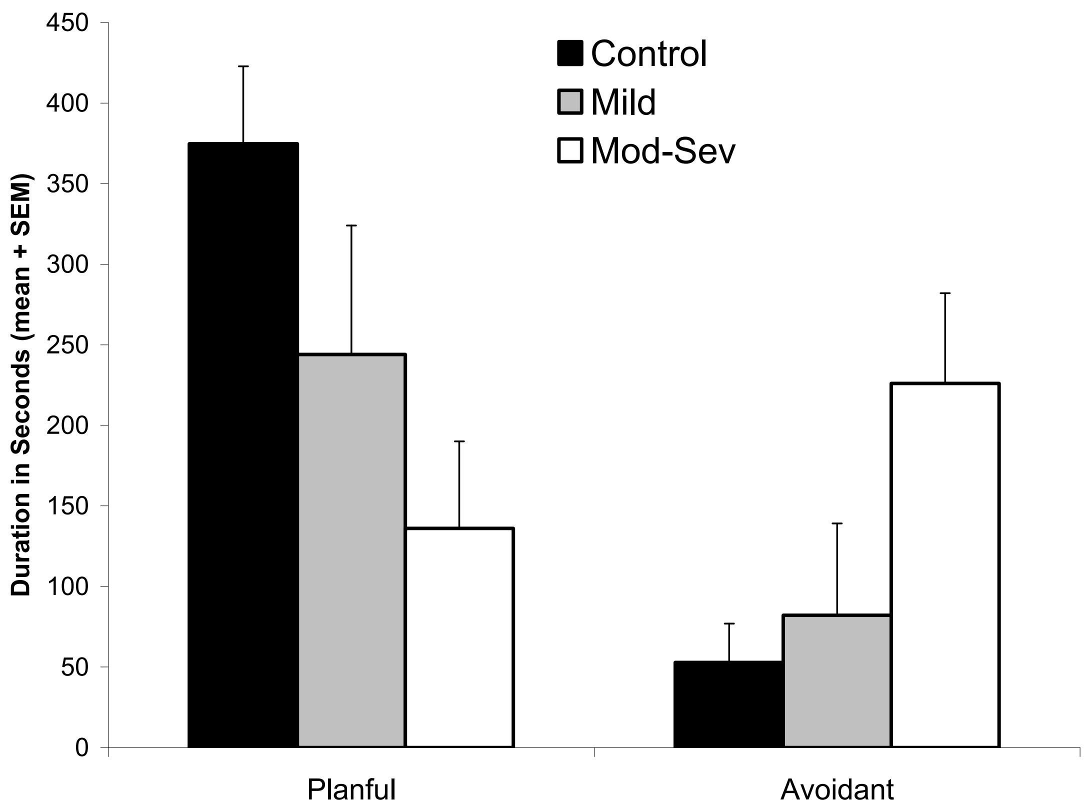

Figure 5: Mean (+ SEM) group differences engaging in planful and avoidant coping behaviour.

Speech Performance: As depicted in Figure 6, there was also a significant effect of group for total speech performance F(2, 44) = 23.13, p <.001, η_p^2 = .513. Post-hoc analyses indicated a significantly better speech performance in controls than both the mild TBIs (p=.014) and the moderate-to-severe TBI's (p <.001). These effects remained when controlling for age, premorbid intelligence, depression, and anxiety. Moreover, planful coping related to better speech performance ρ(**17**)=0.398, and avoidant coping related to lower speech performance ρ(**17**)= -0.408.

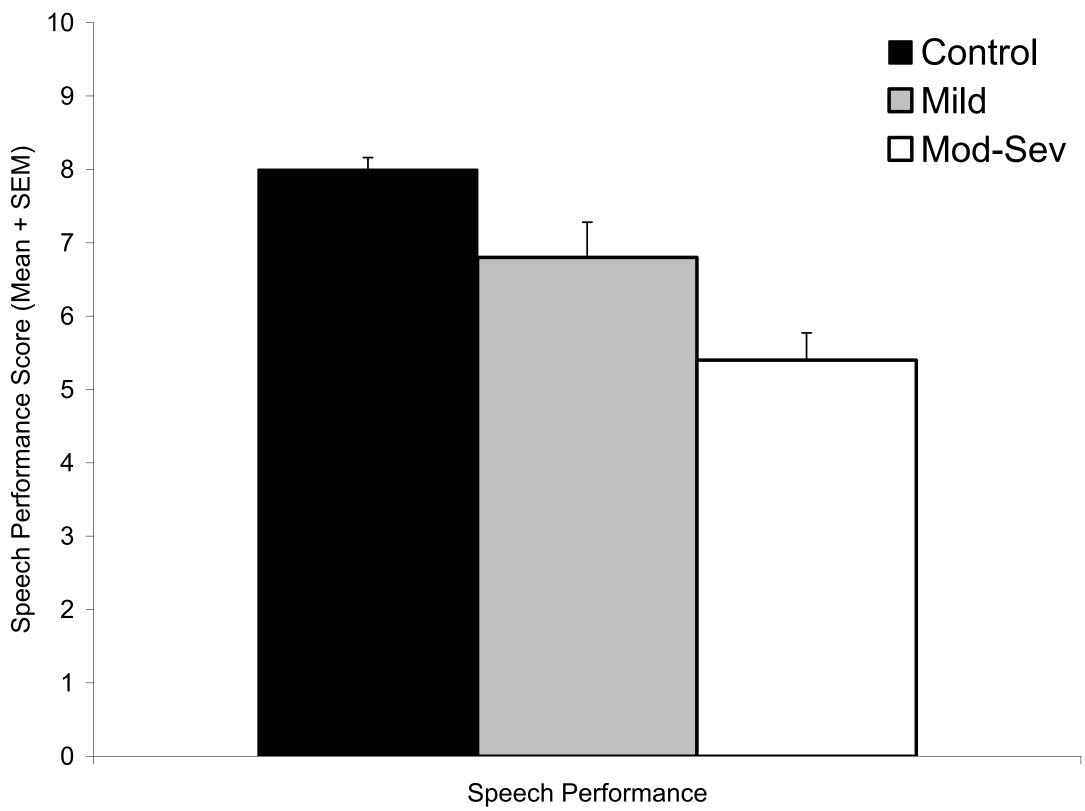

Figure 6: Mean difference (+ SEM) in speech performance scores.

Summary: The moderate-to-severe group engaged in less planful and more avoidant behaviours than did the control or mild group. Moreover, a coping X group interaction revealed that the control and mild groups engaged in more planful than avoidant coping, where the moderate-to-severe group showed the opposite pattern (that is, more avoidant than planful behaviour). Finally, the control group performed better than the TBI groups on the speech. These analyses suggested that there is a fundamental difference in the way a person copes following moderate-to-severe TBI, specifically, and that this may have consequences for performance on a given task.

The Relationship between Self-Reported and Observed Coping Behaviour

The relationship between self and SO-reported coping on the WOC and actual coping behaviour on the BPST was evaluated in the entire group [3](22 controls, 8 mild TBI, 17 moderate-to-severeTBI, 7 mild SO's, and 16 moderate-to-severe SO's) by performing Spearman's correlations. As displayed in Table 3, there were no relationships between self-reported coping and observed coping behaviour. There were also no relationships between significant other reports of coping and observed coping behaviour, with the exception that SO reports of planful behaviour were *negatively* related to observed planful behaviour on the BPST.

[3] The quality of video recording was too poor to code behaviour for one control participant, one mild TBI participant, and one moderate-to-severe participant
One control participant did not complete the BPST due to technical difficulties and time constraints
One mild TBI participant withdrew from the study before completing the WOC
One mild SO participant and one moderate-to-severe SO participant refused to complete the WOC

Table 3: Correlation coefficients (Spearman's *rho*) between self and significant other reported coping on the WOC and coping behaviour on the BPST (n = 47 for group data; n = 23 for SO data).

	BPST Planful Behaviour	**BPST Avoidant Behaviour**
WOC – Escape Avoidant	-.100 *p* = .252	.026 *p* = .431
WOC – Planful Problem Solving	.065 *p* = .331	.021 *p* = .444
SO WOC – Escape Avoidant	-.011 *p* = .481	-.036 *p* =436
SO WOC – Planful Problem Solving	**-.409** ***p* =.026**	.035 *p* =.437

Overall Summary: The BPST induced self-reported anxiety in all three groups. During the preparation period, the control and mild groups engaged in more planful than avoidant behaviour, and the moderate-to-severe group engaged in more avoidant than planful behaviour. Overall, the control group performed better speeches than did either of the TBI groups, even when controlling for age, premorbid intelligence, anxiety and depression. Finally, there were no relations between self-reported coping and behaviour on the BPST. Significant other reports of planful coping were negatively related to planful behaviour on the BPST. These data suggest that the BPST is more sensitive to coping following TBI than are self and SO reports of coping. They also suggest that moderate-to-severe TBI can result in a unique pattern of coping that is not present following less severe injuries.

Discussion

This is the first study, to the author's knowledge, to directly observe coping behaviour during a simulated real-world stress test following TBI. There are three main finding of this study. First, there were no differences between groups in self reported coping on the WOC; second, the BPST induced subjective stress in all three groups; third, people with moderate-to-severe injuries displayed more avoidant and less planful behaviour, where the control and mild groups displayed the opposite pattern. Each of these findings is discussed in turn, below.

There were no significant differences between the controls, mild and moderate-to-severe groups in their self-reported coping style on the WOC. This finding is consistent with studies that have reported no differences in self-reported coping (Curran et al., 2000; Dawson et al., 2006; Krpan et al., 2007). This finding is not so surprising given the reported problems of self-awareness following TBI (e.g., Prigatano, 2005; Stuss, 1991). To address this problem, a close friend or family member of each TBI participant completed the WOC on their behalf. These data also revealed few significant differences between groups in coping style. This finding may be interpreted in several ways. It may reflect that there actually are few differences in coping following TBI. This interpretation is not supported by the behavioural data presented in this study.

Alternatively, the lack of large group differences in SO reports may exist because it is difficult for an outside person to accurately assess coping style. Many of the items on the WOC refer to meta-cognitive coping processes (e.g., I prayed; I reminded myself how much worse things could be; I hoped for a miracle; I told myself things that helped me feel better). As many SO participants commented, it is difficult to assess the internal thoughts of another person. The author suggests then that the lack of group differences in SO reports reflects an insensitivity of the questionnaire. This hypothesis is supported by the finding that SO-reported coping did not positively relate to actual coping behaviour on the BPST. In fact, SO reports of planful coping were negatively correlated with actual planful coping behaviour. This suggests that the SO group thought the TBI groups were engaging in more planful behaviour than they actually were.

The objective of the present study was not to evaluate the level of agreement between TBI and SO reports of coping. The author did, however, observe some unusual trends in the data. The patterns present in Table 2 (showing self and SO reported WOC scores) suggest greater proxy-participant agreement in the moderate-to-severe group than in the mild group. While it has been demonstrated that there can be low proxy-TBI agreement following mild TBI (Ocampo, Colantonio, & Dawson, 1997), this is not consistent with a literature that suggest greater proxy-participant agreement among people with mild as compared to moderate-to-severe TBI (Cusick, Brooks & Whiteneck, 2001; Dawson, Markowitz & Stuss, 2005), or that injury severity is irrelevant (Lanham, Weissenburger, Schwab & Rosner, 2000; Seel, Kreutzer, & Sander, 1996).

These unusual patterns may be a function of a small mild TBI sample size, or alternatively, it may be a function of the type of proxies responding. Spousal proxies have been demonstrated to have the highest agreement to TBI reports of outcome (Dawson, Markowitz, & Stuss, 2005).

The BPST induced subjective anxiety, as indexed by state-STAI scores, in all three groups. This is an important finding because the BPST is the amalgamation of two pre-existing stress tests, and it was necessary to demonstrate that the experience was indeed stressful. As depicted in Figure 2, the mild group entered the stress test with higher state anxiety, and also reported greater state anxiety following the test than either the control or moderate-to-severe groups. This heightened anxiety, however, does not seem to have come at a tremendous cost to the mild group, given that their patterns of behaviour were not different from those of the controls.

The objective evaluation of coping behaviours in a simulated real-world stressful circumstance showed that the moderate-to-severe group displayed an altered pattern of coping compared to the control and mild TBI groups. While control and mild TBI group clearly engaged in more planful than avoidant behaviour, the moderate-to-severe TBI group showed less of dissociation between coping behaviour. That is, the control and mild TBI groups showed a strong bias towards planful coping, and engaged in very little avoidant behaviour at all[4]. The moderate-to-severe group engaged in roughly equal amounts of planful and avoidant behaviour, based on *p*-values, but numerically and based on

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[4] It should be noted that this pattern was less robust in the mild TBI group as compared to the control group.

estimates of effect sizes, they engaged in more avoidant than planful behaviour (η_p^2 = .051; see Figure 5), a pattern *opposite* to the other groups. The moderate-to-severe group also performed more poorly than both the control and mild group on the speech itself. These group differences in performance suggest that the moderate-to-severe group did not strategically engage in planful coping during the preparation period, to the detriment of speech performance. Correlations between speech performance and planful and avoidant coping within the moderate-to-severe group support this interpretation (planful related to better performance, and avoidant to worse performance).

The implications of these findings are very promising; the BPST is sensitive to differences in coping that are not evident by self or SO-report. Moreover, these data also suggest that self and SO-reported coping data have no relation to behaviour in the real world.

A remaining question concerns what drives this effect in the moderate-to-severe group. There are a number of factors that could account for these differences in coping behaviour, including injury severity, neuropsychological function, responsivity to stress, depression, anxiety and personality. Chapter four aims to identify the best co-variates of this effect in the moderate-to-severe group.

CHAPTER FOUR

The Moderators of Altered Patterns of Coping Behaviour Following Moderate-to-Severe Traumatic Brain Injury

Rationale

In the previous chapter it was demonstrated that the moderate-to-severe TBI group exhibited a unique pattern of coping, such that they engaged in more avoidant than planful behaviours on the BPST. An important question concerns the reasons why this group displays this altered patterns of coping. Injury severity, neuropsychological function, responsivity to stress, depression, anxiety and personality may all be important factors influencing coping. The purpose of this chapter is to investigate the possible covariates of coping behaviour in the group of TBI participants that displayed altered patterns of coping in Chapter Three, namely, the moderate-to-severe TBI group.

Methods

The precise methods of this study are described in the general methods section of this thesis. Briefly, participants completed: 1) a neuropsychological test battery assessing premorbid intelligence, fluid intelligence, working memory, executive function, and VMPFC function; 2) a series of questionnaires assessing depression, anxiety and personality; and 3) the BPST during which time their heart-rate, heart-rate variability, skin conductance level and cortisol levels were measured (see General Methods section for details).

One-way ANOVA's were used to assess the differences between controls and people with TBI in their neuropsychological function, psychiatric function, personality, and physiological response to stress. Means, standard deviations and estimates of effect sizes are displayed in Appendices B – D.

As described in the General Methods section, to reduce the large number of possible covariates that were collected, correlation analyses between covariates were conducted (see Appendix F for correlation matrices). Z scores were calculated for variables that were highly related, and then averaged. In this way, it was possible to reduce a large dataset into smaller meaningful components. The composite scores included in the analyses below are as follows: Executive Function, VMPFC Function, Reactivity, Sociodemographic, and Openness/Agreeableness. As there were no correlations between physiological measures, heart rate, heart rate variability and skin conductance level data is represented by difference scores between Blocks 3 and 1 on the BPST, and between Time 3 and Time 1 for cortisol. In addition to these composite and difference scores, measures of fluid intelligence and time since injury were also included in these analyses.

Results

The analyses conducted for this Chapter can be broken down into five components. First, correlational analyses were performed to determine the relations between coping behaviour and potential moderator variables (e.g., neuropsychological function, psychiatric status, personality, and physiological responsivity to stress). Second, regression analyses were performed to determine which variable, or combination of variables, were *most* predictive of coping behaviour. Third, analysis of group variability in coping behaviour was conducted to explore the possibility of distinct groups within the moderate-to-severe sample. Fourth, analysis of co-variance was performed to determine if variables of interest could eliminate group differences in behaviour. Each of these is presented, in turn, below. Sample size is 17 for all analyses with the exception of those including SCL (n = 15 due to technical problems with BioPac), and fluid intelligence (n = 16 due to computer malfunction).

The Relationship between Coping Behaviour and Neuropsychological Function, Psychiatric Status, Personality, and Physiological Response to Stress

To assess the relationship between coping behaviour and variables of interest, Spearman's correlations were performed within the moderate-to-severe group. As seen in Table 4, there were a number of relationships between coping and cognitive, psychiatric, personality and physiological variables. Planful coping was positively related to executive function, fluid intelligence, reactivity, and changes in heart rate and skin conductance response. Planful coping was negatively related to openness/agreeableness and changes in heart-rate variability. Avoidant coping was negatively related to executive function, changes in heart-rate and skin conductance level.

Table 4: Correlation coefficients (Spearman's *rho*) between coping behaviour and variables of interest. Bolded items represent large effect sizes ($rho^2 > .14$)

	Planful Coping	Avoidant Coping
Executive Function	**.380** ***p* = .13**	**-.584** ***p* = .01**
VMPFC Function	-.106 *p* = .69	.121 *p* = .64
Demographic Composite	.247 *p* = .34	-.054 *p* = .84
Raven Standard Progressive Matrices	**.375** ***p* = .152**	-.150 *p* = .58
Time Post	.109 *p* = .68	-.244 *p* = .35
Reactivity	**.563** ***p* = .02**	.029 p = .91
Open/Agree	-.273 *p* = .29	.222 *p* = .39
Heart Rate Change	**.462** ***p* = .06**	**-.462** ***p* = .06**
Heart Rate Variability Change	**-.395** ***p* = .12**	.334 *p* = .19
Skin Conductance Level Change	**.411** ***p* = .13**	**-.495** ***p* = .06**
Cortisol Change	-.064 *p* = .81	.210 *p* = 419

Summary: In the moderate-to-severe group, better executive performance and more physical and psychological reactivity to stress related to planful coping. Lower executive functioning and less physiological and psychological responsivity to stress related to avoidant coping. These data suggest that in order to be planful, an individual must not only have cognitive resources, but must also be reactive to stress.

Exploring the Strongest Predictors of Coping through Stepwise Regression

Based on the patterns of correlations described above, it was evident that a number of factors are related to coping following moderate-to-severe TBI. An important remaining question was which of these factors best predicts coping behaviour. Stepwise linear regression analyses were performed to explore the greatest predictors of coping. The limitations of stepwise regression are acknowledged – the model produced by stepwise regression is contingent on the variables entered into the analysis. Moreover, it is acknowledged that hierarchical regression would have been the ideal regression method to accurately predict coping. In this way, it could be argued that one particular variable accounted for significant variance in the model above and beyond other variables. Hierarchical regression, however, did not yield significant models – likely due to the small sample size and due to power reductions with each additional variable entered into the model. By conducting stepwise regression, it was possible to identify critical variables that contributed to coping.

Predicting Planful Coping: Stepwise regression was first performed to predict planful coping behaviour. The variables that were identified as being strongly related to planful coping in the correlational analyses (i.e., large effect sizes) were entered into the model. These included executive function, fluid intelligence, reactivity, heart rate change, heart rate variability change, and skin conductance level change. Results indicated that reactivity together with skin conductance change were the greatest predictors of planful coping, $F(2, 11) = 19.58$, $p<.001$, $R^2 = .781$, B for reactivity = .710, B for skin conductance change = .392.

Predicting Avoidant Coping: Stepwise regression was then performed to predict avoidant coping behaviour. The variables that were identified as being strongly related to avoidant coping in the correlational analyses (i.e., large effect sizes) were entered into the model. These included executive function, reactivity, heart rate change, and skin conductance level change. Results demonstrated that executive function, alone, was the greatest predictor of avoidant coping, $F(1, 13) = 8.43$, $p<.05$, $R^2 = 393$, B = -.627.

Summary: Psychological reactivity and skin conductance change were the best predictors of planful coping. Executive function, alone, was the best predictor of avoidant coping. These analyses suggest that in order to be planful, it is critical to be responsive, psychologically and physically, to stress. They also suggest that avoidance is driven by a lack of cognitive resources (i.e., participants avoided because they did not have the executive functions to plan).

Fractionating the Moderate-to-Severe Group Based on Coping Behaviour

Variability is a known obstacle in clinical research (e.g., Dawson et al., 2004; Stuss & Binns, 2008; Stuss, Murphy, Binns & Alexander, 2003). In Chapter Three, it was demonstrated that the moderate-to-severe TBI group was clearly different from the control and mild groups. A remaining concern was the possibility of variability *within* the moderate-to-severe group. In order to determine if all people with moderate-to-severe TBI in this sample displayed more avoidant and less planful behaviour, a percent avoidant score was calculated for each participant using the following formula: total avoidant time / (total time avoidant + total time planful) * 100. A histogram displaying the percent of time spent avoiding demonstrated a bimodal distribution. Eleven of the moderate-to-severe TBI participants engaged in predominantly avoidant behaviour (and drove the interaction described in Chapter Three), while six engaged in predominantly planful behaviour[5]. As depicted in Figure 7, repeated measures analyses revealed a coping X group (planners vs avoiders) interaction within the moderate-to-severe TBI group, $F(1,15)= 35.83$, $p<.001$, $\eta_p^2 = .705$. These analyses clearly demonstrated that there were two groups of copers within the moderate-to-severe group; those who engaged in more planful than avoidant coping (planners), and those who engaged in more avoidant than planful behaviour (avoiders).

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[5] These analyses were also performed in the control and mild TBI groups. In the control group, only 5 people were classified as avoidant and 17 as planful. In the mild TBI group only 2 people were avoidant, and 7 were planful. It was, therefore, not possible to conduct these analyses in the mild TBI group.

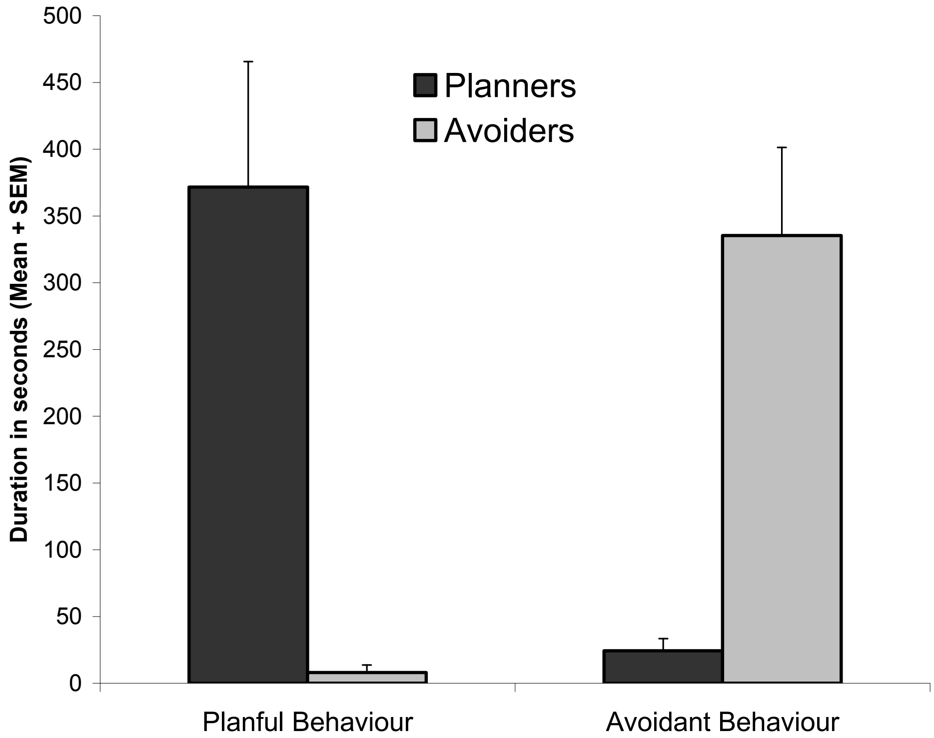

Figure 7: Mean difference (+ SEM) engaging in planful and avoidant behaviour in the moderate-to-severe group

Summary: Analysis of variability in the moderate-to-severe group revealed two groups of copers: planners, and avoiders. Six people were classified as predominately planful, where eleven were classified as predominantly avoidant. An important question concerned what other factors distinguish these groups from each other (e.g., neuropsychological performance, psychological and physical reactivity etc).

Analysis of Group Differences between Planners and Avoiders

Univariate analyses were conducted on a number of variables of interest to determine if there were group differences between those people with moderate-to-severe injuries who were planners, and those who were avoiders. These analyses, along with estimates of effect sizes, are described below.

As displayed in Table 5, there were significant differences between avoiders and planners with moderate-to-severe injuries. Relative to avoiders, planners had higher executive function, $F(1,15) = 10.14$, $p<.05$, $\eta_p^2 = .403$, better sociodemographic[6] status $F(1, 15) = 2.55$, $p=.131$, $\eta_p^2 = .145$, higher fluid intelligence, $F(1,14) = 2.53$, $p=.13$, $\eta_p^2 = .153$, were more reactive, $F(1,15) = 5.56$, $p<.05$, $\eta_p^2 = .271$, had a greater change in heart-rate, $F(1,15) = 5.94$, $p<.05$, $\eta_p^2 = .284$, had a greater change in skin conductance level $F(1,15) = 3.95$, $p=.07$, $\eta_p^2 = .233$, had a greater change in and heart-rate-variability, $F(1,15) = 2.20$, p-.159, $\eta_p^2 = .128$, and performed better on the speech, overall, $F(1,15) = 5.97$, $p<.05$, $\eta_p^2 = .285$, but showed less openness/agreeableness $F(1,15) = 2.68$, $p=.123$, $\eta_p^2 = .151$. There were no differences between groups in their VMPFC function, or time post injury.

[6] This score reflects a younger age, slightly less severe injury, and greater premorbid intelligence. When each of these variables were compared directly (between mod-sev planners and avoiders), the only significant different between groups was that planners had a greater estimated premorbid IQ. When estimated premorbid IQ was entered as a covariate into analyses comparing other composite measures, the same patterns of results remained.

Table 5: Mean (SD) for planners and avoiders in the moderate-to- severe group. *Indicates significant difference (p<.05)

	Planners	Avoiders
Executive Function	**.514 (3.1)*** **$\eta_p^2 = .403$**	**-.378 (.63)***
VMPFC Function	-.267 (.74) $\eta_p^2 = .001$	-.231 (.78)
Sociodemographic Composite	-.162 (.37) $\eta_p^2 = .145$	-.430 (.31)
Raven Standard Progressive Matrices	19 (7.6) $\eta_p^2 = .153$	12.7 (7.7)
Time Post	150.6 (90) $\eta_p^2 = .001$	159.7 (137)
Reactivity	**.270 (.71)*** **$\eta_p^2 = .271$**	**-.407 (.48)***
Open/Agree	-.471 (.37) $\eta_p^2 = .151$	.193 (.94)
Heart Rate Change	**13.72 (6.9)*** **$\eta_p^2 = .284$**	**6.94 (4.6)***
Heart Rate Variability Change	12.34 (36) $\eta_p^2 = .128$	31.75 (18)
Skin Conductance Level Change	5.67 (2.4) $\eta_p^2 = .233$	3.5 (1.7)
Cortisol Change	-.053 (.31) $\eta_p^2 = .010$	.045 (.56)
Speech Score	**6.5 (1.5)*** $\eta_p^2 = .285$	**4.8 (1.3)***

Summary: Planners had better executive functioning than avoiders, were more psychologically and physically reactive to stress, and performed better, overall, on the speech. These data support prior analyses, and suggest that cognitive resource and being responsive to stress are necessary to be planful after moderate-to-severe TBI.

Moderators of the Behaviour X Coping Group Interaction

The analyses above demonstrated that executive function, reactivity and skin conductance level change were the best predictors of coping following moderate-to-severe TBI. The aim of these analyses was to uncover the covariates that moderate the coping X group interaction displayed in Figure 7. In other words, the goal was to make avoiders look like planners by covarying out the variables that best predicted coping. Repeated measures analysis was performed examining planful and avoidant coping in planner and avoiders, and executive function, reactivity and skin conductance level change were entered as covariates. The behaviour X group interaction was diminished, but still significant, F(1, 10) = 6.89, *p* = .025, η_p^2 = .408. Executive function and reactivity did not interact with coping behaviour. Skin conductance level change did interact with behaviour, F(1, 10) = 1.31, *p* = .279, but η_p^2 = .12.

Overall Summary: There were a number of relations between planful and avoidant coping and variables of interest. Planful coping related to better executive function and to being more reactive psychologically and physiologically; avoidant coping related to poorer executive function, and being less reactive psychologically and physiologically. Stepwise regression revealed that executive function, psychological reactivity and changes in skin-conductance level were the best predictors of coping. It was also revealed the moderate-to-severe group consisted of two distinct groups based on their coping behaviour. Planners engaged in more planful and less avoidant behaviour, and had better executive function, higher sociodemographic status, were more reactive psychologically and physiologically, and performed better on the speech than did the avoiders. Together, these data suggest that planful behaviour was driven by having good cognitive resources and a tendency to be responsive to stress. In the context of the BPST, planful coping was advantageous, and related to better speech performance.

Discussion

The purpose of this study was to examine the covariates of coping following moderate-to-severe TBI. There are five main findings to this study. First, relative to avoidant coping, planful coping related to better executive function, being more reactive both psychologically and physiologically, and performing better on the speech. Second, stepwise regression suggested that psychological reactivity and skin conductance change were the best predictors of planful coping, while executive dysfunction, alone, was the best predictor of avoidant coping. Third, the moderate-to-severe group could be fractionated into two distinct groups based on their coping behaviour. One group engaged in more planful than avoidant behaviour, and are referred to as ‘planners’ (similar to the controls described in Chapter 1), and another group engaged in more avoidant than planful coping, and are referred to ‘avoiders’. Fourth, those classified as ‘planners’ had higher executive function, higher sociodemographic status, were more reactive psychologically and physiologically, and performed better on the speech. Fifth, executive function, reactivity, and skin conductance level influenced the coping X group interaction, but were not significant enough to eliminate the interaction (i.e., make avoiders appear as planners). Each of these findings is discussed in turn below.

In this sample of moderate-to-severe TBI patients, executive function, fluid intelligence, reactivity, physiological responding to stress, and overall better performance on the speech task all related to planful coping. That is, those people who engaged in more planful strategies had better executive functioning, and were more responsive to stress both psychologically and physiologically. Planful behaviour also related to better speech performance, suggesting that a planful approach to the stressful situation was advantageous. These results are consistent with earlier findings demonstrating relationships between self-reported coping and executive function (Krpan et al., 2007). A remaining question was which of these variable(s) contributed to coping behaviour most.

Stepwise regression was employed as an exploratory means of examining which factors best predicted coping. The author acknowledges the limitation of step-wise regression, in that the output is contingent on the variables that are entered into the analyses. Hierarchical regression, however, was not possible given the sample size (i.e., power was lost with the addition of each variable, rendering the output insignificant).

When all the variables included in this study that were significantly related to planful coping (by estimates of effect sizes) were entered into a step-wise regression, psychological reactivity and skin conductance level change were the best predictors of planful coping. These data can be interpreted in two ways. The first interpretation is that to be planful, one must recognize and be affected by the stressor (as indexed by the reactivity composite), and then put forth significant cognitive effort to manage the stress (as indexed by the skin conductance change score). This is supported by evidence that changes is skin conductance response have been related to cognitive effort (e.g., Cohen & Waters, 1985), and to active coping (Sosnowski, Nurzynska, & Polec, 1991). An alternative interpretation is that reactivity and autonomic arousal might actually be an index of *affective function*. Translated literally, *affective* functions are *emotional* functions. The composite score of VMPFC function included neuropsychological measures that are sensitive to the VMPFC, but not necessarily affective in nature (e.g., making an intradimensional shift is not emotional). The author suggests that reactivity (being prone to respond emotionally) and skin conductance change (an autonomic response that has been linked to emotional processing, e.g., Damasio, Tranel & Damasio, 1990; Bechara, Damasio, Tranel & Damasio, 1997) are in fact indexing affective functioning, and are related to planful coping. This idea is discussed in more depth in the General Discussion of this thesis.

When all the variables included in this study that were significantly related to avoidant coping were entered into a stepwise regression, executive function, alone, was the best predictor of avoidant coping following moderate-to-severe TBI. That is, lower executive function predicted avoidant behaviour. This finding is consistent with Krpan et al. (2007) who demonstrated the same pattern of relations using a self-report measure of coping. The author interprets this finding to suggest executive dysfunction limits the cognitive resources necessary to successfully plan and execute a problem-focused coping strategy, resulting in a default reliance on emotion-focused coping, in particular avoidance, when problem-focused strategies would have been more efficient. This hypothesis is also supported by qualitative reports from study participants with TBI. In particular, patient D.G. offered that it is sometimes not worthwhile to allot his limited resources to engage in a problem-focused strategy:

> "I know what is needed and understand [my daughter's] position but find it extremely difficult to listen to [her] ... The end result is I either try and avoid listening to the whole story ... or I leave and let my more capable spouse handle it, so that I don't get angry myself or I don't cut off my daughter prematurely because I can't cope with the length of time it is taking or the amount of detail. **In either case I am using an avoidance strategy in order that a negative outcome is not produced. I totally agree that I may be able to put my limited energies into dealing with this situation more effectively,** but my experience is that there will then be no energy to produce more positive outcomes in subsequent family situations. So I am picking and choosing the times in which I can devout my full energy with positive outcome and also choosing which situations to avoid because the outcome is not likely to be positive or I do not have the energy to make it positive. In my mind, this is a problem-solving approach overall in which there are avoidant strategies inherently a part."

It could be argued that D.G. is *planning* to avoid, and it seems clear that he is employing a strategy to do so. The product, however, is avoidance. The author fully acknowledges that there may be different subsets of avoiders; those who avoid strategically (like D.G.), and those who are unaware of a stressor, and thus simply ignore it. It is not possible to make this dissociation with the current data.

Analysis of variability in behaviour within the moderate-to-severe group demonstrated two distinct groups. One group (n = 6), referred to as planners, engaged in more planful than avoidant behaviour. Another group (n = 11), referred to as avoiders, engaged in more avoidant than planful behaviour. Examination of the differences between these groups revealed that the planners had better executive function, fluid intelligence, and higher sociodemographic status. The planners were also more reactive psychologically and physiologically, and were less open/agreeable than the avoiders. In fact, when compared to planners in the control group described in Chapter Three (n = 17), the moderate-to-severe planners had *better* executive function, $F(1, 20) = 2.21$, $p=.153$, $\eta_p^2=.10$, were more reactive $F(1,21) = 4.69$, $p = .043$, $\eta_p^2=.182$, and experienced more of a change in skin conductance level, $F(1, 18) = 6.70$, $p = .019$, $\eta_p^2=.271$. The control planners, on the other hand, did spend more time engaging in planful behaviour, $F(1,21) = 2.67$, $p=.127$, $\eta_p^2 = .113$, performed better on the speech, $F(1, 21) = 11.29$, $p=.003$, $\eta_p^2 = .350$, were more open/agree, $F(1, 21) = 3.98$, $p = .059$, $\eta_p^2=.159$, and had larger changes in heart rate variability, $F(1, 20) = 4.94$, $p = .038$, $\eta_p^2=.198$, compared to the moderate-to-severe planners. Estimates of effect sizes for these group differences are large. These data are interpreted by the author to mean that following moderate-to-severe TBI, there are a number of factors that are critical to engaging in planful coping. These factors include having good executive function, being psychologically reactive to stress, and putting forth considerable cognitive effort. In the absence of TBI, these factors are less important. Controls were able to engage in planful behaviour despite having lower executive function, being less psychologically reactive, and putting forth less cognitive effort (at least as indexed by skin conductance level). Moreover, the outcome of their planful behaviour was greater than the moderate-to-severe group – they performed better on the speech, overall.

The last analyses described in this chapter was aimed to eliminate the group (planners vs. avoiders) X coping (planful vs. avoidant behaviour) interaction in the moderate-to-severe group by entering the critical variables described above as covariates. It was demonstrated that executive function, reactivity and changes in skin conductance level did moderate some of this interaction, but not enough to yield it insignificant. This is interpreted to mean that although these factors appear to be important moderators of coping following TBI, additional variables that were not accounted for in this study also contribute to coping following moderate-to-severe TBI. Other possible moderator variables might include current drug/alcohol use and social support, cultural background, as well as premorbid levels of functioning, social support, personality etc.

A remaining question concerns the consequences of these types of coping on self-report and SO-reported outcomes. Based on the literature, it would be predicted that the planners have better long term outcomes than the avoiders. The purpose of Chapter Five was to determine the relationship between coping behaviour and outcomes following moderate-to-severe TBI.

CHAPTER FIVE

The Relationship between Coping and Self and Significant Other Reported Outcomes Following Moderate-to-Severe Traumatic Brain Injury

Rationale

In the previous chapters it was demonstrated that the moderate-to-severe TBI group exhibited a unique pattern of coping, such that they engaged in more avoidant than planful behaviours on the BPST. Within this group, there were a number of relations between coping behaviour and variables of interest, and stepwise regression suggested that executive function, reactivity and changes in skin conductance level were the most relevant. Analysis of variability in behaviour revealed a bimodal distribution. Two groups, referred to as 'avoiders' and 'planners' differed on a number of measures. Namely, planners had higher executive function, better fluid intelligence, were more reactive, less open/agree, and had greater heart rate and skin conductance responses to stress. Moreover, these variables did not eliminate the group X behaviour interaction.

A remaining question concerns the consequences of these coping behaviours. It has been reported that engaging in less avoidant coping results in good outcomes (Anson & Ponsford, 2006; Malia et al., 1995; McMillan et al., 2003; Leach, Frank, Bouman, & Farmer, 1995; Tomberge, Toomela, Pulver, & Tikk, 2005), whereas engaging in more avoidant coping is associated with more anxiety, depression and psychosocial distress (Anson & Ponsford, 2006; Curran et al., 2000; Dawson et al., 2006; Finset & Andersson, 2000; Lubosko, et al., 1994). However, these relations have been based upon self-report data using coping questionnaires, rather than directly observing coping behaviour.

There are three objectives of this chapter: First, to assess awareness of outcomes following moderate-to-severe TBI by comparing self and SO reports on the SIP and DEX; second, to investigate the relationships between reported (WOC) and observed (BPST) coping and outcome; and third, to compare outcomes in planners and avoiders.

Methods

The precise methods of this study are described in the General Methods Section of this thesis. Briefly, people with moderate-to-severe TBI completed the Baycrest Psychosocial Stress Test, during which time their planning behaviour was recorded. Both participants and a significant other (SO) also completed the SIP and the DEX as estimates of outcome. The SO reports were included to identify lack of awareness of deficits that have been associated with TBI (e.g., Prigatano, 2005).

Results

Awareness of Outcomes in Planners and Avoiders

For the analyses in this chapter, n = 6 for planner in the TBI group, n = 11 for avoiders in the TBI group. SO data was available for all participants in these analyses. Univariate analyses were first conducted between self-report and SO-reported outcomes within the planful and avoidant groups. These analyses were performed in order to determine if people with TBI were aware of their deficits.

Awareness in Planners: As displayed in Figure 8, within the planful group, there were few differences between self and SO reports of outcome. On the SIP, SO's reported more psychosocial, F(1,10) = 1.13, *p*=.311, η_p^2 = .102, and total problems, F(1,10)= .957, *p*=.351, but η_p^2 = .087, relative to the patients themselves. As depicted in Figure 9, there were no differences on the total DEX score, and differences of medium effect sizes on positive affect, F(1,10) = .947, *p*=.353, η_p^2 = .087, and negative affect, F(1,10) = 1.98, *p*=.189, η_p^2 = .166, where SO's reported more problems.

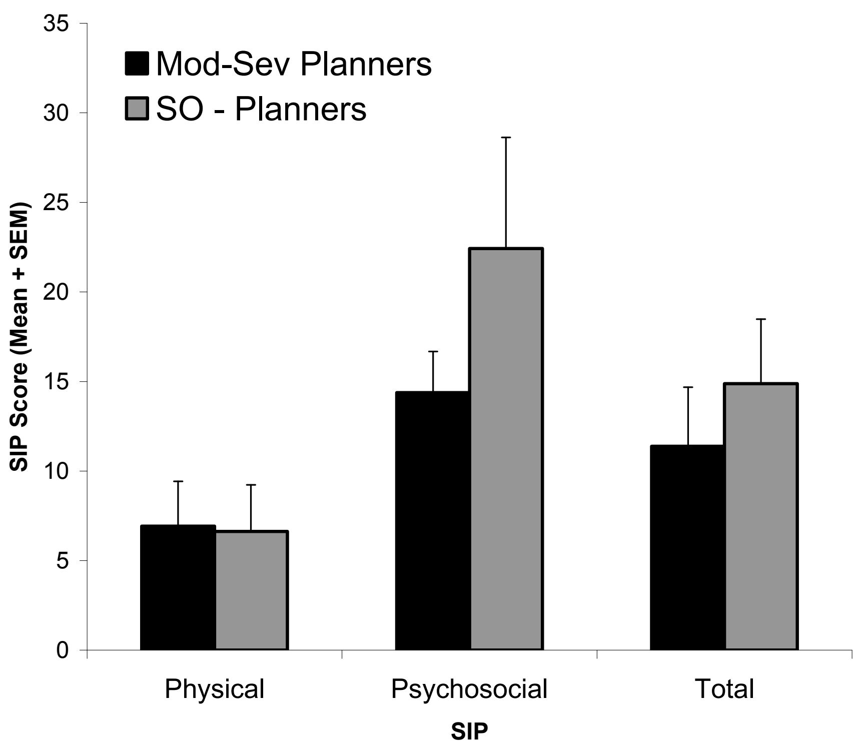

Figure 8: Mean differences in SIP outcomes (+ SEM) between planners and their significant others

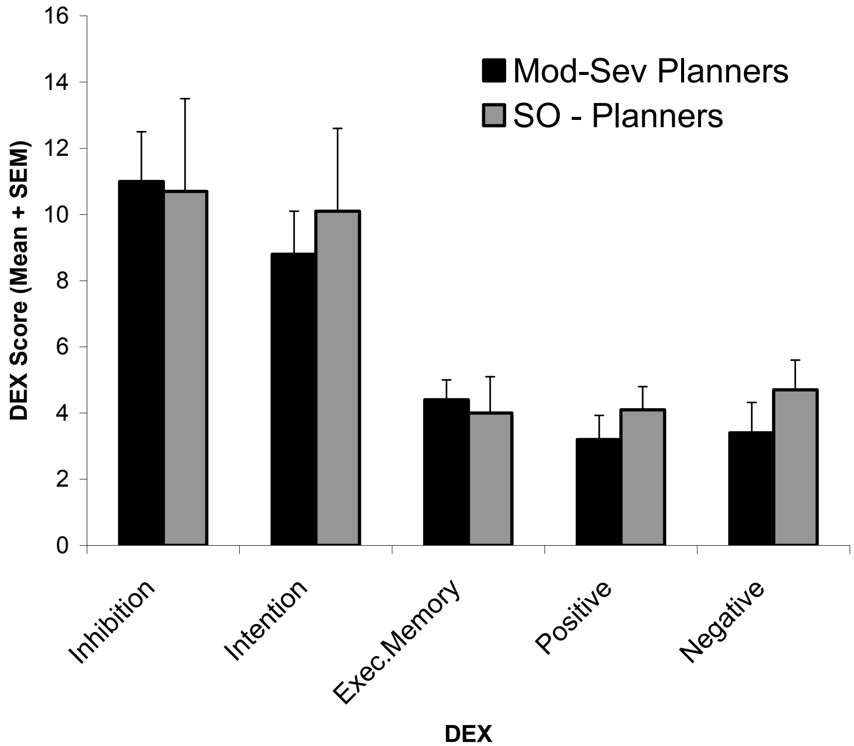

Figure 9: Mean differences in DEX outcomes (+ SEM) between planners and their significant others

Awareness in Avoiders: As depicted in Figure 10, within the avoidant group, there were small differences between self and SO reports on the SIP where SO's reported more psychosocial problems, F(1,19) = .411, *p*=.529, η_p^2 = .021, and total problems F(1,19) = .661, *p*=.426, η_p^2 = .034. As depicted in Figure 11, there were a number of significant differences on the DEX, where SO's reported more problems on all subscales with effect sizes ranging from small to large: Inhibition F(1,20) = 1.33, *p*=.262, η_p^2 = .062; Intention, F(1,20) = 8.27, *p*=.009, η_p^2 = .293; Executive Memory, F(1,20) = 1.46, *p*=.240, η_p^2 = .068;

Positive Affect, F(1,20) = 4.72, *p*=.042, η_p^2 = .191; Negative Affect, F(1,20) = .351, *p*=.56, η_p^2 = .017; and Overall, F(1,20) = 5.37, *p*=..031, η_p^2 = .212.

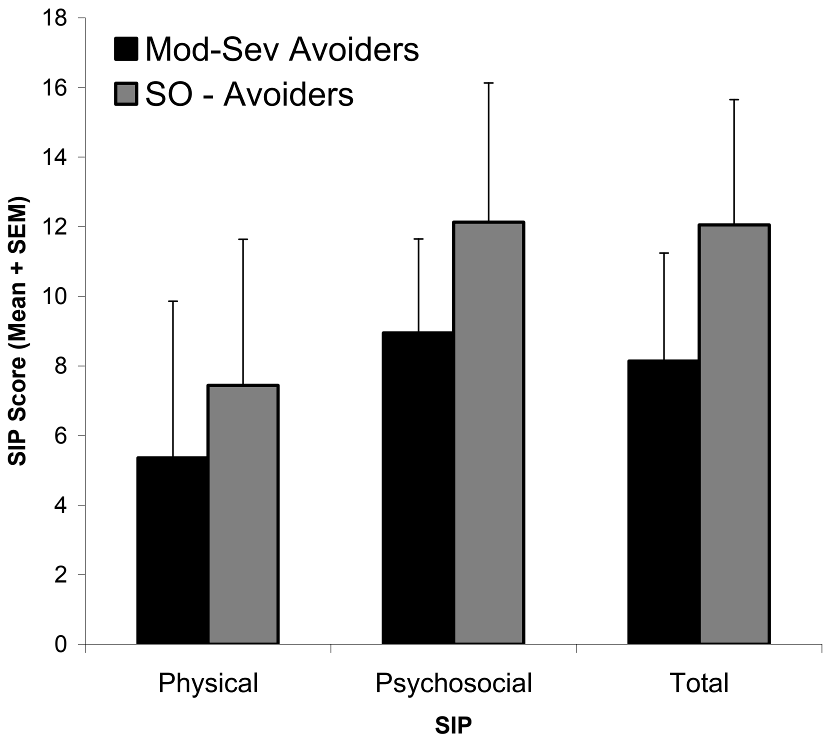

Figure 10: Mean differences in SIP outcomes (+ SEM) between avoiders and their significant others

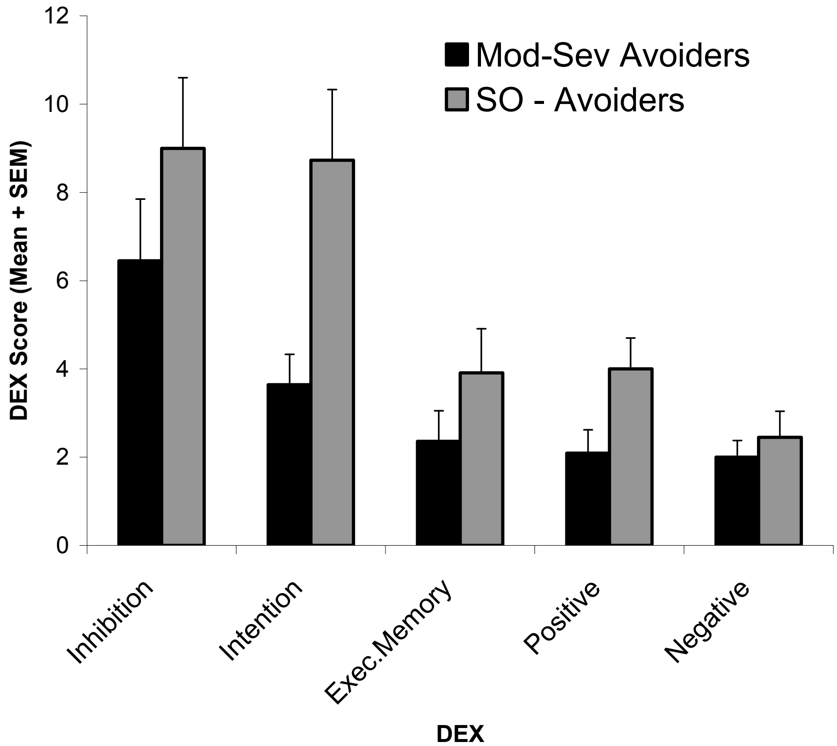

Figure 11: Mean differences in DEX outcomes (+ SEM) between avoiders and their significant others

Summary: People with moderate-to-severe TBI under-reported negative outcomes relative to SO reports, and this appeared to occur more frequently in the avoidant group (as indexed by many discrepancies between self and SO reports on the DEX). A remaining question concerns the relationship between coping and outcomes. Given the problems of awareness described above, correlations were performed for both self and SO reported outcomes.

The Relationship between Self and Significant Other Reported Coping on the WOC and Outcomes

Spearman's correlations were performed between self and SO reported coping on the WOC (planful problem solving and escape avoidant scores) and self and SO reported outcome measures (SIP and DEX).

As depicted in Table 6, there were a number of relations between coping and outcome with effect sizes ranging from small to large. The use of planful problem solving coping related to better SIP outcomes, whereas the use of escape avoidant coping related to worse SIP outcomes. These relations were present in both the self and SO reported data. Based upon self report, the use of planful problem solving coping also related to better outcomes on the DEX, but this was not supported by SO reports.

Table 6: Correlation coefficients (Spearman's *rho*) between self and significant other reported coping, and self and significant other reported outcomes. Bolded items represent medium to large effect sizes ($rho^2 > .06$)

	Planful Problem Solving	SO Planful Problem Solving	Escape Avoidant	SO Escape Avoidant
DEX – Total Score	**-.280** ***p* = .261**	.116 *p* =.657	.233 *p* = .352	.100 *p* =.703
SO DEX – Total Score	.155 *p* =.539	-.063 *p* =.811	-.113 *p =.656*	.157 *p* =546
SIP – Total Score	**-.453** ***p* =.068**	**-.293** ***p =.271***	**.320** ***p =.210***	.162 *p* =.548
SO SIP – Total Score	**-.361** ***p* =.141**	**-.404** ***p* =.108**	.086 *p =.735*	**.247** ***p =.339***

The Relationship between Observed Coping Behaviour on the BPST and Outcomes

Spearman's correlations were performed between coping behaviour (total planful and total avoidant) and outcome measures (SIP and DEX).

As depicted in table 7, there were a number of relations between coping and outcome with effect sizes ranging from small to large. Planful behaviour related to *more* self and SO reported problems on the SIP and DEX (effect sizes ranging from medium to large). Avoidant behaviour related to *less* self-reported problems on the SIP and DEX, but less so to SO-reported problems on the SIP and DEX (effects ranging from none to medium).

Table 7: Correlation coefficients (Spearman's *rho*) between coping behaviour and self and significant other reported outcomes. Bolded items represent medium to large effect sizes ($rho^2 > .06$).

	Planful Behaviour	Avoidant Behaivour
DEX – Total Score	**.537** ***p* = .013**	**-.302** ***p* = .120**
SO DEX – Total Score	**.345** ***p* = .088**	-.071 *p* = .393
SIP – Total Score	**.258** ***p* = .17**	.206 *p* = .222
SO SIP – Total Score	.154 *p* = .15	.119 *p* = .325

Summary: Based upon self and SO reports alone (WOC relating to SIP and DEX), planful coping is related to good outcomes, and avoidant coping to negative outcomes. This finding is consistent with other studies showing that subjective reports of using avoidant coping are related to negative outcomes (Anson & Ponsford, 2006; Curran et al., 2000; Dawson et al., 2006; Finset & Anderson, 2000; Lubosko, et al., 1994; Malia et al., 1995; McMillan et al., 2003; Leach et al., 1995; Tomberge et al., 2005; Lubosko, et al., 1994). In contrast, the behavioural data on coping showed the opposite patterns of relations to outcomes. More planful coping on the BPST related to negative outcomes on the SIP and DEX, and more avoidant coping related to better outcomes (although this relationship was weaker). A remaining question is what drives these unexpected relations between coping behaviour and outcomes. In order to help address this question, group differences in outcomes between planners and avoiders were assessed.

Outcomes in Planners and Avoiders

Univariate analyses were performed to assess the differences between planners and avoiders on self and SO-reported SIP and DEX outcomes.

Self-Reported Differences in Outcomes: As depicted in Figures 12 and 13, according to self report, planners reported having more psychosocial problems on the SIP, $F(1,14) = 4.69$, $p=.312$, $\eta_p^2 = .074$, and more problems on all subscales of the DEX; Inhibition, $F(1, 15) = 2.98$, $p=.105$, $\eta_p^2 = .166$; Intention, $F(1, 15) = 11.79$, $p=.004$, $\eta_p^2 = .440$; Executive Memory, $F(1, 15) = 2.42$, $p=.141$, $\eta_p^2 = .139$; Positive Affect, $F(1, 15) = 1.61$, $p=.223$, $\eta_p^2 = .097$; Negative Affect, $F(1, 15) = 1.77$, $p=.203$, $\eta_p^2 = .105$; Total Score, $F(1, 15) = 6.434$, $p=.023$, $\eta_p^2 = .300$, than did the avoiders.

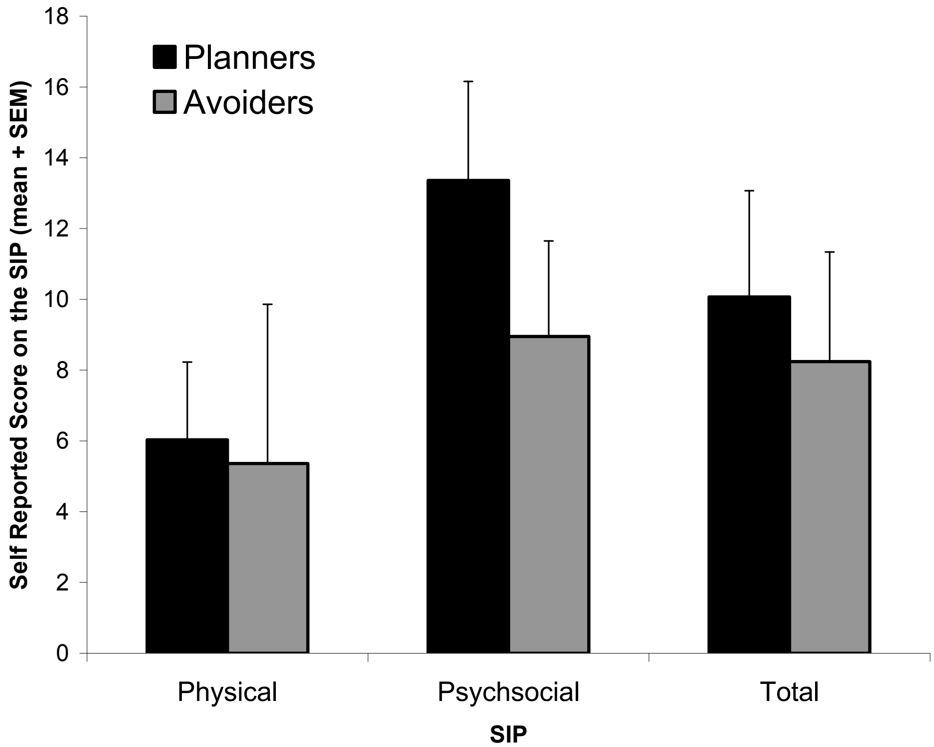

Figure 12: Mean differences in self reported SIP outcomes (+ SEM) between Planners and Avoiders

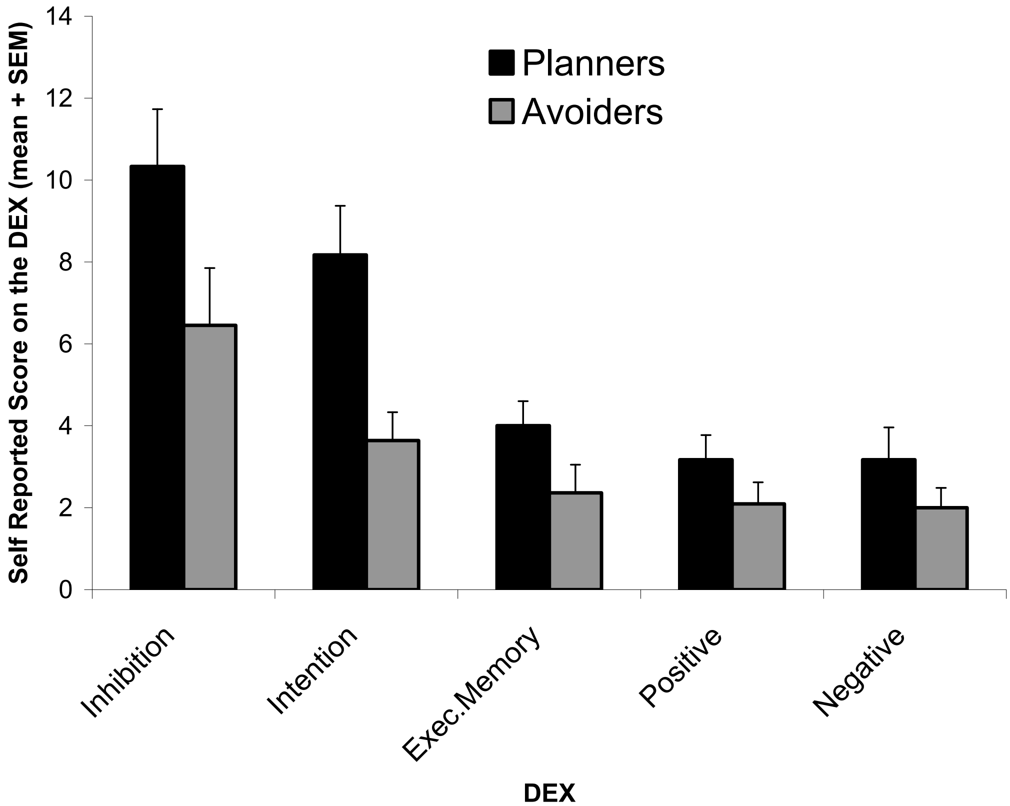

Figure 13: Mean differences in self reported SIP outcomes (+ SEM) between Planners and Avoiders

SO Reported Differences in Outcomes: Analyses were then conducted using SO reports of outcomes. As depicted in Figure 14, SO reports confirmed that planners experienced more psychosocial problems than did avoiders, F(1, 14) = 1.274, *p*=.277, η_p^2 = .078; and also more total problems on the SIP, F(1, 15) = 2.99, *p*=.592, η_p^2 = .02. As depicted in Figure15, there were few differences between planners and avoiders by SO report on the DEX, with the exceptions of Total score, F(1, 15) = .584, *p*=.456, η_p^2 = .038, and especially negative affect, F(1, 15) = 4.69, *p*=.047, η_p^2 = .238.

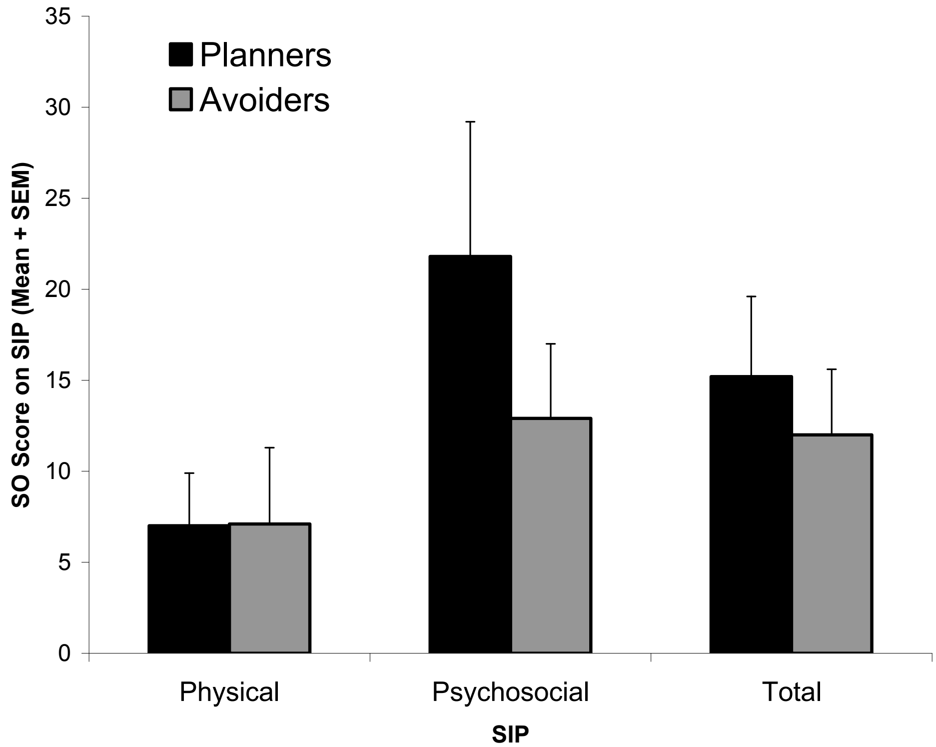

Figure 14: Mean differences in SO reported SIP outcomes (+ SEM) between Planners and Avoiders

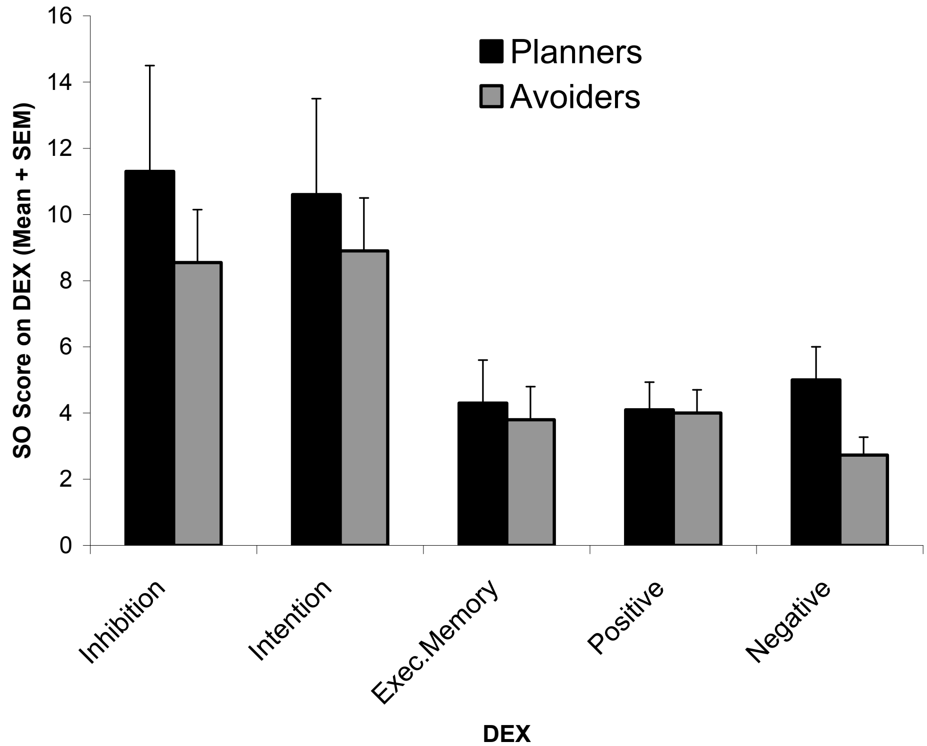

Figure 15: Mean differences in SO-reported DEX outcomes (+ SEM) between planners and avoiders

Outcomes as a Function of Return to Productivity

The last set of data presented in this thesis (see below) was not originally intended to be collected. Analyses of the SIP and DEX outcomes in relation to coping on the BPST yielded results that were not consistent with the literature (Anson & Ponsford, 2006; Curran et al., 2000; Dawson et al., 2006; Finset & Andersson, 2000; Lubosko, et al., 1994; Malia et al., 1995; McMillan et al., 2003; Leach et al., 1995; Tomberge et al., 2005). To better understand those relations, the author further collected data about Return to Productivity (RTP) in the moderate to severe group. Statistical analyses were not possible given the small sample size, but Table 8 depicts the frequency of RTP among planners and avoiders. Of interest, while none of the planner had returned to gainful employment at the time of study, 50% of the group was engaged in long term and regular volunteer activities. Notably, almost 55% of the avoidant group showed no evidence of RTP at the time of the study.

Table 8: Frequency of Return to Productivity among planners and avoiders.

	Planners	**Avoiders**
Work – Full Time	0	0
Work– Part Time	0	2 (18.2%)
Volunteer	3 (50%)	0
Student	1 (16.7%)	2 (18.2%)
Retired	1 (16.7%)	1 (9.1%)
Not Working	1 (16.7%)	6 (54.5%)

Overall Summary: People with moderate-to-severe TBI under-reported negative outcomes relative to their SO's, and this was particularly evident in the avoidant group. As a result, the remaining analyses were conducted using both self and SO reported outcome data. Correlational analyses revealed that coping was related to outcomes in different ways depended upon how it was measured. Self and SO reported coping on the WOC related to outcomes in the predicted direction (planful related to good outcomes, and avoidant related to negative outcomes). The behavioural data, however, demonstrated the opposite pattern of relations. Contrary to previous findings, planful coping on the BPST related to *negative* outcomes, and avoidant behaviour related to *better* outcomes. Moreover, analysis of group differences in outcomes revealed that avoiders had better outcomes than did planners. However, planners showed greater evidence of RTP than did avoiders.

Discussion

This was the first study to examine the relationship between observed coping behaviour and self and significant other reported outcomes following moderate-to-severe TBI. There are five main findings to this study. First, people with moderate-to-severe TBI under-report negative outcomes, and this appeared to be more prominent in the avoidant group. Second, based upon self and SO reports, planful coping related to good outcomes, and avoidant coping to negative outcomes. Third, based upon observed coping behaviour, planful coping related to *negative* outcomes, and avoidant coping behaviour related to *better* outcomes. Fourth, planners had worse outcomes than avoiders. Fifth, planners showed evidence of greater RTP than did avoiders. Each of these findings will be discussed in turn below.

Consistent with previous work, people with moderate-to-severe injury under-reported negative outcomes relative to their significant others (e.g., Prigatano, 2005; Stuss, 1991). These differences were most pronounced in the avoidant group, where SO's reported more cognitive problems on the DEX (effects ranging from small to large across subscales). The planners also under-reported psychosocial and total problems on the SIP, as well as problems with affect on the DEX, but the effect sizes were generally smaller than those found in the avoidant group. In order to control for these problems of awareness, the remaining analyses were performed using both self-reported and SO-reported outcomes data.

Correlational analyses revealed self and SO reported coping on the WOC related to outcomes on the SIP and DEX. Self and SO reports of planful problem solving coping on the WOC related to good outcomes on the SIP, and self and SO reports of escape avoidant coping on the WOC related to negative outcomes on the SIP. These findings are consistent with earlier work showing similar relations between coping and outcome (Anson & Ponsford, 2006; Curran et al., 2000; Dawson et al., 2006; Finset & Andersson, 2000; Lubosko, et al., 1994; Malia et al., 1995; McMillan et al., 2003; Leach et al., 1995; Tomberge et al., 2005).

Correlational analyses also revealed that, contrary to previous research using self-report measures, planful behaviour on the BPST was related to negative outcomes. This pattern of relations was consistent across self and SO-reported outcome data. Moreover, avoidant behaviour related to positive outcomes by self-report, although these relations were not entirely consistent in the SO data. Analysis of group differences between planners and avoiders revealed that planners had worse outcomes, and avoiders had better outcomes. These results do not support earlier work showing that people with TBI who engage in less avoidant coping have better psychosocial outcome (Anson & Ponsford, 2006; Malia et al., 1995; McMillan et al., 2003; Leach et al., 1995; Tomberge et al., 2005), whereas those engaging in more avoidant coping experience more anxiety, depression and increased psychosocial distress (Anson & Ponsford, 2006; Curran et al., 2000; Dawson et al., 2006; Finset & Andersson, 2000; Lubosko, et al., 1994).

One explanation for this discrepancy is that previous studies have always depended on self-report measures of coping. It was demonstrated in Chapter Three of this thesis that self-report measures on coping on the WOC did not relate to actual coping behaviour observed during the BPST. These findings suggest that the relations between coping and outcome that are described in the literature are skewed by self report, and do not accurately reflect the relationships between how people actually behave in the real world, and their outcomes. This is a critical finding when considering application to rehabilitation. These behavioural data suggest that it may be advantageous, at least in the longer term (insofar as the SIP and DEX can index longer term outcomes), to advocate avoidant, rather than planful, coping strategies after TBI. These data, however, must be interpreted with caution.

It is not clear how avoidance might moderate good SIP and DEX outcomes. It has been suggested in the literature that a moderate amount of denial is an adaptive response to TBI (Moore & Stambrook, 1994; Moore, Stambrook & Peters, 1989) because individuals do not have the cognitive capacity to employ effective planful coping strategies. In this case, it may be that a lack of awareness or denial of deficits is protective against the negative consequences of TBI. This notion is supported by the data presented in Chapters Three through Five of this thesis. It appears that those people with moderate-to-severe TBI who engage in planful strategies are more aware of their deficits, and struggle to engage in planful strategies, at the cost of negative outcomes. This hypothesis is discussed in more depth in the General Discussion that follows.

The finding that planners seemingly have better RTP than avoiders is more consistent with the literature (Anson & Ponsford, 2006; Curran et al., 2000; Dawson et al., 2006; Finset & Andersson, 2000; Lubosko, et al., 1994; Malia et al., 1995; McMillan et al., 2003; Leach et al., 1995; Tomberge et al., 2005). These data may also help to explain why planners report more negative outcomes on the SIP and DEX; planners are more engaged in the community, and as such, are likely exposed to the stressors of the real-world. Their avoidant counterparts, however, are less engaged in the community, instead spending large amounts of time in the home environment, with familiar people. Considering these data, it is not so surprising that the group that is attempting to re-integrate into society following TBI (i.e., the planners) also reports experiencing more psychosocial and dysexecutive distress. This idea is considered in the context of the neuropsychological, personality, psychiatric and physiological data in the General Discussion that follows.

CHAPTER SIX

GENERAL DISCUSSION

This was the first study, to the author's knowledge, to assess coping behaviour in people with TBI during a simulated real-world stressful situation. The series of analyses described in Chapters Three through Five of this thesis yield an important story about coping following moderate-to-severe traumatic brain injury, and may well have important implications for rehabilitation. The main findings of this thesis are discussed in relation to each of the four main hypotheses that were described in the General Introduction. The final section of this thesis discusses the potential application of this work to rehabilitation.

The first hypothesis of this thesis was that people with TBI would engage in more avoidant coping strategies on behavioural measures (BPST), but under-report the use of such strategies on self-report measures (WOC). This hypothesis was supported by the data. As a group, people with moderate-to-severe TBI coped differently when under stress than did either people with mild TBI, or controls. Rather than engaging in more planful than avoidant behaviour, they engaged in more avoidant than planful behaviour. In contrast, and consistent with the literature, there were no significant differences between the control and moderate-to-severe group in their self or significant other reported coping style on the WOC (Curran et al., 2000; Dawson et al., 2006; Krpan et al., 2007). Even more striking, there were no relationships between coping behaviours on the BPST and self-reported coping style. This finding suggests that there is a discrepancy between how people with TBI think they cope, and how they actually cope in the real world, and may reflect known problems with self-awareness following TBI (e.g., Prigatano, 2005; Stuss, 1991).

The second hypothesis of this thesis was that within the TBI group, executive and affective function would be positively related to planful coping, and negatively related to avoidant coping. This hypothesis was partly supported by the data. Consistent with the literature (e.g., Krpan et al., 2007) within the moderate-to-severe group, people who had higher executive function engaged in more planful behaviour and less avoidant behaviour on the BPST. Moreover, the effect sizes of these relations were large. These data are interpreted by the author to suggest that executive dysfunction limits the cognitive resources necessary to successfully plan and execute a problem-focused coping strategy. The consequence of this cognitive dysfunction might be a default reliance on emotion-focused coping, in particular avoidance, when problem-focused strategies would have been more efficient.

The story regarding the relationship between affective function and coping is less clear. The composite measure of VMPFC function (which included object alternation errors, smell identification, and intradimensional shift reversal errors) did not relate to coping. Despite these null findings, the author is hesitant to disregard the hypothesis that affective function does relate to coping post TBI. It is posited that affective function does influence avoidant coping, but that the neuropsychological tools used to assess affective function were not sensitive or specific enough to demonstrate these relations. Within the neuropsychological world, there remains the persistent problem of sufficiently assessing VMPFC integrity. The object alternation task and gambling task, for example, are sensitive to TBI (e.g., Fujiwara et al., 2008), but not necessarily specific to VMPFC damage. As the neuropsychological tools to assess the VMPFC become more advanced, it is suspected so too will the relationship between affective function and coping.

An alternative way of conceptualizing affective functioning might be to assess a person's emotional and physical response to stress. Taken literally, *affective* responses are *emotional* responses. While the neuropsychological tests described above are sensitive (if not specific) to VMPFC damage, they are not *affective* in nature (e.g., emotional processing is not necessary to make an intradimensional shift). It might be that actually measuring an individual's emotional responses to stress is a better index of affective function. Fortunately, the psychological reactivity composite score, described in the methods section of this thesis, lends itself to this hypothesis. In a similar vein, physiological reactivity may also be a good index of affective function. Autonomic arousal has been related to performance on more emotional neuropsychological tests, for example the Iowa Gambling Task (e.g., Damasio, Tranel & Damasio, 1990; Bechara, Damasio, Tranel & Damasio, 1997; also see Ziino & Ponsford, 2006). There is also evidence that patients with right hemisphere frontal lobe damage, particularly involving right ventral and medial PFC, show attenuated skin conductance response and heart rate to psychologically relevant or emotional stimuli (Andersson & Finset, 1998; Angrilli et al., 1999; O'Keeffe, Dockree, & Robertson, 2004; Sanchez-Navarro et al., 2005; Zahn, Grafman & Tranel, 1999).

The physiological change scores collected during the BPST made it possible to examine this possibility, and is discussed in relation to the third hypothesis of this thesis, below.

The third hypothesis of this thesis was that other factors such as psychiatric status, personality and physiological responsivity to stress would moderate the relationship between neuropsychological function and coping. The data provide support this hypothesis. In addition to executive function, fluid intelligence, psychological reactivity, and physiological reactivity all related positively to planful coping, and negatively to avoidant coping in the moderate-to-severe group. Moreover, stepwise regression models suggested that executive function, alone, was the best predictor of avoidant coping (consistent with Krpan et al., 2007), and reactivity, together with skin conductance change, were the best predictors of planful coping. If affective functions are conceptualized as emotional reactions to stress, these findings are more consistent with the initial hypotheses of this thesis. Affective function indexed by the reactivity and skin conductance level change best predicted planful coping, and executive function best predicted avoidant coping.

The author argues affective function (as indexed by higher reactivity and skin conductance level change scores) moderates planful coping by providing the emotional processing and social awareness necessary to employ a planful coping strategy. The ability to accurately decode social-emotional information (externally from the environment, or internally within the self) results in the ability to detect that a situation requires coping and the application of an efficient strategy because emotion is guiding behaviour (see Gordon, Cantor, Ashman & Brown, 2006 for discussion of cognitive-emotional interactions post-TBI; also, Bechara et al. 1994; Levine et al., 2005).

The author also argues that executive function mediates avoidant coping by limiting the cognitive resources necessary to successfully plan and execute a problem-focused coping strategy, resulting in a default reliance on avoidant strategies when problem-focused strategies would have been more efficient. This hypothesis is supported by earlier work demonstrating that decreased executive function relates to increased use of avoidant coping (Krpan et al., 2007).

In order to better understand the relations between executive and affective function and coping, the author adopted the approach of deconstructing the moderate-to-severe group (i.e., the group that displayed altered coping; see Stuss & Binns, 2008 regarding the importance of removing group variability when conducting clinical research). This approach was fruitful and demonstrated heterogeneity in coping behaviour on the BPST following moderate-to-severe TBI. Two groups of people were identified based on their pattern of coping behaviour. Planners, similar to controls, engaged in more planful than avoidant behaviour. Avoiders engaged in more avoidant than planful behaviour. These two groups differed on a number of variables that related to coping. First, the planners had significantly better executive functioning than did the avoiders, and the effect size of this difference was substantial. Planners also had greater fluid intelligence, higher sociodemographic status, and were more psychologically and physiologically reactive. These data were interpreted to mean that in order to engage in planful behaviour, a person with moderate-to-severe TBI must be reactive to stress (i.e., have a disposition to attend to and react to stress) and also put forth considerable effort (as indexed by skin conductance level change; see Cohen & Waters, 1985; Sosnowski, Nurzynska, & Polec, 1991). In the absence of these factors, reduced executive dysfunction appears to predict avoidant coping, which in turn was related to poor speech performance. Based on these analyses, however, it was not possible to determine if the long term effects of planful coping were greater than avoidant coping, which leads to the fourth hypothesis of this thesis.

The fourth hypothesis of the thesis was that planful coping would relate to positive outcomes, and avoidant coping would relate to negative outcomes following TBI. This hypothesis has already gained support from the literature (e.g., Anson & Ponsford, 2006; Malia et al., 1995; McMillan et al., 2003; Kendall et al., 2001; Leach et al., 1995; Tomberge et al., 2005), and was also supported based upon the self-reported WOC data collected in the present study (see Chapter Five). Moreover, the group differences between planners and avoiders described above (e.g., greater executive function, greater responsivity to stress) provided additional reason to expect that planners would have better long term outcomes than avoiders. It is logical to reason that better executive functioning and a predisposition for detecting stress would provide an individual with the tools necessary to strategically select the most appropriate coping strategy. Indeed, in the short term, this hypothesis was supported. In the moderate-to-severe group, planful behaviour related to better speech performance during the

BPST. Analysis of group differences also demonstrated that planners performed better than avoiders on the speech task. This suggested that the planful behaviour, at least in contrast to the avoidant behaviour, had paid off.

Contrary to the literature and intuition, however, planful behaviour on the BPST did not relate to positive long term outcomes as indexed by the SIP and DEX. In fact, planful behaviour related to *negative* outcomes on the SIP and DEX. Analyses of group differences in outcomes supported the correlational findings and revealed that planners had worse outcomes. Together, these data suggest that although the planners were able to employ a successful strategy of coping in the short term (i.e., those who planned did better on the speech task), it was at a longer term cost of negative self and SO reported outcomes, in general.

This last finding is inconsistent with the literature. Several studies have demonstrated that self-reported avoidant coping relates to negative outcomes (Anson & Ponsford, 2006; Malia et al., 1995; McMillan et al., 2003; Leach et al., 1995; Tomberge et al., 2005). A critical difference between the methodology employed by those studies and the one employed here is that the majority of the relationships presented in this thesis are based on behavioural observation during a simulated real-world stress test, rather than self report. It is well established that there are deficits of self-awareness in TBI that could confound self-report findings (e.g., Prigatano, 2005; Stuss, 1991) not to mention the problem of demand characteristics. Perhaps even more importantly, questionnaires are completed by participants who are no longer in the acute phases of stress, and are required to reflect back upon a situation. This requires a participant to have recently experienced stress and to have recognized it as such, and to recall accurately the situation and how they behaved. Problems with awareness and memory following TBI might have consequences for how participants complete this questionnaire. These problems are not relevant during the BPST, where actual behaviour is recorded as it happens. For these reasons, the author argues that the BPST is likely more reflective of real-world coping behaviours than the WOC. The finding that there were no positive relations between self-reported coping and behaviour supports this hypothesis. It is conceivable, however, that there are no relations because the WOC and BPST are measuring different types of coping, and or because the type of stress experienced during the BPST does not generalize to real-world stressful events. To address the latter concern, future research may employ the BPST, and then require participants to complete the WOC about how they coped during the task.

The reasons why planful coping might be advantageous in the short- term (speech performance), but a disadvantageous in the longer-term (SIP and DEX) are speculative. It is possible that the moderate-to-severe planners were more aware of their shortcomings, and exerted considerable psychophysiological effort in order to achieve the outcomes that they did (see Ziino & Ponsford, 2006). This notion is supported by data showing large differences in skin conductance change between planners and avoiders in the moderate-to-severe group, and even between control and moderate-to-severe planners. Skin conductance response has been argued to reflect cognitive effort (Cohen & Waters, 1985), and also active coping (Sosnowski et al., 1991). It is also supported by the finding that there were fewer differences in self SO reports of outcome in planners, as compared to avoiders. It is feasible then, that over time, this effort has an effect on general well-being. It is also reasonable to argue that a greater self-awareness among planners (as indexed by SO reports) has contributed to negative outcomes (but see Anson & Ponsford 2006c). The long term effect of realizing the consequences of moderate-to-severe TBI may be reduced psychosocial and psychological well-being, even in the absence of severe neuropsychological impairment. Indeed, the notion that denial may be a positive coping strategy following TBI has been discussed in the literature. Moore, Stambrook and Peters (1989) suggested that denial serves as a ‘reality buffer’ that protects an individual from the negative consequences of TBI. They have also suggested, however, that there is an optimal level of denial, where too much or too little maybe be associated with negative outcomes (Moore & Stambrook, 1994). However, it is unclear what level of awareness would be considered optimal; for example, Anson and Ponsford (2006c) reported greater benefits of Cognitive Behavioural Therapy among people with greater awareness following TBI.

An alternative explanation for the unusual relations between coping behaviour and SIP and DEX outcomes is that planners experienced more psychosocial and dysexecutive problems because they were exposing themselves to more challenging and stressful situations than were the avoiders. This hypothesis is supported by the RTP data showing that planners more frequently re-integrated into the community (in this sample, largely by volunteering in the community), while the majority of avoiders displayed no evidence of RTP.

Summary and Implications for Rehabilitation: Given the data presented in this thesis, the author puts forth a tentative model of coping following moderate-to-severe TBI. The data suggest that there are three critical variables that are related to coping in this group: executive function, reactivity, and SCL change. It is argued, that in order to be planful, a person with moderate-to-severe TBI must first have a basic level of executive function. While executive function did not predict planful coping in the regression model described in Chapter 4, planners had significantly better executive function than avoiders (see Table 5), and the effect sizes were large. Once some threshold of executive function is present, it is reactivity together with SCL change that best predict planful coping. This suggests that being emotionally sensitive, having a tendency to experience negative emotions, being self disciplined and able to regulate impulses, in addition to being physiologically reactive and putting forth considerable cognitive effort are all necessary to be planful after moderate-to-severe TBI (together, interpreted as having good affective function). The resulting planful behaviour is advantageous in the short term (speech performance), and in the long term (RTP), but comes at a cost of psychosocial distress (SIP). The author interprets these data to suggest that planners are susceptible to stress, and that they are attempting to re-integrate with the community and putting forth significant cognitive effort to do so. By exposing themselves more to the real-world, they are also exposing themselves to more potential stressors, at the cost of psychosocial well-being.

The factor that best predicts avoidance coping, by contrast, is executive dysfunction. While the avoidant group also showed dampened psychological and physiological reactivity relative to the planners, those variables did not predict avoidant coping on the BPST. Avoidant coping is disadvantageous in the short term (speech), and long term (RTP), but also associated with better psychosocial wellbeing (SIP). The author argues that this group does not have the cognitive resources to execute planful strategies, are less susceptible to experiencing stress, and are also not exposing themselves to stress. Since this group has not re-engaged with the community and is not putting forth considerable effort to adapt to a stressful environment, their psychosocial distress is lower than their planful counterparts.

An important question that cannot yet be answered concerns the implications of these findings for rehabilitation. The ultimate goal of these studies was to better understand the mechanisms of avoidant coping following TBI so that rehabilitative intervention could be more specific. The findings presented in this thesis raise some important questions. Should we be targeting coping through rehabilitation? If so, should we be attempting to engage ‘avoiders’ in planful coping behaviour to improve short term outcomes and re-integrate them into the community? How might we then protect them form the psychosocial cost of being planful? Alternatively, should we targeting the ‘planners’ through rehabilitation? Rehabilitative interventions aimed to improve self efficacy and stress management might allow the planners to achieve the rewards of planful behaviour, but without psychosocial consequences. Planful coping has been successfully targeted through Cognitive Behavioural Therapy following TBI (Anson & Ponsford, 2006b). Unfortunately, however, increases in self-reported planful coping were not accompanied by changes in anxiety, depression, self-esteem or psychosocial outcome in that study (Anson & Ponsford, 2006b). Moreover, people with low self-awareness actually became *more* depressed after interventions designed to encourage planful coping (Anson & Ponsford, 2006c). This highlights the importance of identifying subgroups of people with TBI for rehabilitative purposes. The next step in this body of research is to study in vivo, coping behaviour in more individuals with TBI, to consider alternate measures of assessing affective functions, and to evaluate more closely real-world long term outcomes (e.g., more data on return to productivity, quality of life etc.) as they relate to coping behaviour. It is likely that this line of research will further reveal a distinct subset of people with TBI, and that rehabilitative intervention can then be tailored accordingly (see Anson & Ponsford, 2006c).

Study Limitations

It should be acknowledged, that although the data presented in this thesis generate novel and interesting questions, they should be interpreted with caution. The author is encouraged by the estimates of effect sizes, but it should be recognized that sample size reported here is modest, particularly when further fractionating a heterogeneous group by coping behaviour, and when conducting multiple analyses. The participants were recruited through the community (i.e., they were self selected and highly motivated), and there was a gender balance towards females in the control and mild TBI groups. As such, the sample described here may not represent the TBI population at large. The BPST itself is an experimental task, and the psychometric properties have not yet been established, and these data do not provide information on how coping on the BPST might relate to perceived quality of life. Moreover, individual differences with respect to life experience giving speeches, or prior knowledge about crime in Toronto, may well have influenced coping behaviour, and speech performance on the BPST. Finally, beyond estimates of premorbid IQ, this study did not evaluate how pre-injury factors (e.g., premorbid coping, personality, social support, culture, etc.) may have influenced coping. These limitations should be addressed in future research.

REFERENCES

Adair, J. C., Williamson, D.J., Schwartz, R.L. & Heilman, K.M. (1996). Ventral tegmental area injury and frontal lobe disorder. *Neurology, 46*, 842-843.

Allerdings, M. D., & Alfano, D.P. (2006). Neuropsychological correlates of impaired emotion recognition following traumatic brain injury. *Brain and Cognition, 60*(2), 193-194.

Andelic, N., Hammergren, N., Bautz-Holter, E., Sveen, U., Brunborg, C & Roe, C. (2009). Functional outcome and health-related quality of life 10 years after moderate-to-severe traumatic brain injury. *Acta Neurologica Scandanavia, 120*(1), 16-23.

Anderson, S. W., & Tranel, D. (2002). Neuropsychological consequences of dysfunction in human dorsolateral prefrontal cortex. In J. Grafman (Ed.), *Handbook of Neuropsychology* (2 ed., Vol. 7, pp. 145-156). Amsterdam: Elsevier.

Andersson, S., & Finset, A. (1998). Heart rate and skin conductance reactivity to brief psychological stress in brain-injured patients. *Journal of Psychosomatic Research, 44*(6), 645-656.

Angrilli, A., Palomba, D., Cantagallo, A., Maietti, A., & Stegagno, L. (1999). Emotional impairment after right orbit frontal lesion in patient without cognitive deficits. *Neuroreport, 10*(8), 1741-1746.

Anson, K., & Ponsford, J. (2006). Coping and emotional adjustment following traumatic brain injury. *Journal of Head Trauma Rehabilitation, 21*(3), 248-259.

Anson, K., & Ponsford, J. (2006b). Evaluation of coping skills group following traumatic brain injury. *Brain Injury, 20*(2), 167-178.

Anson, K., & Ponsford, J. (2006c). Who benefits? Outcome following a coping skills group intervention for traumatically brain injured individuals. *Brain Injury, 20*(1), 1-13.

Antonak, R. E., Livneh, H., & Antonak, C. (1993). A review of research on psychosocial adjustment to impairment in persons with traumatic brain injury. *Journal of Head Trauma Rehabilitation, 8*(4), 87-100.

Army Individual Test Battery (1944). Manual of directions and scoring, Washington, DC: War Department, Adjutant General's Office.

Ashman, T. A., Gordon, W.A., Cantor, J.B., & Hibbard, M.R. (2006). Neurobehavioural consequences of traumatic brain injury. *Mount Sinai Journal of Medicine, 73*(7), 999-1005.

Baddeley, A. (1986). *Working Memory*. Oxford: Clarendon.

Barger, S. D., Kircher, J., & Croyle, R.T. (1997). The effects of social context and defensiveness on the physiological responses of repressive copers. *Journal of Personality and Social Psychology, 73*(5), 1118-1128.

Bechara, A., Damasio, A.R., Damasio, H., & Anderson, S.W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition, 50*, 7-15.

Bechara, A., Damasio, H, Tranel, D., & Damasio, A. (1997). Deciding advantageously before knowing the advantageous strategy. *Science, 275*, 1293-1295.

Beck, A. T., Steer, R., & Brown, G.,. (1996). *Beck Depression Inventory-II*. San Antonio: Psychological Corporation.

Bendig, A. W. (1956). The development of a short form of the Manifest Anxiety Scale. *Educational & Psychological Measurement, 16*, 516-523.

Bernstein, D. A. (1999). Recovery from mild head injury. *Brain Injury, 13*, 151-172.

Bornhofen, C., & McDonald, S. (2008). Emotion perception deficits following traumatic brain injury: a review of the evidence and rationale for intervention. *Journal of the International Neuropsychological Society, 14*(4), 511-525.

Brown, J. (1958). Some tests of the decay of immediate memory. *Quarterly Journal of Experimental Psychology, 10*, 12-21.

Brown, L. T., Tomarken, A.J., Orth, D.N., Loosen, P.T., Kalin, N.H., & Davidson, R.J. (1996). Individual differences in repressive-defensiveness predict basal salivary cortisol levels. *Journal of Personality & Social Psychology, 70*, 362-371.

Bryant, R. A., Creamer, M., O'Donnell, M., Silove, D., Clark, C.R., & McFarlane, A.C. (2009). Post-traumatic amnesia and the nature of post-traumatic stress disorder after mild traumatic brain inury. *Journal of the International Neuropsychological Society, E-Pub Ahead of Print August 25*.

Busch, C. R., & Alpern, H.P. (1998). Depression after mild traumatic brain injury: a review of current research. *Neuropsychology Review, 8*(2), 95-108.

Burgess, P. W., Alderman, N., Evans, J., Emslie, H. & Wilson, B.A. (1998). The ecological validity of tests of executive function. *Journal of the International Neuropsychological Society, 4*, 547-558.

Burgess, P. W., & Robertson, I.H. (2002). Principles of the Rehabilitation of Frontal Lobe Function. In D. T. S. a. R. T. Knight (Ed.), *Principles of Frontal Lobe Function*. Oxford: Oxford University Press.

Callahan, C. D., & Hinkebein, J. (1999). Neuropsychological significance of anosmia following traumatic brain injury. *Journal of Head Trauma Rehabilitation, 146*(6), 581-587.

Canadian Institute for Health Information, Analysis in Brief entitled *Head Injuries in Canada: A decade of Change (1994-1995 to 2003 -2004),* August 2006.

Cantor, J. B., Ashman, T.A., Schwartz, M.E., Gordon, W.A., Hibbard, M.R., Brown, M., Spielman, L., Charatz, H.J., & Cheng, Z. (2005). The role of self-discrepancy theory in understanding post-traumatic brain injury affective disorders: A pilot study. *Journal of Head Trauma Rehabilitation, 20*(6), 527-543.

Center for Disease Control and Prevention. (2004). Heads up: Factors for physicians about mild traumatic brain injury (MTBI). *Available on line at: http://cdc.gov/Migrated_Content/Brochures_and_Catalogs/tbi_factos_for_physicians.pdf*

Christensen, B. K., Colella, B., Inness, E., Hebert, D., Monette, G., Bayley, M., & Green, R.E. (2008). Recovery of cognitive function after traumatic brain injury: a multilevel modeling analysis of Canadian outcomes. *Archives of Physical and Medical Rehabilitation, 89 (12 Suppl)*, S3-15.

Cicerone, K., Levin, H., Malec, J., Stuss, D., & Whyte, J. (2006). Cognitive rehabilitation interventions for executive function: moving from bench to bedside in patients with traumatic brain injury. *Journal of Cognitive Neuroscience, 18*(7), 1212-1222.

Cicerone, K. D., & Azulay, J. (2007). Perceived self-efficacy and life satisfaction after traumatic brain injury. *Journal of Head Trauma Rehabilitation, 22*(5), 257-266.

Cohen, J. (1973). ETA-Squared and partial ETA-squared in fixed factor ANOVA designs. *Educational and Psychological Measurement, 33* (107 -112).

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (second edition). Lawrence Erlbaum Associates.

Cohen, R. A., & Waters, W.F. (1985). Psychophysiological correlates of levels and stages of cognitive processing. *Neuropsychologia, 23*(2), 243-256.

Comalli, P. E., Wapner, S., & Werner, H. (1962). Interference effects of Stroop colour-word test in childhood, adulthood, and aging. *Journal of General Psychology, 100*, 47-53.

Corrigan, J. D., & Deutschle, J.J. Jr. (2008). The presence and impact of traumatic brain injury among clients in treatment for co-occurring mental illness and substance abuse. *Brain Injury, 22*(3), 223-231.

Courville, C. B. (1937). *Pathology of the Central Nervous System, Part 4*. Mountain View, CA: Pacific Press Publishing.

Craik, F. I. M. (1990). Changes in memory with normal aging: a functional view. *Advances in Neurology, 51*(210-205).

Crepeau, F., & Scherzer, P. (1993). Predictors and indicators of work status after traumatic brain injury: a meta-analysis. *Neuropsychological Rehabilitation, 3*, 5-35.

Curran, C. A., Ponsford, J.L., & Crowe, S. (2000). Coping strategies and emotional outcome following traumatic brain injury: a comparison with orthopaedic patients. *Journal of Head Trauma Rehabilitation, 15*(6), 1256-1274.

Cusick, C. P., Brooks, C.A., & Whiteneck, G.G. (2001). The use of proxies in community integration research. *Archives of Physical Medicine and Trauma Rehabilitation, 20*(5), 426-435.

Dahlquist, L. M., Czyewski, D.I., Copeland, K.G., Jones, C.L., Taub, E., & Vaughan, J.K. (1993). Parents of children newly diagnosed with cancer: anxiety, coping, and marital distress. *Journal of Paediatric Psychology, 18*(3), 365-376.

Damasio, A. R., Tranel., D., & Damasio, H. (1990). Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. *Behavioural Brain Research, 41*, 81-94.

Dawson, D. R., Levine, B., Schwartz, M.L., & Stuss, D.T. (2004). Acute predictors of real-world outcomes following traumatic brain injury: a prospective study. *Brain Injury, 18*(3), 221-238.

Dawson, D. R., Markowitz, M., & Stuss, D.T. (2005). Community integration status 4 years after traumatic brain injury: participant-proxy agreement. *Journal of Head Trauma Rehabilitation, 82*, 1018-1024.

Dawson, D. R., Cantanzaro, A.M., Firestone, J., Schwartz, M., & Stuss, D.T. (2006). Changes in coping style following traumatic brain injury and their relationship to productivity status. *Brain and Cognition, 60*(2), 214-216.

Dawson, D. R., Schwartz, M.L., Winocur, G., & Stuss, D.T. (2007). Return to productivity following traumatic brain injury: cognitive, psychological, physical, spiritual, and environmental correlates. *Disability and Rehabilitation, 29*, 310-313.

Dawson, M. E., Schell, A.M., & Filion, D.L. (1990). The electrodermal system. In J. T. Cacioppo, & Tassinary, L.G. (Ed.), *Principles of Psychophysiology: Physical, social and inferential elements* (pp. 295-324). New York: Cambridge University Press.

De Leon, M. B., Kirsch, N.L., Maio, R.F., Tan-Schriner, C.T., Millis, S.R., Frederiksen, S., Tanner, C.L & Breer, L. (2009). Baseline predictors of fatigue 1 year after mild head injury. *Archives of Physical Medicine and Rehabilitation, 90*(6), 956-965.

DeYoung, C. G., Quilty, LC., & Peterson, J.B. (2007). Between facets and domains: 10 aspects of the Big Five. *Journal of Personality and Social Psychology, 93*(5), 880-896.

Dikmen, S., Reitan, R.M., & Temkin, N,R. (1983). Neuropsychological recovery in head injury. *Archives of Neurology, 40*, 333-338.

Dikmen, S., McLean, A., & Temkin, N. (1986). Neuropsychological and psychosocial consequences of minor head injury. *Journal of Neurology Neurosurgery and Psychiatry, 49*, 1227-1232.

Dikmen, S., Machamer, L., & Temkin, N. (1993). Psychosocial outcomes in patients with moderate to severe head injury: 2-year follow up. *Brain Injury, 7*, 113-124.

Dikmen, S., Ross, B.L., Machamer, J.E., & Temkin, N.R. (1995). One year psychosocial outcome in head injury. *Journal of the International Neuropsychological Society, 1*(1), 67-77.

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

Dischinger, P. C., Ryb, G.E., Kufera, J.A., & Auman, K.M. (2009). Early predictors of postconcussive syndrome in a population of trauma patients with mild traumatic brain injury. *Journal of Trauma, 66*(2), 289-296.

Doty, R. L., Shaman, P., Dann, M. (1984). Development of the University of Pennsylvania Smell Identification Test: a standardized microencapsulated test of olfactory function. *Physiological Behaviour*, 489-502.

Doty, R. L., Yousem, D.M., Pham, L.T., Kreshak, A., A., Geckle, R., & Lee, W.W. (1997). Olfactory dysfunction in patients with head trauma. *Archives of Neurology, 54*, 1131-1140.

Draper, K., Ponsford, J., & Schonberger, M. (2007). Psychosocial and emotional outcomes 10 years following traumatic brain injury. *Journal of Head Trauma Rehabilitation, 22*(5), 278-287.

Eslinger, P. J., & Damasio, A.R. (1985). Severe disturbance of higher cognition following bilateral frontal lobe ablation: patient EVR. *Neurology, 35*, 1731-1741.

Finset, A., & Andersson, S. (2000). Coping strategies in patients with acquired brain injury: the relationships between coping, apathy, depression and lesion location. *Brain Injury, 14*(10), 887-905.

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

Fleming, A. S., Steiner, M., & Corter, C. (1997). Cortisol, hedonics, and maternal resposivness in human mothers. *Hormones and Behavior, 32*(2), 85-98.

Fleminger, S., & Ponsford, J. (2005). Long term outcome after traumatic brain injury: More attention needs to be paid to neuropsychiatric functioning. *British Medical Journal, 331*, 1419-1420.

Folkman, S., & Lazarus, R. (1988). *Ways of Coping Questionnaire Manual*. Palo Alto: Consulting Psychologists Press.

Freedman, M. (1990). Object alternation and orbitofrontal system dysfunction in Alzheimer's and Parkinson's disease. *Brain and Cognition, 14*, 134-143.

Freedman, M., Black, S., Ebert, P., & Binns, M. (1994). Orbitofrontal function, object alternation and perseveration. *Cerebral Cortex, q8*(1), 18-27.

Fujiwara, E., Schwartz, M.L., Gao, F., Black, S.E., & Levine, B. (2008). Ventral frontal cortex functions and quantified NRI in traumatic brain injury. *Neuropsychologia, 46*(2), 461-474.

Fuster, J. M. (1997). *The Prefrontal Cortex: Anatomy, Physiology, and Neuropsychology of the Frontal Lobe*. New York: Raven.

Gentry, L., Godersky, J.C., & Tompson, B. (1988). MR imaging of head trauma: review of the distribution and radiopathologic features of traumatic lesions. *American Journal of Neuroradiology, 9*, 101-110.

Gerberding, J. L., & Binder, S. (2003). *Report to Congress on mild traumatic brain injury in the United States: Steps to prevent a serious public health problem.* As cited in Moore, E.L, Terryberry-Spohr, L. & Hope, D.,A. (2006).

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

Gordon, W. A., Cantor, J., Ashman, T., & Brown, M. (2006). Treatment of post-TBU executive dysfunction: application of theory to clinical practice. *Journal of Head Trauma Rehabilitation, 21*(2), 156-167.

Grant, D. A., & Berg, E.A. (1948). A behavioural analysis of degree of reinforcement and ease of shifting to new responses in a weight-type card-sorting problem. *Journal of Experimental Psychology, 38*, 404-411.

Green, R. E., Turner, G.R., & Thompson, W.F. (2004). Deficits in facial emotion perception in adults with recent traumatic brain injury. *Neuropsychologia, 42*(2), 133-141.

Green, R. E., Colella, B., Christensen, B., Johns, K., Frasca, D., Bayley, M., & Monette, G. (2008). Examining moderators of cognitive recovery trajectories after moderate to severe traumatic brain injury. *Archives of Physical and Medical Rehabilitation, 89 (12 Suppl).* S16-24.

Green, R. E., Colella, B., Hebert, D.A., Bayley, M., Kang, H.S., Till, C., & Monette, G. (2008). Prediction of return to productivity after severe traumatic brain injury: investigation of optimal neuropsychological tests and timing of assessment. *Archives of Physical and Medical Rehabilitation, 89 (12 Suppl)*, S51-60.

Greenberg, G., Mikulis, D.J., Ng., K., DeSouza, D., & Green, R.E. (2008). Use of diffusion tensor imaging to examine subacute white matter injury progression in moderate to severe traumatic brain injury. *Archives of Physical and Medical Rehabilitation, 89 (12 Suppl)*, S45-50.

Haboubi, N. H., Long, J., Koshy, M., & Ward, A.B. (2001). Short-term sequelae of minor head injury (6 years experience of minor head injury clinic). *Disability Research, 23*(14), 635-638.

Harlow, J. M. (1868). Recovery from the passage of an iron bar through the head. Reprinted in Miller, E. (1993). *History of Psychiatry, 4*, 271-278.

Hawthorne, G., Kaye, A.H., & Gruen, R. (2009). Traumatic brain injury and long-term quality of life: findings from an Australian study. *Journal of Neurotrauma, 23 (epub ahead of print)*.

Hibbard, M. R., Uysal, S., Kepler, K., Bogdany, J., & Silver, J. (1998). Axis I psychopathology in individuals with traumatic brain injury. *Journal of Head Trauma Rehabilitation, 13*, 24-39.

Hiott, D. W., & Labbate, L. (2002). Anxiety disorders associated with traumatic brain injuries. *Neurorehabilitation, 17*, 345-355.

Jennett, B., Snoek, J., Bond, M.R., & Brooks, N. (1981). Disability after severe head injury: observations on the use of the Glasgow Outcome Scale. *Journal of Neurology, Neurosurgery, and Psychiatry, 44*, 285-293.

Jones-Gotman, M., & Zatorre, R.J. (1988). Olfactory identification deficits in patients with focal cerebral excision. *Neuropsychologia, 26*(3), 387-400.

Kaplan, S. P. (1993). Five-year tracking of psychosocial changes in people with severe traumatic brain injury`. *Rehabilitation Counseling Bulletin, 36*(3), 151-159.

Kendall, E., & Terry, D.J. (1996). Psychosocial adjustment following closed head injury: A model for understanding individual differences and predicting outcome. *Neuropsychological Rehabilitation, 6*, 101-132.

Kendall, E., Shum, D., Lack, B., Bull, S., & Fee, C. (2001). Coping following traumatic brain injury: the need for contextually sensitive assessment. *Brain Impairment, 2*, 81-96.

Kirschbaum, C., Pirke, K.M., & Hellhammer, D.H. (1993). The Trier Social Stress Test -- a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76-81.

Klonoff, P. S., Costa, L.D., & Snow, W.G. (1986). Predictors and indicators of quality of life in patients with closed head injury. *Journal of Clinical and Experimental Neuropsychology, 8*(5), 469-489.

Kraus, J. F., & McArthur, D.L. (1996). Epidemiologic aspects of brain injury. *Neurology Clinics, 14*(2), 435-450.

Kraus, M. F., Susmaras, T., Caughlin, B.P., Walker, C.J., Sweeney, J.A., & Little, D.M. (2007). White matter integrity and cognition in chronic traumatic brain injury: a diffusion tensor imaging study. *Brain, 130*, 2508-2519.

Krpan, K. M., Coombs, R., Zinga, D., Steiner, M., & Fleming, A.S. (2005). Experiential and hormonal correlates of maternal behavior in teen and adult mothers. *Hormones and Behavior, 47*(1), 112-122.

Krpan, K. M., Levine, B., Stuss, D.T., & Dawson, D.R. (2007). Executive function and coping at one-year-post traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 29*(1), 36-46.

Kurtz, J. E., Putnam, S.H., & Stone, C. (1998). Stability of normal personality traits after traumatic brain injury. *Journal of Head Trauma Rehabilitation, 13*, 1-14.

Labbate, L. A., & Warden, D.L. (2000). Common psychiatric syndromes and pharmacologic treatments of traumatic brain injury. *Current Psychiatry Reports, 2*, 268-273.

Lanham, R. A., Weissenburger, J.E., Schwab, K.A., & Rosner, M.M. (2000). A longitudinal investigation of the concordance between individuals with traumatic brain injury and family or friends rating on the Katz Adjustment Scale. *Journal of Head Trauma Rehabilitation, 15*, 1123-1138.

Lazarus, R. S., & Folkman, S. (1984). *Stress, Appraisal and Coping*. New York: Springer.

Leach, L. R., Frank, R.G., Bouman, D.E.,. & Farmer, J. (1994). Family functioning, social support an depression after traumatic brain injury. *Brain Injury, 8*(7), 599-606.

Lehermitte, F. (1983). "Utilization Behaviour" and its relation to lesions of the frontal lobes. *Brain 106*, 237-164.

Levin, H. S., & Eisenberg, H.M. (1991). Management of head injury. Neurobehavioural outcomes. *Neurosurgery Clinics of North America, 2*(2), 457-472.

Levine, B., Katz, D., Dade, L, & Black, S. (2002). Novel approaches to the assessment of frontal damage and executive deficits in traumatic brain injury. In D. T. Stuss, Knight, R. (Ed.), *Principles of Frontal Lobe Function* (pp. 448-465). New York: Oxford University Press.

Levine, B., B lack, S.E., Cheung, G., Campbell, A., O'Toole, C., & Schwartz, M.L. (2005). Gambling task performance in traumatic brain injury: relationships to injury severity, atrophy, lesion location , and cognitive and psychosocial outcome. *Cognitive and Behavioral Neurology, 18*(1), 45-54.

Levine, B., Turner, G.R., & Stuss, D.T. (2008). Rehabilitation o frontal lobe functions. In D. T. Stuss, Winocur, G., & Robertson, I.H. (Ed.), *Cognitive Neurorehabilitation: 2nd Edition: Evidence and Application* (pp. 464-486). Cambridge: Cambridge University Press.

Levine, B., Kovacevic, N., Nica, E.I., Cheung, G., Gao, F., Schwartz, M.L., & Black, S.E. (2008). The Toronto traumatic brain injury study: injury severity and quantified MRI. *Neurology, 70*(10), 771-778.

Lo, C., Shifteh, K., Gold, T., Bellow, J.A., & Lipton, M.L. (2009). Diffusion tensor imaging abnormalities in patients with mild traumatic brain injury and neurocognitive impairment. *Journal of Computer Assisted Tomography, 33*(2), 293-297.

Lubusko, A. A., Moore, A.D., Stambrook, M. & Gill, D.D. (1994). Cognitive beliefs following severe traumatic brain injury: association with post-injury employment status. *Brain Injury, 8*(1), 65-70.

Malia, K., Powell, G., & Torode, S. (1995). Coping and psychosocial function after brain injury. *Brain Injury, 9*(6), 607-618.

Martz, E., Livneh, H., Priebe, M., Wuermser, LA., & Ottomanelli, L. (2005). Predictors of psychosocial adaptation among people with spinal cord injury or disorder. *Archives of Physical Medicine and Rehabilitation, 86*(6), 1182-1192.

McCauley, S. R., Boake, C., Levin H.S., Contant, C.F., & Song, J.X. (2001). Postconcussional disorder following mild to moderate traumatic brain injury: anxiety, depression, and social support as risk factors and comorbidities. *Journal of Clinical and Experimental Neuropsychology, 23*, 792-808.

Mcdonald, S., Flanagan, S., Rollines, J., & Kinch, J. (2003). TASIT: A new clinical tool for assessing social perception after traumatic brain injury. *Journal of Head Trauma Rehabilitation, 18*(3), 219-238.

McDonald, S., & Flanagan, S. (2004). Social perception deficits after traumatic brain injury: Interaction between emotion recognition, mentalizing ability, and social communication. *Neuropsychology, 18*(3), 572-579.

McDonald, S., Saunders, J.C. (2005). Differential impairment in recognition of emotion across different media in people with severe traumatic brain injury. *Journal of the International Neuropsychological Society, 11*, 392-399.

McLean, A., Dkimen, S.S., & Temkin, N.R. (1993). Psychosocial recovery after head injury. *Archives of Physical Medicine and Rehabilitation, 74*, 1041-1046.

McLean, S. A., Kirsch, N.L, Tan-Schriner, C.U., Sen, A., Frederiksen, S., Harris, R.E., Maixner, W., & Maio, R.F. (2009). Health status, not head injury, predicts concussion symptoms after minor injury. *American Journal of Emergency Medicine, 27*(2), 182-190.

McMillan, T. M., Williams, W.H., & Bryant, R.A. (2003). Post-traumatic stress disorder and traumatic brain injury: A review of causal mechanisms, assessment and treatment. *Neuropsychological Rehabilitation, 13*, 149-164.

Meares, S., Shores, E.A., Taylor, A.J., Batchelor, J., Bryant, R.A., Baquley, IJ., Chapman, J., Gurka, J., Dawson, K., Capon, L., Marosszeky, J.E. (2008). Mild traumatic brain injury does not predict acute postconcussion syndrome. *Journal of Neurology, Neurosurgery, and Psychiatry, 79*(3), 300-306.

Mishkin, M. (1964). Perseveration of central sets after frontal lesions in monkeys. *See Warren & Akert 1964, pp 219-241*.

Moore, A. D., Stambrook, M. & Peters, L.C. (1989). Coping strategies and adjustment after closed-head injury: a cluster analytical approach. *Brain Injury, 3*(2), 171-175.

Moore, A. D., & Stambrook, M. (1992). Coping strategies and locus of control following traumatic brain injury:relationship to long-term outcome. *Brain Injury, 6*(1), 89-94.

Moore, A. D., & Stambrook, M. (1994). Coping following traumatic brain injury (TBI): Derivation and validation of a TBI sample ways of coping revised subscales. *Brain Injury, 7*, 193-200.

Moore, A. D., & Stambrook., M. (1995). Cognitive moderators of outcome following traumatic brain injury: a conceptual model and implications for rehabilitation. *Brain Injury, 9*(2), 109-130.

Moore, E. L., Terryberry-Spohr, L., & Hope, D.A. (2006). Mild traumatic brain injury and anxiety sequelae: A review of the literature. *Brain Injury, 20*(2), 117-132.

Morton, M. V., & Wehman, P. (1995). Psychosocial and emotional sequelae of individuals with traumatic brain injury: a literature review and recommendations. *Brain Injury, 9*(1), 81-92.

National Centre for Injury Prevention and Control. *Traumatic brain injury in the United States: a report of congress.* Atlanta (GA): Centre for Disease Control and Prevention; 1999.

Nevin, N. C. (1967). Neuropathological changes in the white matter following head injury. *Journal of Neuropathology and Experimental Neurology, 26*, 77-84.

Ng, K., Mikulis, D.J., Glazer, J., Kabani, N., Till, C., Greenberg, G., Thompson, A., Lazinski, D., Agid, R., Colella, B., & Green, R.E. (2008). Magnetic resonance imaging evidence of progression for sub-acute brain atrophy in moderate to severe traumatic brain injury. *Archives of Physical and Medical Rehabilitation, 89 (12 Suppl)*, S35-44.

Ocampo, S., Colantonio, A., & Dawson, D. (1997). Outcomes after head injury: Level of agreement between subjects and their informants. *Occupational Therapy International, 4*, 161-177.

O'Keeffe, F. M., Dockree, P.M., & Robertson, I.H. (2004). Poor insight in traumatic brain injury mediated by impaired error processing? Evidence from electrodermal activity. *Brain Research. Cognitive Brain Research, 22*(1), 101-120.

Ommaya, A. K., & Gennarelli, T.A. (1974). Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. *Brain, 97*, 633-654.

Ouellet, M. C., Sirois, M.J., & Lavoie, A. (2009). Perceived mental health and needs for mental health services following trauma with and without brain injury. *Journal of Rehabilitation Medicine, 41*(3), 179-186.

Owen, A. M., Roberts, A.C., Polkey, C.E., Sahakian, B.J., & Robbins, T.W. (1991). Extra-dimentional versus intra-dimensional set shifting performance folloing frontal lobe excisions, temporal lobe excisions or amygdalo-hippocampectomy in man. *Neuropsychologia, 29*(10), 993-1006.

Ownsworth, T., McFarland, K., & Young, R. (2000). Self-awareness and psychosocial functioning following acquired brain injury: an evaluation of a group support programme. *Neurropsychological Rehabilitation, 10*, 465-484.

Ownsworth, T. I., & Oei, T. P.S. (1998). Depression after traumatic brain injury: Conceptualization and treatment considerations. *Brain Injury, 12*(9), 735-751.

Pagulayan, K. F., Hoffman, J.M., Temkin, N.R., Machamer, J.E., & Dikmen, S.S. (2008). Functional limitations and depression after traumatic brain injury: examination of the temporal relationship. *Archives of Physical and Medical Rehabilitation, 89*(10), 1887-1892.

Pandya, D. N., & Barnes, C.L. (1987). Architecture and connections of the frontal lobes. In E. Perecman (Ed.), *The Frontal Lobes Revisited* (pp. 41-72). New York: IRBN Press.

Pandya, D. N., & Yeterian, E.H. (1996). Morphological correlations of human and monkey frontal lobes. In H. D. AR Damasio, Y Christen (Ed.), *Neurobiology of Decision Making* (pp. 13-46). New York: Springer-Verlag.

Peterson, L. R., & Peterson, M.J. (1959). Short-term attention of individual verbal items. *Journal of Experimental Psychology, 58*, 193-198.

Ponsford, J. L., Olver, J.H., Curran, C., & Ng, K. (1995). Prediction of employment status 2 years after traumatic brain injury. *Brain Injury, 9*(1), 11-20.

Ponsford, J., Whelan-Goodinson, R., & Bahar-Fuchs, A. (2007). Alcohol and drug use following traumatic brain injury: A prospective study. *Brain Injury, 21*(13-14), 1385-1392.

Povlishock, J. T. (1993). Pathobiology of traumatically induced axonal injury in animals and man. *Annals of Emergency Medicine, 22*, 980-986.

Prigatano, G. P. (2005). Disturbance of self-awareness and rehabilitation of patients with traumatic brain injury: a 20-year perspective. *Journal of Head Trauma Rehabilitation, 20*(1), 19-29.

Radice-Neumann, D., Zupan, B., Babbage, D.R. & Willer, B. (2007). Overview of impaired facial affect recognition in persons with traumatic brain injury. *Brain Injury, 21*(8), 807-816.

Rappaport, M., Herrero-Backe, Rappaport, M.L. & Winterfield, K.M. (1989). Head injury outcome up to ten years later. *Archives of Physical and Medical Rehabilitation, 70*, 885-892.

Raven, J. C. (1960). *Guide to the Standard Progressive Matrices*. London: H.K. Lewis.

Raven, J. C., Court, J.H., & Raven, J. (1976). *Manual for Raven's Progressive Matrices*. London: H.K. Lewis.

Robertson, I. H., Manly, T., Andrade, J., Baddeley, B.T., & Yiend, J. (1997). 'Oops!' Performance correlates of everyday attentional failures in traumatic brain injury and normal subjects. *Neuropsychologia, 35*(6), 747-758.

Rolls, E. T. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex, 10*, 284-294.

Ruffolo, C. F., Friedland, J.F., Dawson, D.R., Colantonio, A., & Lindsay, P.H. (1999). Mild traumatic brain injury from motor vehicle accidents: factors associated with return to work. *Archives of Physical Medicine and Rehabilitation, 80*(4), 392-398.

Ruttan, L., Martin, K., Liu, A., Colella, B., & Green, R.E. (2008). Long-term cognitive outcome in moderate to severe traumatic brain injury: a meta-analysis examining timed and untimed tests at 1 and 4.5 or more years after injury. *Archives of Physical and Medical Rehabilitation, 89 (12 Suppl)*, S69 - 76.

Sanchez-Navarro, J. P., Martinez-Selva, M., & Roman, F. (2005). Emotional response in patients with frontal brain damage: Effects of affective valence and information content. *Behavioural Neuroscience, 119*(1), 87-97.

Seel, R. T., Kreutzer, J.S., & Sander, A.M. (1997). Concordance of patient's and family members' ratings of neurobehavioural functioning after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*(78).

Seel, R. T., Kreutzer, J.S., Rosenthal, M., Hammond, F.M., Corrigan, J.D.,& Black, K. (2003). Depression after traumatic brain injury: a National Institute on Disability and Rehabilitation Research model systems multicenter investigation. *Archives of Physical and Medical Rehabilitation, 84*, 177-184.

Shallice, T., & Burgess, P.W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain, 114*, 727-741.

Shames, J., Treger, I., Ring, H., & Giaquinto, S. (2007). Return to work following traumatic brain injury. *Disability and Rehabilitation, 29*(17), 1387-1395.

Shane, M. S., & Peterson, J.B. (2004). Defensive copers show a deficit in passive avoidance learning on Newman's Go/No-Go Task: Implications for self-deception and socialization. *Journal of Personality, 12*(5), 939-966.

Sosnowski, T., Nurzynska, M., & Polec, M. (1991). Active-passive coping and skin conductance and heart rate changes. *Psychophysiology, 28*(6), 665-672.

Spielberger, C. D. (1983). *State-Trait Anxiety Inventory for Adults.* Palo Alto, CA: Consulting Psychologists Press.

Spreen, O., & Strauss, E. (1998). *A compendium of Neuropsychological Tests; Administration, norms, and commentary* (Second ed.). New York: Oxford University Press.

Statistics Canada. Health Division. Institutional Care Statistics Section: *Hospital Morbidity: 1979-80 and 1980-81.* Supply and Services Canada. Ottawa, 1984.

Stuss, D. T., Ely, P., Hugenholtz, H., Richard, M.T., LaRochelle, S., Poirier, C.A., & Bell, I. (1985). Subtle neuropsychological deficits in patients with good recovery after closed head injury. *Neurosurgery, 17*(1), 41-47.

Stuss, D. T., & Benson, D.F. (1986). *The frontal lobes*. New York: Raven Press.

Stuss, D. T. (1991). Disturbance of self-awareness of Deficit After Brain Injury. In G. P. Prigatano, Schacter, D. (Ed.), *Awareness of Deficit After Brain Injury.* (pp. 63-83). New York: Oxford University Press.

Stuss, D. T., & Gow, C.A. (1992). 'Frontal Dysfunction after traumatic brain injury. *Neuropsychiatry, Neuropsychology and Behavioural Neurology, 5*(4), 272-282.

Stuss, D. T., Gow, C.A., & Hetherington, C.R. (1992). "No longer Gage": frontal lobe dysfunction and emotional changes. *Journal of Consulting and Clinical Psychology, 60*(3), 349-359.

Stuss, D. T., Alexander, M.P., Hamer, L., Palumbo, C., Dempster, R., Binns, M., Levine, B., & Izukawa, D. (1998). The effects of focal anterior and posterior brain lesions on verbal fluency. *Journal of the International Neuropsychological Society, 4*(3), 265-278.

Stuss, D. T., & Alexander, M.P. (2000). Executive functions and the frontal lobes: a conceptual view. *Psychological Research, 63*, 289-298.

Stuss, D. T., Levine, B., Alexander, M.P., Hong, J., Palumbo, C., Hamer, L., Murphy, K.J., & Izukawa, D. (2000). Wisconsin card sorting test performance in patients with focal frontal and posterior brain damage: effects of lesion location and test structure on separable cognitive processes. *Neuropsychologia, 38*, 388-402.

Stuss, D. T., Floden, D., Alexander, M.P., Levine, B., & Katz, D. (2001). Stroop performance in focal lesion patients: dissociation of processes and frontal lobe lesion location. *Neuropsychologia, 39*, 771-786.

Stuss, D. T., Bisschop, S.M., Alexander, M.P., Levine, B., Katz, D., & Izukawa, D. (2001). The trail making test: A study in focal lesion patients. *Psychological Assessment, 13*(2), 230-239.

Stuss, D. T., & Levine, B. (2002). Adult clinical neuropsychology: Lesions from studies of the frontal lobes. *Annual Reviews in Psychology, 53*, 401-433.

Stuss, D. T., Alexander, M.P., Floden, D., Binns, M.A., Levine, B., McIntosh, A.R., Rajah, N., & Hevenor, S.J. (2002). Fractionation and localization of distinct frontal lobe processes: Evidence from focal lesions in humans. In Stuss. D.T., & Knight, R.T. (Ed.), *Principles of Frontal Lobe Function* (pp. 392-407). New York: Oxford University Press.

Stuss, D. T., Murphy, K.J., Binns, M.A. & Alexander, M.P. (2003). Staying on the job: the frontal lobes control individual performance variability. *Brain, 126 (Pt11)*, 2363-2380.

Stuss, D. T. (2007). New approaches to prefrontal lobe testing. In B. Miller, & Cummings, J (Ed.), *The Human Frontal Lobes: Functions and Disorders* (2nd ed., pp. 292-305). New York: Guilford Press.

Stuss, D. T., & Binns, M.A. (2008). The patient as a moving target; The importance to rehabilitation of understanding variability. In D. T. Stuss, Winocur, G. & Robertson, I.H. (Ed.), *Cognitive Neurorehabilitation, Second Edition, Evidence and Application* (pp. 39-61): Cambridge University Press.

Tate, R. L., Lulham, J.M., Broe, G.A. Strettles, B., & Pfaff, A. (1989). Psychosocial outcome for the survivors of severe blunt head injury: The results from consecutive series of 100 patients. *Journal of Neurology, Neurosurgery, and Psychiatry, 52*, 1128-1134.

Teasdale, G., Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet, 2*(7872), 81-84.

Temkin, N., McLean, A., Dikmen, S., Gale, J., Berger, M., & Almes, M.J. (1988). Development and evaluation of modifications to the Sickness Impact Profiles for head injury. *Journal of Clinical Epidemiology, 41*, 41-57.

Thomsen, I. V. (1992). Late psychosocial outcomes in severe traumatic brain injury. Preliminary results of a third follow-up study after 20 years. *Scandinavian Journal of Rehabilitation Medicine Supplement, 26*, 142-152.

Thurman, D. J., Alverson, C.M.S., Dunn, K.A., Guerrero, J.M.S., & Sniezek, J.E. (1999). Traumatic brain injury in the United States: A public health perspective. *Journal of Head Trauma Rehabilitation, 14*(6), 602-615.

Till, C., Colella, B., Verwegen, J., & Green, R.E. (2008). Post recovery cognitive decline in adults with traumatic brain injury. *Archives of Physical and Medical Rehabilitation, 89 (12 Supp)*, S25-343.

Tomberg, T., Toomela, A., Pulver, A., & Tikk, A. (2005). Coping strategies, social support, and life orientation and health-related quality of life following traumatic brain injury. *Brain Injury, 19*(14), 1181-1190.

Troyer, A. K., Moscovitch, M., Winocur, G., Alexander, M.O.,& Stuss, D. (1998). Clustering and switching on verbal fluency: the effects of focal frontal- and temporal-lobe lesions. *Neuropsychologia, 36*, 499-504.

Tyerman, A., & Humphrey, M. (1984). Changes in self-concept following severe head injury. *International Journal of Rehabilitation Research, 7*(1), 11-23.

Vanderploeg, R. D., Belanger, H.G., & Curtiss, G. (2009). Mild traumatic brain injury and posttraumatic stress disorder and their associations with health symptoms. *Archives of Physical Medicine and Rehabilitation, 90*(7), 1084:1093.

van Reekum, R., Bolago, I., Finlayson, M.A., Garner, S. & Links, P.S. (1996). Psychiatric disorders after traumatic brain injury. *Brain Injury, 10*(5), 319-327.

Weinberger, D. A., Schwartz, G.E., & Davidson, R.J. (1979). Low-anxious, high-anxious, and repressive coping styles: Psychometric patterns and behavioural and physiological responses to stress. *Journal of Abnormal Psychology, 88*, 396-380.

Whelan-Goodinson, R., Ponsford, J., & Schonberger, M. (2008). Association between psychiatric state and outcome following traumatic brain injury. *Journal of Rehabilitation Medicine, 40*(10), 850-857.

Wilson, B. A., Alderman, N., Burgess, P.W., Emslie, H., & Evans, J.J. (1996). England Patent No. Thames Valley Test Company.

Wood, R. L., & Rutterford, N.A. (2006). Demographic and cognitive predictors of long-term psychosocial outcomes following traumatic brain injury. *Journal of the International Neuropsychological Society, 12*(3), 350-358.

Wood, R. L., & Williams, C. (2008). Inability to empathize following traumatic brain injury. *Journal of the International Neuropsychological Society, 14*(2), 289-296.

Yousem, D. M., Geckle, R.J., Bilker, W.B. et al. (1999). Posttraumatic smell loss: relationship of psychophysical tests and volumes of the olfactory bulbs and tracts and the temporal lobes. *Academic Radiology, 6*, 264-272.

Zachary, R. A. (1982). *Shipley Institute of Living Scale, Revised Manual*. Los Angeles: Western Psychological Services.

Zahn, T. P., Grafman, J. & Tranel, D. (1999). Frontal lobe lesions and electrodermal activity: effects of significance. *Neuropsychologia, 37*(11), 1227-1241.

Ziino, C., & Ponsford, J. (2006). Vigilance and fatigue following traumatic brain injury. *Journal of the International Neuropsychological Society, 12*, 100-110.

APPENDIX A

Baycrest Psychosocial Stress Test Script

"As you know, we are interested in the relationship between brain function and physiological arousal. This next procedure involves you giving a speech about crime in Toronto. In a moment, I will leave the room. There will be a ten minute delay before a colleague of mine, Dr. X, will enter the room. In the meantime, you are free to browse through any of the materials found in this room, if you wish, but you do not have to do so. When Dr. X prompts you to do so, you should introduce yourself and proceed to your speech detailing crime in Toronto. Your speech should be 5 minutes in duration. Dr. X will listen to your speech, and evaluate your performance. She is especially trained in voice frequency analysis, as well as analysis of both verbal and non-verbal behaviour. Also, while you are giving your speech, these two recorders will be operating. One will record your speech; the other will record your movements. The videotape will be reviewed by a panel of expert judges (including Dr. X). Your performance will be judged a second time based on voice frequency analysis, nonverbal behaviour and content. This research is very important, so please take it seriously. Do you have any questions? Do you understand what the camera is for? Do you understand what the tape recorders are for? Fine, I'll start the tape recorders, and I'll be back in about 20 minutes."

List of Prompts for the BPST

In the event that a participant ceased speaking before 5 minutes had elapsed, the confederate prompted the participant with the following questions.

1. Do you think that gun control laws in this province are adequate?
2. Do you agree with allowing convicted felons out of jail before their sentence is complete?
3. Is Toronto a safe place to live?
4. How could we make Toronto a safer place to live?
5. Would you consider leaving Toronto because of the crime rate? Why?

Table 1A. Correlation coefficients (Spearman's *rho*) among behaviours included in the Total Planful Score (n = 48)

	Write	**Review**	**Think/Write**
Write	-	.543 *p* <.001	.734 *p* <.001
Review	-	-	.646 *p* <.001

Table 2A Correlation coefficients (Spearman's *rho*) among behaviours included in the Total Avoidant Score

	Magazine	**Puzzle**	**Stare**	**Other**
Magazine	-	.514 *p* <.001	.187 *p* = .102	.292 *p* = .022
Puzzle	-	-	.309 *p* = .016	.211 *p* =.075
Stare	-	-	-	.402 *p* =.002

APPENDIX B

Table 1B: Mean (SD) group differences on the Alpha Span and Brown Peterson Procedure
* Indicates a significant difference between control and TBI groups ($p<.05$)

	Control (n=23)	**Mild (n=10)**	**Mod/Sev (n=17)**
Alpha Score	6.3 (1.7)	5.9 (1.7) η_p^2 =.01	5.9 (1.7) η_p^2 =.01
Alpha Span	5.3 (1.4)	4.7 (0.9) η_p^2 =.05	4.5 (1.0) η_p^2 =.08
Brown 0 delay	15.0 (0)	15.0 (0) η_p^2 = .00	15.0 (0) η_p^2 =.00
Brown 3 delay	13.7 (1.4)	13.2 (1.9) η_p^2 =.02	12.1 (2.9) η_p^2 =.12
Brown 9 delay	10.6 (3.3)	10.3 (2.4) η_p^2 =.00	8.2 (3.3) η_p^2 =.12
Brown 18 delay	8.7 (3.0)	7.0 (3.9) η_p^2 =.06	6.6 (3.7) η_p^2 =.09
Brown Total Recalled	48.1 (6.3)	45.5 (7.8) η_p^2 =.03	**38.8 (11.7)*** **η_p^2 =.21**

Table 2B: Mean (SD) scores on the Stroop Test
* Indicates a significant difference between control and TBI groups (p<.05)

	Control (n=23)	**Mild (n=10)**	**Mod/ Sev (n=14)**
Raw Time WN (sec)	42.3 (6.8)	**79.0 (83.0)*** **η_p^2=.13**	56.1 (20.7) η_p^2=.20
Raw Time CN (sec)	57.4 (10.4)	65.9 (11.8) η_p^2=.12	**78.0 (28.3)*** **η_p^2=.22**
Raw Time INT (sec)	102.9 (27.1)	135.1 (48.6) η_p^2=.16	130.0 (47.7) **η_p^2=.12**
WN errors	0.17 (0.49)	0.10 (0.31) η_p^2=.01	**0.79 (1.0)*** **η_p^2=.14**
CN errors	0.48 (0.79)	0.90 (1.1) η_p^2=.04	0.71 (0.9) η_p^2=.02
INT errors	0.6 (0.98)	2.2 (3.0) η_p^2=.14	3.1 (5.4) η_p^2=.12
(CN-WN)/WN	0.36 (0.21)	0.15 (0.37) η_p^2=.12	0.40 (0.21) η_p^2=.01
(INT-CN)/CN	0.78 (0.31)	1.0 (0.48) η_p^2=.09	**0.66 (0.24)*** **η_p^2=.04**

Table 3B: Mean (SD) group differences on the Trail Making Test
* Indicates a significant difference between control and TBI groups ($p<.05$)

	Control (n=23)	Mild (n=10)	Mod/ Sev (n=17)
Raw Time A (sec)	24.0 (6.4)	30.9 (8.8) $\eta_p^2=.17$	**39.0 (16.1)*** $\boldsymbol{\eta_p^2=.30}$
Raw Time B (sec)	53.5 (22.8)	70.2 (22.2) $\eta_p^2=.11$	**91.2 (55.2)*** $\boldsymbol{\eta_p^2=.18}$
Errors on A	2.3 (10.4)	0.3 (0.6) $\eta_p^2=.01$	0.1 (0.3) $\eta_p^2=.02$
Errors on B	0.3 (0.6)	0.2 (0.4) $\eta_p^2=.01$	0.3 (0.6) $\eta_p^2=.01$
(B-A)/A	1.2 (0.8)	1.3 (0.4) $\eta_p^2=.00$	1.2 (0.7) $\eta_p^2=.00$

Table 4B: Mean (SD) differences on the Wisconsin Card Sorting Test

	Control (n=22)	**Mild (n=10)**	**Mod/ Sev (n=15)**
Total Correct	104 (12)	101 (13) η_p^2 =.01	94 (25) η_p^2 =.08
Ppc	13.7 (6.2)	17.4 (6.5) η_p^2 =.07	19.5 (26.7) η_p^2 =.03
Ppr	2.8 (5.7)	3.6 (4.2) η_p^2 =.01	3.4 (3.9) η_p^2 =.00
Set Loss	1.4 (1.4)	1.5 (1.1) η_p^2 =.00	1.6 (2.0) η_p^2 =.00
Categories Complete	8.5 (1.9)	7.9 (2.2) η_p^2 =.03	6.2 (4.1) η_p^2 =.13

Table 5B: Mean (SD) group differences on the Zoo Map Test.

	Control (n=23)	Mild (n=10)	Mod/ Sev (n=18)
Plan (sec)	96 (77)	124 (128) η_p^2 =.02	166 (183) η_p^2 =.07
Total (sec)	223 (124)	269 (124) η_p^2 =.03	322 (180) η_p^2 =.09
Errors	2.6 (4.0)	2.5 (2.1) η_p^2 =.00	3.5 (3.7) η_p^2 =.01
Sequence	4.6 (2.6)	3.8 (2.4) η_p^2 =.02	4.4 (3.6) η_p^2 =.00

Table 6B: Mean (SD) group differences on the Verbal Fluency Test
* Indicates a significant difference between control and TBI groups ($p<.05$)

	Control (n=21)	**Mild (n=10)**	**Mod/ Sev (n=16)**
FAS – F	14.8 (3.7)	15.2 (6.8) η_p^2 =.00	13.1 (4.1) η_p^2 =.05
FAS – A	12.7 (3.7)	11.8 (5.4) η_p^2 =.01	12.0 (3.4) η_p^2 =.01
FAS – S	15.6 (4.7)	14.9 (4.2) η_p^2 =.01	12.2 (4.1) η_p^2 =.13
Category	29.5 (7.3)	28.2 (6.2) η_p^2 =.01	**23.0 (8.5)*** **η_p^2 =.15**
Incorrect	1.4 (2.1)	1.1 (0.9) η_p^2 =.01	1.0 (1.0) η_p^2 =.02
Perseveration	1.2 (1.5)	1.4 (1.5) η_p^2 =.00	1.1 (1.1) η_p^2 =.00

Table 7B: Mean (SD) group differences in errors of commission during the SART.
* Indicates a significant difference between control and TBI groups ($p<.05$)

	Control (n=23)	Mild (n=9)	Mod/Sev (n=18)
SART 1 – Commission	7.9 (5.3)	10.5 (6.3) η_p^2 =.05	9.1 (4.8) η_p^2 =.02
Awareness – Commission	6.8 (3.9)	8.5 (4.9) η_p^2 =.04	**9.3 (4.0)* η_p^2 =.10**
Feedback – Commission	8.0 (5.1)	10.1 (6.0) η_p^2 =.03	**12.1 (6.6)* η_p^2 =.11**
SART 2 – Commission	4.7 (3.7)	6.3 (5.0) η_p^2 =.03	**7.8 (6.1)* η_p^2 =.10**

Table 8B: Mean (SD) group differences in errors of omission on the SART
* Indicates a significant difference between control and TBI groups ($p<.05$)

	Control (n=23)	**Mild (n=9)**	**Mod/Sev (n=18)**
SART 1 – Omission	2.3 (4.4)	3.5 (4.6) η_p^2 =.01	6.5 (9.2) η_p^2 =.08
Awareness – Omission	2.3 (2.9)	**11.4 (16.4)*** **η_p^2 =.19**	**8.1 (11.0)*** **η_p^2 =.13**
Feedback – Omission	3.0 (5.0)	10.6 (19.7) η_p^2 =.09	11.9 (24.0) η_p^2 =.07
SART 2 – Omission	1.4 (3.7)	7.6 (20.7) η_p^2 =.06	**14.5 (14.7)*** **η_p^2 =.14**

Table 9B: Mean (SD) group differences on the Object Alternation Task and the Smell Identification Test.
* Indicates a significant difference between control and TBI groups ($p<.05$)

	Control (n=23)	**Mild (n=10)**	**Mod/ Sev (n=17)**
OA Errors	26.5 (17.7)	32.2 (22.2) $\eta_p^2=.02$	37.8 (15.4) $\eta_p^2=.11$
OA Trials	63.8 (31.5)	71.2 (36.4) $\eta_p^2=.01$	85.3 (21.1) $\eta_p^2=.14$
Smell ID	10.9 (0.9)	10.0 (1.9) $\eta_p^2=.10$	**9.1 (2.7)*** $\boldsymbol{\eta_p^2=.18}$

Table 10B: Mean (SD) group differences on The Awareness of Social Inference Task
* Indicates a significant difference between control and TBI groups (p<.05)

	Control (n=22)	**Mild (n=10)**	**Mod/Sev (n=16)**
Positive	10.2 (1.2)	9.2 (1.0) η_p^2=.17	9.4 (1.3) η_p^2=.10
Negative	14.4 (1.3)	13.1 (1.5) η_p^2=.17	**11.6 (2.4)*** **η_p^2=.35**
Total A	24.7 (1.8)	**22.3 (1.4)*** **η_p^2=.31**	**21.1 (3.0)*** **η_p^2=.35**
Sincere	15.8 (2.4)	15.1 (3.5) η_p^2=.02	16. 6(3.6) η_p^2=.02
Sarcasm	18.5 (2.1)	16.1 (2.4) η_p^2=.22	**15.4 (4.1)*** **η_p^2=.21**
Paradoxical Sarcasm	18.7 (1.7)	18.0 (2.0) η_p^2=.04	17.6 (3.4) η_p^2=.05
Total B	54.0 (4.7)	49.2 (4.3) η_p^2=.20	50.0 (6.8) η_p^2=.11
Lies	28.1 (2.7)	26.0 (4.7) η_p^2=.08	26.3 (4.9) η_p^2=.05
Sarcasm	28.0 (3.1)	25.3 (3.3) η_p^2=.14	**23.7 (4.8)*** **η_p^2=.23**
Total C	56.1 (5.0)	51.1 (4.3) η_p^2=.20	**50.1 (8.0)*** **η_p^2=.19**

Table 11B: Mean (SD) group differences on the Iowa Gambling Task

	Control (n=23)	**Mild (n=10)**	**Mod/Sev (n=17)**
Overall Diff	-1.8 (29.4)	-7.0 (9.5) η_p^2 =.01	-6.8 (27.0) η_p^2 =.01
Block 1 Diff	-4.1 (3.8)	-2.6 (4.2) η_p^2 =.03	-1.6 (4.3) η_p^2 =.09
Block 2 Diff	-1.8 (5.6)	0.0 (4.1) η_p^2 =.03	-1.1 (4.1) η_p^2 =.00
Block 3 Diff	0.6 (6.8)	0.4 (5.1) η_p^2 =.00	-1.6 (7.9) η_p^2 =.03
Block 4 Diff	1.0 (11.1)	-3.2 (4.7) η_p^2 =.04	-1.8 (6.7) η_p^2 =.02
Block 5 Diff	2.3 (11.6)	-1.6 (4.9) η_p^2 =.03	-0.3 (8.2) η_p^2 =.02

Table 12B: Mean (SD) group differences on the ID/ED Reversal Learning Task
* Indicates a significant difference between control and TBI groups ($p<.05$)
Note: Statistical differences based on LOG transformed data

	Control (n=23)	**Mild (n=10)**	**Mod/Sev (n=17)**
SD errors	8.3 (4.0)	7.8 (1.3) $\eta_p^2 = .00$	7.5 (1.3) $\eta_p^2 = .01$
SDR errors	7.9 (2.4)	7.5 (1.2) $\eta_p^2 = .01$	7.8 (1.6) $\eta_p^2 = .00$
CD errors	10.1 (8.4)	12.1 (11.0) $\eta_p^2 = .01$	10.7 (10.2) $\eta_p^2 = .00$
CDR errors	10.7 (6.2)	7.6 (0.8) $\eta_p^2 = .09$	11.5 (8.2) $\eta_p^2 = .00$
ID errors	7.5 (2.4)	**8.4 (2.9)*** $\boldsymbol{\eta_p^2 = .94}$	**9.1 (4.9)*** $\boldsymbol{\eta_p^2 = .95}$
IDR errors	8.2 (2.9)	9.3 (4.0) $\eta_p^2 = .03$	9.7 (6.5) $\eta_p^2 = .07$
ED errors	11.6 (6.6)	21.1 (16.6) $\eta_p^2 = .14$	14.7 (13.8) $\eta_p^2 = .01$
EDR errors	12.4 (12.1)	15.9 (14.8) $\eta_p^2 = .01$	9.7 (4.8) $\eta_p^2 = .01$

APPENDIX C

Table 1C: Mean (SD) group differences on the Big Five Aspect Scale
* Indicates a difference between TBI and control groups ($p<.05$)

	Control (n=23)	**Mild (n=10)**	**Mod/Sev (n=17)**
Neuroticism	30.6 (3.9)	33.3 (2.7) $\eta_p^2 = .10$	32.1 (4.7) $\eta_p^2 = .03$
Agreeableness	30.9 (3.0)	30.47 (2.4) $\eta_p^2 = .01$	30.8 (2.3) $\eta_p^2 = .00$
Conscientiousness	32.0 (2.9)	**35.5(4.7)*** $\boldsymbol{\eta_p^2 = .18}$	30.9 (3.4) $\eta_p^2 = .03$
Extraversion	33.2 (3.3)	36.1 (8.4) $\eta_p^2 = .06$	32.7 (4.5) $\eta_p^2 = .00$
Openness	32.7 (2.7)	**30.2 (3.0)*** $\boldsymbol{\eta_p^2 = .14}$	31.8 (3.2) $\eta_p^2 = .03$

APPENDIX D

Table 1D: Mean (SD) group differences on the Beck Depression Inventory, and the Taylor Manifest Anxiety Scale.
* Indicates a difference between TBI and control groups ($p<.05$)
** Indicates a difference between TBI and control groups ($p<.001$)

	Control (n=24)	**Mild (n=9)**	**Mod/ Sev (n=17)**
Beck Depression Inventory	6.5 (7.6)	**20.7 (7.2)**** η_p^2 **=.43**	**12.3 (9.4)*** η_p^2 **=.11**
Taylor Manifest Anxiety Scale	12.7 (8.3)	**25.1 (7.0)**** η_p^2 **=.33**	14.3 (8.0) η_p^2 =.01

APPENDIX E

Table 1E: Mean (SD) group differences in heart rate across blocks of the BPST.

	Control (n=22)	Mild (n=10)	Mod/ Sev (n=18)
Baseline Heart Rate for BPST	71.1 (8.4)	72.9 (13.3) η_p^2=.00	73.1 (11.8) η_p^2=.01
Heart Rate for Prep Period	80.0 (11.1)	73.3 (6.4) η_p^2=.09	78.6 (12.0) η_p^2=.00
Heart Rate for Speech	87.3 (12.3)	79.8 (10.7) η_p^2=.08	82.1 (14.2) η_p^2=.04
Heart Rate for Math	83.3 (13.4)	76.7 (12.3) η_p^2=.06	80.4 (14.1) η_p^2=.01
Return to Baseline Heart Rate	68.9 (7.8)	70.6 (13.5) η_p^2=.01	71.1 (10.0) η_p^2=.02

Table 2E: Mean (SD) group differences in heart rate variability across blocks of the BPST.

	Control (n=22)	**Mild (n=10)**	**Mod/ Sev (n=18)**
Baseline Variability for BPST	114.7 (34.5)	118.6 (57.6) η_p^2 =.00	101.4 (36.5) η_p^2 =.04
Variability for Prep Period	98.0 (32.5)	91.2 (17.2) η_p^2 =.01	100.6 (46.6) η_p^2 =.00
Variability for Speech	66.7 (25.1)	79.8 (31.5) η_p^2 =.05	75.5 (41.9) η_p^2 =.02
Variability for Math	56.9 (23.0)	62.5 (22.9) η_p^2 =.01	67.8 (55.6) η_p^2 =.02
Return to Baseline Variability	118.8 (34.1)	110.1 (34.8) η_p^2 =.01	120.2 (48.9) η_p^2 =.00

Table 3E: Mean (SD) group differences in skin conductance level across blocks of the BPST.

	Control (n=20)	**Mild (n=10)**	**Mod/ Sev (n=16)**
Baseline skin conductance level	3.9 (2.6)	4.8 (4.1) η_p^2 =.02	3.3 (1.6) η_p^2 =.02
Prep skin conductance level	6.2 (3.3)	7.2 (5.1) η_p^2 =.01	6.5 (2.7) η_p^2 =.00
Speech skin conductance level	6.7 (3.5)	9.2 (5.2) η_p^2 =.07	7.6 (3.0) η_p^2 =.02
Math skin conductance level	6.5 (3.3)	8.7 (5.6) η_p^2 =.06	7.5 (3.0) η_p^2 =.02
Return to baseline skin conductance level	6.6 (3.7)	8.5 (5.2) η_p^2 =.04	6.8 (3.1) η_p^2 =.00

Table 4E: Mean (SD) group differences in cortisol across blocks of the BPST.

	Control (n=23)	**Mild (n=10)**	**Mod/ Sev (n=16)**
Time 1 Cortisol	0.30 (0.24)	0.24 (0.10) η_p^2 =.02	0.27 (0.19) η_p^2 =.00
Time 2 Cortisol	0.26 (0.16)	0.20 (0.09) η_p^2 =.04	0.26 (0.17) η_p^2 =.00
Time 3 Cortisol	0.36 (0.25)	0.17 (0.10) η_p^2 =.14	0.23 (0.20) η_p^2 =.07

APPENDIX F

Table 1F: Correlation coefficients (Pearson's r) among measures included in the executive function composite.

	Stroop (INT-CN)/CN	**Alpha Score**	**Brown Peterson 18 sec**	**WCST Set Loss**	**Zoo Map Sequence**
Stroop (INT-CN)/CN	-	-.403 *p* = .005 n = 47	-.417 *p* = .004 n = 47	.312 *p* = .033 n = 47	-.371 *p* = .010 n = 47
Alpha Score	-	-	.277 *p* =.052 n = 50	-.455 *p* =.001 n = 48	.401 *p* = .004 n = 50
Brown Peterson 18 sec	-	-	-	-.412 *p* =.004 n = 48	.034 *p* =.815 n = 50
WCST Set Loss	-	-	-	-	-.054 *p* =.714 n = 48

Table 2F: Correlation coefficients (Pearson's r) among measures included in the VMPFC function composite.

	Smell ID Total Correct	**Object Alternation Tot Errors**	**ED Reversal Errors**
Smell ID Total Correct	-	-.246 *p* = .096 n = 47	-.257 *p* = .075 n = 49
Object Alternation Tot Errors			.375 *p* = .009 n = 48

Table 3F: Correlation coefficients (Pearson's r) among measures included in the reactive composite.

	Beck Depression Inventory	Taylor Manifest Anxiety	BFI Neuroticism	BFI Concien-tiousness	STAI Post BPST
Beck Depression Inventory	-	.809 *p* = .000 n = 50	.400 *p* = .004 n = 50	.350 *p* = .013 n = 50	.370 *p* = .010 n = 48
Taylor Manifest Anxiety	-	-	.455 *p* = .001 n = 51	.479 *p* = .000 n = 51	.421 *p* = .003 n = 49
BFI Neuroticism	-	-	-	.310 *p* = .027 n = 51	.273 *p* = .058 n = 49
BFI Concien-tiousness	-	-	-	-	.354 *p* = .012 n = 49

Table 4F: Correlation coefficients (Pearson's r) among measures included in the openness/agreeableness composite.

	BFI Agreeableness	**BFI Openness**
BFI Agreeableness	-	.291 *p* = .038 n = 51

Table 5F: Correlation coefficients (Pearson's r) among measures included in the sociodemographic composite.

	Shipley	6 Hour CGS	Age In Years
Shipley	-	.287 *p* = .139 n = 28	.327 *p* = .019 n = 51
6 Hour CGS	-	-	.472 *p* = .011 n = 28