Predictors of the Development and Maintenance of Posttraumatic Stress Disorder Following a Motor Vehicle Accident:
A Path Analytic Approach

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BA (Psych) Hons I

A thesis submitted in partial fulfilment of the requirements for the degree of
Doctor of Philosophy (Clinical Psychology)
The University of Newcastle
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Declaration

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(Signed): ..........................................................
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Abstract

Motor vehicle accidents (MVAs) are ubiquitous events that often result in psychological distress. Identification of factors associated post-MVA psychopathology is important from a treatment perspective to reduce the burden on individual and community resources. The main aim throughout this thesis was to investigate the influence and interaction of pre, peri, and post-trauma factors on the development and maintenance of Posttraumatic Stress Disorder (PTSD) and, in doing so, address some of the inconsistent findings from previous studies. The sample \( (n = 337, 54\% \text{ female}) \) comprised hospital accident and emergency attendees who completed a self-report postal screening survey approximately 1-month post-accident, follow-up surveys at 3-months \( (n = 128, 63\% \text{ female}) \) and 6-months \( (n = 58, 65\% \text{ female}) \) and a structured clinical interview at 9-months post-MVA \( (n = 51, 63\% \text{ female}) \). A path analytic approach, which facilitates the use of multiple dependent variables and mediation models, was used to investigate the proposed sequential interaction of pre, peri, and post-MVA factors on the development and maintenance of PTSD following an MVA.

Results from the Phase 1 component of the study showed the important role of previous emotional vulnerability factors, and highlighted the mediating role of peritraumatic dissociative experiences, in the presence of extreme fear due to increasing levels of accident severity, on initial post-MVA distress. An exploratory component of this phase of the study showed that participants who were more fatigued at the time of the accident were more likely to experience peritraumatic dissociation.
Results from Phase 2 showed that interrelated premorbid psychological factors and exposure characteristics independently predicted peritraumatic dissociation which, in turn, contributed to initial post-trauma distress and subsequent PTSD symptom severity. Participants who experienced initial sadness (partly influenced by prior emotional problems) in the first month following MVA, and physical disability at 3-months (influenced by injury severity and initial sadness), were at greater risk for depression as well as PTSD symptom severity, which suggests a partial shared vulnerability. The consumption of alcohol at hazardous levels was evident at 3-months post-accident, however, this was not associated with post-MVA psychopathology. Results indicated that experiencing an MVA did not increase the likelihood of alcohol usage since participants were drinking at hazardous levels before their MVA. An exploratory component of this phase of the study revealed that altered awareness (a peritraumatic dissociative response) was predictive of PTSD and symptom severity.

The Phase 3 study highlighted the influential role of subjective appraisal of injury severity and emotional response in maintaining the perception of physical disability. Results also provided evidence that those participants who felt anxious and fearful at 1-month, had sustained physical disability, and who continued to dissociate, were at greater risk of PTSD. At 6-months post-MVA an increase in the consumption of alcohol at hazardous levels was associated with severity of PTSD symptoms. Results suggest that a change in drinking patterns reflects a form of maladaptive coping in response to distressing intrusions. A neurotic predisposition was a vulnerability factor for acute dissociation, but not for PTSD at either 3 or 6-months post-accident, increased levels post-MVA suggest that neuroticism may be a consequence rather than a cause of PTSD.
The Phase 4 component of the study showed that for the majority of MVA survivors with PTSD, symptoms had remitted at 9-months. This phase of the study also provided evidence, central to contemporary cognitive models of PTSD, that internally driven negative appraisals of self played a prominent role in maintaining PTSD.

This thesis has demonstrated that the development and maintenance of PTSD following an MVA are influenced by a complex interaction of, and between, pre, peri, and post-MVA factors. These results have provided support and extend previous findings in the identification of factors associated with post-trauma psychopathology. The identification of vulnerabilities for those at risk could reduce much personal suffering, and in the process reduce the long term psychological costs of experiencing a MVA. The clinical and theoretical implications of findings as well as consideration of the direction for future research are discussed.
Chapter 1

1. Introduction

1.1 Trauma and Posttraumatic Stress Reactions

Historically the formal classification of Posttraumatic Stress Disorder (PTSD) is relatively new. However, the human response to adverse traumatic events is certainly not. Psychological distress in the face of overwhelming traumatic experiences has been documented for centuries (Daly, 1983; Jones, 2006; Turnbull 1998). Menachem (2002) suggests that possibly the earliest evidence recording PTSD symptoms were described in verse some 4,000 years ago. Recorded in cuneiform writing were the reactions to an attack(s) carried out by the Elamites and the Sumerians on the city of Ur between 2027 and 2003 B.C. Given the atrocity of the assault which ultimately destroyed the city, it is not unreasonable to assume the experience and post-trauma psychological responses conveyed in the verses (e.g., *I tremble, terror has filled me, no peace for me, dead bodies lying about*) bears some resemblance to symptoms of PSTD and reactions similar to those who have experienced the human carnage of more recent wars (see Kramer, 1969, for a detailed description of the verses).

Further historical accounts from which post-combat trauma symptoms are said to be found include Homer’s *Iliad* (Shay, 1991; Trimble, 1981), from the writings about the battle of Marathon (Greece 490 BC) as well as from accounts of early Roman and Egyptian wars (Bentley, 2005). However, it could be argued that searching historical literature descriptions for symptoms of PTSD may potentially neglect differential presenting symptoms, that is, there may be alternative explanations for symptoms (Jones et al., 2003). Similarly, etymological issues regarding changes to word meanings, the conceptualisation of words and subsequent descriptions of emotions may
not always lend themselves to clearly describe the clinical phenomena for which they have been since attributed (Jones, 2009).

Samuel Pepys’ account of the Great Fire of London occurring in 1666 is said to provide evidence of reactions that are quite similar to PTSD symptoms (Turnbull, 1998). Daly, (1983) reports that Pepys experienced a range of symptoms including; fear, irritability, anger, insomnia, mild depersonalisation, memory impairment, flashbacks and nightmares for months after the fire. Daly concluded that Pepys “has helped to establish the temporal constancy of post-traumatic stress disorder” (p. 67) through his subjective recordings.

In the mid to late 19th and early 20th century it was recognised that patterns of responses frequently developed in the aftermath of exposure to distressing traumatic events. This recognition paved the way for the creation of numerous post-trauma syndrome terminologies (Bentley, 2005; Turnbull, 1998). With the industrialised modernity of the Victorian era came a new traumatic stressor; the railway accident. The phenomenon known as Railway Spine was a term often given to the survivors of railway accidents, characterised by the appearance of an array of physical disorders in uninjured otherwise healthy accident survivors (Harrington, 2003). Arguably, with the inception, categorisation and subsequent medical investigations into railway spine by medical purveyors (particularly Erichsen and later Page), the role of psychological (non-somatic) factors were recognised as influential in the development of railway spine (Harrington, 2003; Lamprecht & Sack, 2002).
1.2 Combat Trauma

From antiquity to current times the experience of combat trauma and subsequent stress reactions has been documented. Within contemporary nomenclature various terminologies have been used to describe war syndromes of medically unexplained symptoms (Kinzie, & Goetz, 1996). Pre-dating World War I (WWI), and including the American Civil War, terms such as nostalgia neurasthenia, palpitation, soldier’s heart, irritable heart, disordered action of the heart and psychogenic rheumatism were used. Then in WWI terms included shell-shock, effort syndrome, neurocirculatory asthenia and gas hysteria (Jones, 2006). Shell-shock denoted by a dazed and disoriented state was said to be caused by neurological damage from exploding artillery bombs. However, symptoms were also observed in soldiers who had not experienced any artillery fire. Subsequently it was contended that shell-shock was associated with cowardice or malingering and that soldiers who experienced mental breakdown and collapsed with a range of (psychological) symptoms were deemed to be weak in character (Bentley, 2005; Scott, 1990). It is estimated that up to 80,000 British soldiers were diagnosed with shell-shock, approximately 306 of whom were killed by firing squad after being accused and convicted of cowardice or desertion (Babington, 1997; McFarlane, 1992). Toward the end of WWI came the realisation that psychiatric causalities (e.g., shell-shock) were not attributable to neuro-physiological (e.g., brain damage) causes, but rather by emotions with a psychological derivation.

Following World War II, terms (some with a psychodynamic emphasis) including psychoneurosis, war neurosis, non-ulcer dyspepsia, effort syndrome, old sergeant syndrome and combat exhaustion were introduced. Due to the increasing number of soldiers reported to be suffering from mental problems (according to McFarlane, 1992,
850,000 American soldiers were admitted to army hospitals suffering from psychiatric problems) it became clear that the predisposition of a weak character was an insufficient explanation and that emotional symptoms associated with war syndromes started to be viewed as a normal reaction of exposure to the extraordinary stresses of warfare (Jones, 2009; Pols, 2006).

With the previous categorisations of war trauma syndromes came a gradual shift in perceived causality from soma to psyche. However, the importance and recognition of the psychological impact of war was most evident with the Vietnam War. From August 1964 to May 1975 an estimated 3.14 million American soldiers served in the Vietnam Theater of operations (Dohrenwend, et al. 2007). It is estimated that 2.5 million soldiers experienced combat, over 400,000 were wounded and 58,000 soldiers were killed in the 11 year period (Friedman, 1981). From 1962 until 1972 more than 50,000 Australian military personal served in Vietnam with over 2,500 sustaining injury and over 500 Australian troops were killed. (O’Toole, Catts, Outram, Pierse, & Cockburn, 2009). War syndromes associated with Vietnam comprised; combat fatigue, delayed stress response syndrome and post-Vietnam syndrome. These terms acknowledged the immediate effects of trauma reactions as well as delayed and long-term responses with many veterans not developing symptoms for several months post-Vietnam. Although post-Vietnam syndrome accounted for a number of the clinical symptoms experienced by many veterans (intrusions, numbing, sleep disturbance, depressed mood, anxiety, exaggerated startle response, anhedonia, difficulty concentrating and somatic complaints) it lacked any official diagnostic recognition (Friedman, 1981). At that time the diagnostic nomenclature did not provide a specific category for war related trauma reactions as the DSM-II (APA, 1968) “Transient Situational Disturbance” did not have
any operational criteria and consequently diagnosis lacked reliability (Lamprecht, & Sack, 2002; Scott, 1990). Vietnam veterans who reported post-trauma symptoms were said to be experiencing traumatic war neurosis and diagnosed using the DSM-I (APA, 1952) with “Gross Stress Reaction”, a transient response acknowledging the potential influence of predisposing character traits (Friedman, 1981; Lamprecht & Sack; Scott, 1990; Turnbull, 1998).

Given that there was no current official diagnosis for combat veterans amidst an emerging recognition of post-Vietnam psychological (re-adjustment) problems, a new diagnostic classification was warranted. The Vietnam War provided the impetus for the formulation of a new disorder that took into account not only the psychological effects of combat (trauma created by humans) but also naturally occurring traumas (in part de-emphasising the distinction) while discarding the assumption of the imperative importance of predisposing character traits (Friedman, 1981).

With the inception of the DSM-III in 1980 came the acceptance of the formal category of Posttraumatic Stress Disorder (PTSD) as a legitimate psychiatric (anxiety based) disorder making it possible to name the effects of the overwhelming experiences on soma and psyche associated with traumatic events. Essentially this paved the way for scientific investigation into the many notions and prejudices surrounding the effects of trauma and conceptualised the varied and extraordinary symptoms associated with intense and pervasive traumatic stressors. Ignorance and scepticism which at times had previously surrounded the authenticity of symptoms often led to prejudice against many victims. However, diagnosis allowed victims (particularly veterans at that time) to finally validate their psychic distress without stigmatisation and provided a sense of
communality with other victims. Furthermore, from a medico-legal perspective, clarification and formal acknowledgement which recognised the psychosocial impact of trauma related psychopathology was crucial for victims (not only veterans) seeking financial compensation, and/or treatment as well as rehabilitation (Alexander, 1999; Foa & Meadows, 1997; Jaycox & Foa, 1998; Solomon, Laor & McFarlane, 1996; Wilson, 1994).

1.3 Posttraumatic Stress and Diagnosis: Nosologic Changes Over Time

1.3.1 DSM-I

From the first official categorisation of traumatic stress reactions several changes have occurred in both definition and diagnosis which have been influenced to a large extent by both dominant theoretical and political persuasions. Given the acknowledgement of the inadequacy of the psychiatric nomenclature to explain the psychological causalities of war, in particular WWII and at that time the Korean War (McFarlane, 1991; Turnbull, 1998), “gross stress reaction” was included in the first edition of the DSM (APA, 1952) within the category of “Transient Situational Personality Disorders”. Whilst recognising the acute impact of both war and civilian trauma, the response to severe physical or extreme emotional stress was considered transient in nature (parallel to the duration of the stressor) with chronicity of symptoms (neurotic reactions) indicating predisposing personality traits. The assumption, based on psychoanalytic theory (further explained in section 1.7.1) highlights the implied yet imperative role of pre-morbid traits, that is, underlying psychopathology when stress reactions are maintained beyond the duration of the stressor, with trauma experiences viewed as incidental (McFarlane, 1991; Turnbull, 1998; Wilson, 1994; Scott, 1990).
1.3.2 DSM-II

Some 16 years later with the DSM-II (APA, 1968) “gross stress reaction” was absent and, within the category of “Transient Situational Disturbances”, was replaced with “adjustment reaction of adult life”. This partly acknowledged the impact of war, for example “fear associated with military combat and manifested by trembling, running, and hiding” (APA, p. 49). The omission of stress reactions in the DSM-II was perplexing (Andreasen, 2004; Bloom, 1990; Wilson, 1994) and possibly attributed to political motivation (Blank, 1985; Figley, 1978) as, coincidently the DSM-II was published at the height of the Tet offensive in Vietnam.

Similar to the DSM-I, the DSM-II lacked elaborate diagnostic classifications, but rather provided brief indistinct acute symptom details, which according to Mayes and Horwitz (2005) represent broad underlying dynamic conditions that disguised underlying conflicts. A lack of operational criteria, no distinct diagnostic features, assumptions of causality (predominately psychodynamic, social or biological), inconsistent diagnosis amongst clinicians, and a general dissatisfaction with the classification system, provided the momentum for the development of a new classification system (Klerman, 1984). Additionally, as Andreasen (2004) pointed out, the Veterans Administration and military psychiatrists alike had no official diagnosis to give to Vietnam veterans’ suffering from the pervasive traumatic effects of combat.

1.3.3 DSM-III

Headed by Spitzer, a group of psychiatrists regarded as neo-Kraepelinians created a paradigm shift and changed the diagnostic orientation of the mental health community with the publication of the DSM-III (APA, 1980; Klerman, 1984; Mayes & Horwitz,
There was a shift away from presumed underlying etiology to a standardised, dichotomous categorical classification format, incorporating a multiaxial system with operational (diagnostic) criteria using descriptive psychopathology to explain (overt) symptoms and define mental disorders.

A diagnosis of PTSD in the DSM-III required a stressor which is “generally outside the range of usual human experience” (p. 238) including accidental, natural and man-made disasters. Symptoms (see Table 1) comprised three re-experiencing, three numbing of responsiveness symptoms/avoidance responses and six miscellaneous symptoms not present prior to the trauma. Furthermore, PTSD could be categorised as acute with the onset or duration of symptoms occurring within 6-months post-trauma, as chronic with symptoms lasting 6-months or more or as delayed with symptom onset occurring at least 6-months post-trauma.

Comparatively the DSM-I and DSM-II were both descriptively asymptomatic as well as being theoretically and etiologically based. However, the DSM-III sought to eliminate these explanatory foundations with a more descriptive derivation. The DSM-III provided diagnostic operational criteria emphasising the significance of the stressor criterion, and described the symptoms that define the disorder including not only acute but also chronic reactions and acknowledged the potential role of both pre and comorbid psychopathology. Whilst the DSM-III was relatively atheoretical and not aetiologically based, PTSD was an unusual exception with a presumed pathogenesis. However, the primary focus was on the stressor and its impact on the individual “…a stressor that would evoke significant symptoms of distress in almost everyone” (APA, 1980, p. 238) as opposed to previous definitions in which stress responses were viewed as transient
unless premorbid psychopathology was present in which case the etiological focus, in explaining chronicity of symptoms, was on individual dispositional factors (Brett, Spitzer & Williams, 1988; Davidson, 1992).

1.3.4 DSM-III-R

The DSM-III was revised in 1987 (DSM-III-R) with the stressor criterion conceptually defined as being “outside the range of usual human experience and that would be markedly distressing to almost anyone” (APA, 1987, p. 250). Further clarification of trauma characteristics included serious threat or harm to family or friends including threat to life or physical integrity, destruction of home or community and witnessing an accident or physical violence where severe injury or death occurs. Symptoms were grouped into 3 categories comprising, four intrusion, seven avoidance and six arousal symptoms, as displayed in Table 1. The distinctions between acute and chronic sub-types in the DSM-III were abolished. To meet diagnosis the duration of the disturbance had to be at least 1-month; symptoms fluctuating within that time frame were considered non-pathological.

One of the most important changes in the DSM-III-R was the emphasis on avoidance/numbing symptoms. In addition to increasing the number of avoidance symptoms from three to seven, the number of symptoms required for diagnosis also increased from one to three. Avoidance symptoms were grouped with numbing responsiveness as opposed to separate groups in the DSM-III, with diagnosis requiring either numbing or avoidance. A growing body of literature has provided a greater understanding of the importance of avoidance and associated clinical features. Avoidance and numbing are viewed as both a cognitive and a behavioural response to
defend against persistent and distressing intrusive re-experiencing of trauma memories (Breslau, 1990). Horowitz, Wilner, and Kaltrender (1980) reported that numbing and avoidance were the result of effortful circumvention in order to prevent distressing intrusive trauma memories. Similarly, in a study of motor vehicle accident (MVA) survivors using the DSM-III-R criteria, Steil and Ehlers (2000) found that avoidance strategies associated with reducing distressing intrusions were related to PTSD symptom severity.

A general concordance exists between contemporary theoretical models of PTSD (further explained in section 1.7) in that avoidance of distressing trauma reminders are a central factor in maintaining symptoms as it impedes emotional processing of traumatic experiences, reduces habituation to traumatic memories and interferes with integration and restructuring of dysfunctional trauma cognitions (Ehlers & Clark, 2000; Foa, Steketee & Rothbaum, 1989; Foa & Riggs, 1993; Horowitz, 1976; Jones & Barlow, 1990; van der Kolk & van der Hart, 1991).

Changes in diagnostic criteria have potential implications for the prevalence of the disorder and also in relation to classifying diagnosis, which from a medico-legal perspective can determine, for example, whether or not compensation (including war and civilian trauma as well as disasters), legal action or treatment is warranted. These implications have a direct bearing on the quality of life and wellbeing of trauma survivors.

In a study by Creamer (1990) with a treatment seeking sample who had been exposed to a multiple homicidal shooting, it was found that the difference of the numbing criteria...
between the DSM-III requiring one symptom and DSM-III-R requiring three symptoms, accounted for a 41% difference in caseness (33% and 74% respectively). Results also showed that 38% of the sample endorsed feelings of guilt which had been excluded from the DSM-III-R criteria. Another notable change in the DSM-III-R criteria was the addition of a minimum duration of disturbance of at least 1-month. In a study of female rape victims (Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992) results showed that a 30% difference of PTSD caseness existed between the diagnosis time-frame of 1-week and that of 4-weeks post-trauma (94% and 64% respectively).

The stressor criterion is foremost in meeting diagnosis and for this reason has been referred to as the ‘gatekeeper’ of PSTD (Davidson & Foa, 1991). Criticism regarding the adequacy of the stressor criterion definition has created much discussion questioning the validity of diagnosis (Breslau & Davis, 1987; Green, 1993; Horwitz, Weiss, & Marmar, 1987; Long & Elhai, 2009; March, 1993; Solomon & Canino, 1990; Ursano, 1987; Weathers & Keane, 2007). Breslau (1990) stated that the DSM-III stressor definition was ambiguous and had not been clarified in the DSM-III-R. Others have stated that the definition lacks specificity and that there is no clear information provided to determine if in fact an event is outside the range of usual experiences (O’Donohue & Elliott, 1992). Sexual assault and MVA’s for example, unfortunately are not uncommon nor are they outside the range of usual experiences; however this does not preclude the possibility of experiencing post-trauma psychopathology (Kessler, Sonnega, Bromet, Hughes, & Nelson 1995; Norris, 1992).

1.3.5 DSM-IV
Changes in the description of the stressor criterion in the DSM-IV (APA, 1994) provided a more explicit definition comprising two parts. Other changes in the DSM-IV
were the transfer of the physiological reactivity D6 criterion into the re-experiencing B5 criterion. Criterion C required both avoidance and numbing symptoms as opposed to either in the DSM-III-R. An additional criterion (F) was added to account for the specific behavioural manifestations of distress or impairment of functioning in daily life. Other changes were minor alterations to the wording of B, B1, B3, B4, B5, C1, C2, C3, C4, C7, and E, as displayed in Table 1. The descriptions of children’s symptoms in A2, B1, B2, and B3 in the DSM-IV, B1 and C4 in the DSM-III-R as well as additional criteria descriptions for B3, C6, and C7 in DSM-III-R and in the DSM-IV have not been included in Table 1.

1.4 Current Diagnostic Features

An important shift of emphasis in the diagnosis of PTSD as defined in the DSM-IV relates to the nature of the external stressor (the traumatic event) and subsequent symptomatic reactions. Exposure to a traumatic event must involve: (1) witnessing, experiencing or being confronted with an event involving actual or threatened serious bodily injury or death, “or threat to physical integrity of self or others”, and be accompanied by (2) an emotional response to the event involving “intense fear, helplessness, or horror” (APA, 1994, pp. 427-428). The revised definition of the stressor criterion places greater emphasis upon the subjective experience of the traumatic event which in part de-emphasises the objective features.
<table>
<thead>
<tr>
<th>DSM-III</th>
<th>DSM-III-R</th>
<th>DSM-IV</th>
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<tbody>
<tr>
<td><strong>A.</strong> Existence of a recognizable stressor that would evoke significant symptoms of distress in almost everyone</td>
<td><strong>A.</strong> The person has experienced an event outside the range of usual human experience and that would be markedly distressing to almost anyone</td>
<td><strong>A.</strong> The person has been exposed to an event in which both of the following were present:  1. The person experienced, witnessed or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others  2. The person's response involved intense fear, helplessness or horror</td>
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<tr>
<td><strong>B.</strong> Re-experiencing of the trauma as evidenced by at least one of the following</td>
<td><strong>B.</strong> Persistently re-experienced the traumatic event in at least one of the following ways</td>
<td><strong>B.</strong> The traumatic event is persistently re-experienced in one (or more) of the following ways</td>
</tr>
<tr>
<td>1. Recurrent and intrusive recollections</td>
<td>1. Recurrent and intrusive disturbing recollections</td>
<td>1. Recurrent and intrusive distressing recollections of the event, including images, thoughts or perceptions</td>
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<tr>
<td>2. Recurrent dreams of the event</td>
<td>2. Recurrent distressing dreams of the event</td>
<td>2. Recurrent distressing dreams of the event</td>
</tr>
<tr>
<td>3. Sudden action or feeling as if the traumatic event were reoccurring because of an association with an environmental or ideational stimulus</td>
<td></td>
<td>3. Acting or feeling as if the traumatic event were recurring</td>
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<tr>
<td></td>
<td></td>
<td>4. Intense psychological distress at exposure to events that symbolize or resemble an aspect of the traumatic event</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event</td>
</tr>
<tr>
<td><strong>C.</strong> Numbing of responsiveness to or reduced involvement with the external world, beginning some time after the trauma, as shown by at least one of the following</td>
<td><strong>C.</strong> Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma) as indicated by at least one of the following:  1. Efforts to avoid thoughts or feelings associated with the trauma  2. Efforts to avoid activities or situations that arouse recollections of the trauma</td>
<td><strong>C.</strong> Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma) as indicated by three (or more) of the following:  1. Efforts to avoid thoughts, feelings or conversations associated with the trauma  2. Efforts to avoid activities, places or people that arouse recollections of the trauma</td>
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</table>
**PTSD Diagnostic Criteria Comparisons (continued)**

<table>
<thead>
<tr>
<th>DSM-III</th>
<th>DSM-III-R</th>
<th>DSM-IV</th>
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<tr>
<td><strong>3. Constricted affect</strong></td>
<td>3. Inability to recall an important aspect of the trauma (psychogenic amnesia)</td>
<td>3. Inability to recall an important aspect of the trauma</td>
</tr>
<tr>
<td></td>
<td>4. Markedly diminished interest in significant activities</td>
<td>4. Markedly diminished interest or participation in significant activities</td>
</tr>
<tr>
<td></td>
<td>5. Feeling of detachment or estrangement from others</td>
<td>5. Feeling of detachment or estrangement from others</td>
</tr>
<tr>
<td></td>
<td>6. Restricted range of affect</td>
<td>6. Restricted range of affect</td>
</tr>
<tr>
<td></td>
<td>7. Sense of a foreshortened future</td>
<td>7. Sense of a foreshortened future</td>
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</table>

**D. At least two of the following symptoms that were not present before the trauma**

<table>
<thead>
<tr>
<th>DSM-III</th>
<th>DSM-III-R</th>
<th>DSM-IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Hyperalertness or exaggerated startle response</td>
<td>1. Difficulty falling or staying asleep</td>
<td>1. Difficulty falling or staying asleep</td>
</tr>
<tr>
<td>2. Sleep disturbance</td>
<td>2. Irritability or outbursts of anger</td>
<td>2. Irritability or outbursts of anger</td>
</tr>
<tr>
<td>3. Guilt about surviving or about behaviour required for survival</td>
<td>3. Difficulty concentrating</td>
<td>3. Difficulty concentrating</td>
</tr>
<tr>
<td>5. Avoidance of activities that arouse recollection of the traumatic event</td>
<td>5. Exaggerated startle response</td>
<td>5. Exaggerated startle response</td>
</tr>
<tr>
<td>6. Intensification of symptoms by exposure to events that symbolize or resemble the traumatic event</td>
<td>6. Physiologic reactivity upon exposure to events that symbolize or resemble an aspect of the traumatic event</td>
<td><strong>E. Duration of the disturbance (symptoms in B, C and D) of at least 1 month</strong></td>
</tr>
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</table>

**E. Duration of the disturbance (symptoms in Criteria B, C and D) is more than 1 month**

**F. The disturbance causes clinically significant distress or impairment in social, occupational or other important areas of functioning**
1.4.1 Symptom clusters

Additionally, the three symptom clusters of persistent re-experiencing, avoidance, and increased arousal must also be met for diagnostic eligibility. Re-experiencing the traumatic event can occur in recurrent intrusive thoughts, nightmares, emotional distress or physiological arousal in response to internal or external cues that act as reminders of the trauma, or a sense of re-living the trauma through dissociative flashbacks or hallucinations. Avoidance symptoms involve effortful circumvention of thoughts, feelings, and behaviours, such as detachment or distance from other people, avoidance of thinking about the trauma, emotional numbing, a sense of foreshortened future, diminished interest in activities, psychogenic amnesia, or avoidance of reminders of the event. Increased arousal symptoms consist of heightened physiologic responses resulting in concentration problems, sleep disturbance, irritability, outbursts of anger, exaggerated startle response, and hypervigilance (APA, 1994).

If the symptom criteria are met and persist for more than 1-month, and cause either considerable distress or impairment in daily living, a diagnosis of PTSD may then be appropriate. The inclusion of the F criterion was an important addition in the DSM-IV as it determines the subjective impact of the disorder upon daily life. As previously noted, changes in diagnostic criteria can alter diagnostic rates. Solomon and Horesh (2007) found with Lebanon War veterans that the DSM-IV criteria provided the lowest diagnostic rates in comparison to the DSM-III and DSM-III-R, noting the important role of the F criterion.

When symptom duration is less than 3-months, the diagnosis is specified as Acute PTSD. If symptoms persist for more than 3-months the diagnosis is specified as
Chronic, and when the onset of symptoms are not evidenced until at least 6-months post-trauma, the diagnosis of PTSD with Delayed Onset is applicable. Symptoms that develop within 1-month of trauma exposure may be defined as Acute Stress Disorder (ASD). This disorder was included in the nosology to account for the acute phase of trauma symptomatology, potentially allowing for early identification and subsequent treatment intervention, hence, potentially circumventing the development of PTSD, (APA, 1994; Marshall, Spitzer, Liebowitz, 1999). When a text-revision of the DSM-IV was published in 2000, the diagnostic criteria remained the same. However, additional and updated information was provided including vulnerability, prevalence, course, and comorbidity.

1.4.2 Malingering and Secondary Gain

Though the term ‘malingering’ is not new to the DSM, malingering was first listed (under differential diagnosis) for PSTD in the DSM-IV stating “malingering should be ruled out in those situations in which financial remuneration, benefit eligibility, and forensic determinants play a role” (p. 427). Compensation for trauma induced psychological injury dates back to late 1870’s (re; “compensation neurosis” - Railway Spine) and continues to date (Jones & Wessely, 2007; Sterling, Hendrikz, & Kenardy, 2010). It is beyond the scope of this thesis to provide a thorough review of PTSD malingering, although it is certainly an issue clinicians and researchers alike need to be mindful of, which if left unchecked, given the likelihood of trauma exposure and society’s litigious nature, could lead to inflated rates of PTSD (Hall & Hall, 2006; Rosen, 2006). However, it is interesting to note that a number of studies have found that financial compensation once granted had little to no effect on remission of symptoms or functionality; in fact it could be argued that the often drawn-out legal
process may impede recovery, acting as a constant reminder of the event (Horne, 1993; 1995; Jones, Palmer, & Wessely, 2002; Mendelson, 1982; Pitman, Sparr, Saunders, & McFarlane, 1996; Tarsh & Royston, 1985; Thompson, 1965). Similarly, some studies on MVA survivors have found that litigation did not affect assessment of symptoms nor treatment (Blanchard & Hickling, 2004), with no differences in symptom presentation between MVA litigants and non-litigants (Blanchard, Hickling et al., 1998; Bryant & Harvey, 2003a; Mayou, 1995; Mayou, Bryant, & Duthie, 1993). However, Blanchard et al. (1996) reported that litigation predicted PTSD. Similarly, Ehlers, Mayou, and Bryant (1998) found that a range of variables including litigation predicted PTSD symptom severity at 3-months and at 1-year post-MVA. Although they suggested that litigation maintained symptoms, they noted that the relationship (causality) remains unclear. More recently Sterling et al. (2010) reported that there was a detrimental psychological effect associated with lodging a compensation claim following whiplash injury in a sample of Australian MVA survivors, reiterating earlier findings that the stressful and often protracted legal process of seeking compensation can exacerbate symptoms and impede natural recovery. O’Donnell, Creamer, McFarlane, Silove, and Bryant (2010) found that MVA survivors seeking compensation had significantly higher levels of anxiety than those not seeking compensation. The difference they noted appeared to be due to the stressful compensation process. Their results did not support earlier findings that access to health compensation is associated with poor recovery post-injury. However, they noted that the relationship between compensation and health outcomes is highly complex.
1.5 ICD-10

The World Health Organisation’s tenth revision of the International Classification of Mental and Behavioural Disorders (ICD-10) provides an alternate psychiatric classification system. Both the DSM-IV and the ICD-10 have a similar underlying concept of PTSD; however there are some differences between their diagnostic criteria. For example, the ICD-10 does not include the F criterion of impairment and does not require numbing as an essential feature, with only one avoidance symptom necessary to meet diagnostic criteria. Research has shown considerable differences in the rates of diagnoses due to differing criteria with the stricter DSM-IV providing approximately 50% lower rates (Andrews, Henderson, & Hall, 2001; Peters, Slade, & Andrews, 1999; Rosenman, 2002; Rosner & Powell, 2007). The ICD-10 requires greater judgement regarding the clinical relevance as well as the interpretation of the traumatic event whereas the DSM-IV provides a strict set of guidelines and rules that govern diagnosis in addition to clinical judgement (Lee & Young, 2001). Reference to PTSD literature throughout this thesis is predominately based on the DSM-IV criteria.

1.6 Traumatic Stressors

Although there is no universal definition of what constitutes a traumatic event, traumas associated with PTSD aetiology are often described as frightening, unexpected (yet not uncommon), powerful, and uncontrollable (Janoff-Bulman, 1992; Schnurr & Friedman, 1997; Turnbull, 1999). In addition, events where exposure is close (proximally) and prolonged, involving multiple deaths or mutilation, especially where children are involved, have been found to be the most traumatic (Turnbull, 1999; Ursano, Fullerton & Norwood, 1995). However, the experience and degree of emotional response to traumatic events varies subjectively. Moreover, natural disasters, life-threatening illness
and man-made calamities including events such as war, may have a slow onset and not always occur suddenly or unexpectedly, though potentially causing multiple affective, behavioural and cognitive reactions (Van Etten & Taylor, 1998; Turnbull, 1999). The risk of developing PTSD can vary dependent upon the trauma type. Breslau et al. (1998) examined the risk of PTSD in specific trauma sub-groups, and found considerable variation in the rates of PTSD between trauma types, such as sudden unexpected death of a loved one accounted for 31% of PTSD cases compared to a serious car accident at 2%. Norris’s (1992) study on the frequency and impact of traumatic events reported a lifetime MVA exposure prevalence rate of 23.4% and 2.6% in the past year. Her results showed that 11.5 % of MVA survivors developed PTSD, and she concluded that when frequency and severity are considered together, MVA’s are perhaps the most significant type of traumatic event (of the 10 examined). A study by Breslau et al. (1991) indicated that approximately 10% of the sample had experienced an MVA, 12% of whom developed PTSD. In the United States it is estimated (on average) that within a person’s driving career they will experience three MVA’s (Hickling & Blanchard, 1992), and although there is no comparative Australian data, it is not unreasonable to assume a similar rate with Australian drivers.

1.7 PTSD Epidemiology

According to Norris et al. (2002), on average somewhere around the world each day at least one disaster occurs. These include accidents (transportation, industrial, and nuclear), shooting sprees, floods, earthquakes, and hurricanes, as well as terrorist attacks. Not surprisingly individual exposure to traumatic events unfortunately is quite high, with lifetime estimates in American community samples ranging from 43% to 92% for males and 37% to 87 % for females (Breslau, Davis, Andreski, & Peterson,
1991; Breslau et al., 1998). Similarly, Kessler, Sonnega, Bromet, Hughes, and Nelson (1995) reported that 60% of males and 50% of females had experienced a traumatic event at some point in their lives, with higher proportion of males experiencing a life-threatening accident (25% of males and 14% of females), experiencing a natural disaster, fire or flood (19% of males and 15% of females), and witnessing someone being killed or badly injured (36% of males and 15% of females). Results from the Australian national survey of mental health and well-being found that the lifetime trauma exposure prevalence rate was 57.4%, with 65% of males and 50.9% of females reporting trauma exposure at some point in their lives (Rosenman, 2002). Overall, more males experience physical attack, combat experience, being threatened with a weapon, being kidnapped or held captive, whereas significantly more women reported experiencing rape, sexual molestation, childhood neglect and physical abuse (Breslau et al., 1998; Kessler et al., 1995).

Breslau et al. (1991) reported lifetime PTSD prevalence rates of 6% for males and 11.3% for females. Similarly, Kessler et al. (1995) found lifetime prevalence estimates of 5% for males and 10.4% for females, with an overall PTSD prevalence rate of 7.8%. In terms of chronicity of the disorder, Kessler et al. reported that approximately 66% of participants who developed PTSD still had the condition more than one year after the trauma and approximately 33% experienced symptoms three years post-trauma. Results from the Australian National Survey of Mental Health and Well-being found that the 12-month prevalence rate of PTSD was 1.5% (using unweighted multilevel analysis, cf., Creamer, Burgess & McFarlane, 2001). This 12-month rate is considerably lower than that found by Kessler et al. (1999), which was 3.9%, and is more aligned with the results of Stein, Walker, Hazen, and Forde (1997) who found a current PTSD rate of
1.2% for males and 2.7% rate for females in a Canadian community sample, and with the results of Perkonigg, Kessler, Storz, and Wittchen (2000) who found a 1% current prevalence rate for males and 2.2% rate for females within a German community sample. When they attempted to explain the discrepancy between the Australian and United States 12-month PTSD prevalence rate, given similar frequencies of trauma exposure, Creamer et al. noted possible cross-cultural differences in resilience to stress, and speculated that an increased rate in pre-existing psychopathology in the American sample increases their vulnerability. They also noted that differing assessments and interpretations of the F criterion may explain differences in prevalence rates (as previously discussed). Furthermore, prevalence rate estimates are also influenced by the diagnostic criteria used, response rate, country, sample characteristics and age of participants, as well as the use of different methodologies (Somers, Goldner, Waraich, & Hsu, 2006).

Notwithstanding the comparative methodological confines, it is evident that the chance of experiencing at least one traumatic event within a lifetime is relatively high and the subsequent risk of development of PTSD and other psychological disorders by comparison is low. Overall, males are more likely to experience trauma but, following trauma exposure, females are more likely to develop PTSD. To date explanations of female vulnerability are inconsistent and remain unclear. Previous explanations such as type of traumatic event, prior trauma experiences, pre-existing psychopathology and gender bias in reporting, according to Breslau (2009), can be ruled out, hence warranting the need for further examination as to why females are at greater risk of developing PTSD.
Research on combat samples has found higher prevalence rates compared to community samples. Results from the national Vietnam veterans’ readjustment study reported a PTSD incidence rate of 15.2% and a lifetime prevalence rate of 30.9%, (Kulka et al., 1990). Proportionally similar rates were reported for Australian Vietnam veterans with an incidence rate of 11.6% and a lifetime prevalence of 20.9%, (O’Toole et al., 1996).

1.8 Comorbidity

PTSD commonly occurs with at least one other disorder. Co-occurring disorders can exacerbate PTSD symptoms, add considerably to the chronicity of symptoms impacting on quality of life and can have important implications in regard to treatment decisions (Masthoff, Trompenaars, Van Heck, Hodiamont, & de Vires, 2006; McFarlane, 2004). Research involving a range of traumas, including natural disasters (Carr, Lewin, Webster, & Kenardy, 1997; McFarlane & Papay, 1992; Chan, Gao, & Griffiths, 2009) combat veterans (Green, 1994; O’Toole, Catts, Outram, Pierse, & Cockburn, 2009; Richardson, Frueh, & Acierno 2010), refugees (Carlson & Hogan, 1991: Silove, Momartin, Marnane, Steel, & Manicavasagar, 2010; Weine et al., 1998) and MVA’s (Blanchard, Buckley, Hickling, & Taylor, 1998; Jenewein et al., 2009; Matsuoka et al., 2008), has reported high rates of PTSD comorbidity.

Kessler et al. (1995) found that approximately 80% of participants with chronic PTSD had at least one other psychiatric diagnosis. For males and females the most common comorbid diagnoses were affective, anxiety, and substance abuse disorders. Perkonigg et al. (2000) reported that 87.5% of participants with PTSD had one additional diagnosis and 77.5% had two or more disorders occurring simultaneously or secondary to PTSD. Similar comorbid results were found within an Australian community sample, with 12-
month prevalence rates showing that 85.2% of males and 79.7% of females with PTSD met the criteria for one other Axis-I disorder and approximately 60% of males and 50% of females had two or more disorders. Major depression co-occurred most frequently, with 51.6% of males and 65.1% of females, followed by Generalised Anxiety Disorder, with 40.2% of males and 22% of females. Substance use disorders were also found to be comorbid though more so for males, with 37.2% reporting alcohol and 22.6% drug abuse/dependence compared to 12.4% and 15% (respectively) for females (Creamer et al., 2001).

1.8.1 Comorbid Aetiology

In order to explain the consistently high rate of comorbidity, four etiological hypotheses have been developed. The first proposes that pre-existing psychiatric disorders constitute a vulnerability factor for the development of PTSD either through an increased risk of trauma exposure or increased susceptibility to the PTSD-inducing effects of trauma. Secondly, PTSD is a causal risk factor for depression and anxiety which are independent and occur secondary to PTSD. The third proposes that shared risk factors (e.g., traumatic event, genetic vulnerability and personality traits, such as neuroticism lead to the co-occurrence of PTSD, depression and other anxiety). The fourth hypothesis suggests that comorbidity is a diagnostic artefact due to symptom overlap (Breslau, 2002; Solomon & Bleich, 1998). For example, restricted range of affect and emotional detachment in PTSD may be confused with psychomotor retardation and depressed mood in major depression. Also both disorders share symptoms such as poor concentration, sleep difficulties and diminished interest/anhedonia (Schnurr, Friedman, & Bernardy, 2002). Empirical evidence has supported each of these perspectives, though conversely each has been refuted,
providing somewhat contradictory conclusions. However, more recently Wittmann, Moergeli, Martin-Soelch, Znoj, and Schnyder (2008) used structural equation modelling to compare comorbid models, and found that PTSD was the strongest single predictor of comorbidity. They concluded that an integrative model in which PTSD and satisfaction with health provided the strongest prediction, explaining 82% of the variance. Similarly, results from a 20-year longitudinal study of war veterans which examined comorbidity found that PTSD was the primary disorder following trauma exposure with comorbid depression and anxiety occurring later (Ginzburg, Ein-Dor, & Solomon, 2010). These results support the hypothesis that comorbidity occurs subsequent to PTSD. However, this does not preclude the influence of pre-existing psychopathology and comorbid causality interpretations may need to consider trauma specific mechanisms, such as the influence of injury and disability experienced by MVA survivors in the development and maintenance of PTSD and depression.

1.9 Theoretical Models of PSTD

1.9.1 Psychodynamic

Early theoretical accounts of the etiology of trauma reactions date back to the late 19th century. The seminal contribution of Sigmund Freud and his conceptualisation of traumatic neurosis, as noted by Wilson (1994), dominated psychiatry from 1895 until towards the end of the Vietnam War. Although it is beyond the scope of this thesis to provide a thorough review, it would be remiss not to provide a brief account of Freud’s influence given his prominent role in the development of psychiatry and his theoretical account of trauma reactions, which has since both influenced and generated research. Kinzie and Goetz (1996) explain that Freud was initially influenced by Janet. He elaborated on Janets’ theoretical account of the aetiological role of dissociation in
neurosis (see section 2.2.2), but later abandoned the dissociation explanation. Freud’s traumatic theory of neurosis suggested that childhood repressed conflicts resultant from sexual trauma occurred in all cases of hysteria as subsequent events triggered the childhood trauma. Therefore, the focus was on individual vulnerability and not on the recent traumatic event. Freud’s emphasis on sexuality as a cause of neurosis was not well received and he revised his seduction theory. However, he still maintained that conflict between the libido and sexual repression was the cause of hysteria. In explaining traumatic neurosis of war, Freud believed that the terrifying event broke the protective shell of the ego causing the ego to escape into neurosis and that neurosis was a conflict as the ego is defending itself against the horror of warfare causing disequilibrium within the ego (Kinzie & Goetz; Wilson, 1994). Whilst influential in shaping earlier official formulations of traumatic stress reactions, the psychodynamic emphasis on pre-trauma personality as the determining factor in the development of PTSD has since been refuted and, though current literature acknowledges the role of pre-morbid status, it is generally accepted that there are multiple pathways in the development of PTSD.

1.9.2 Cognitive/Psychodynamic

Horowitz’s (1976) cognitive theory of information processing, which incorporated a psychodynamic underpinning, has been very influential to such an extent that it is said to have formed the cornerstone for the diagnostic criteria in DSM-III (Peterson, Prout, & Schwarz, 1991). Horowitz proposed that when the initial reaction to trauma is experienced the response is one of ‘outcry’ in which memories, thoughts and images of the trauma are structurally unorganised. The interpretation and perception of incoming information is guided by core assumptions and beliefs (i.e., schemas). Processing
trauma information involves congruency between pre-existing and ‘new’ trauma schemata. This process is accomplished via two processes based on Piaget’s (1971) model of cognitive development; assimilation and accommodation. Tension arises when traumata-related memories and information do not match “pre-existing inner schematisations” and information then remains unprocessed (Horowitz, 1986a, p. 246). Defence mechanisms such as denial and numbing are employed to repress and keep trauma information unconscious. However, the psychological need to process and reconcile new with existing information causes trauma memories to break into consciousness in the form of flashbacks, nightmares and intrusions. These two opposing processes create a defence by suppressing the trauma information and reconciling information via intrusions, causes an oscillation between these processes. Gradually the traumatic information is assimilated into schematic representations as it is cleared from active memory. Long term memory structures representing the self and future are adjusted so as to be consistent with the new information at which time processing of the trauma is said to be complete. Persistent posttraumatic reactions occur if trauma information remains in active memory causing intrusions and avoidance as processing of trauma information is not integrated and remains incomplete (Horowitz, 1986b, 1997).

Horowitz’s model has made a significant contribution to the understanding of trauma process and was one of the first models to highlight the cognitive impact of trauma on beliefs of self, the world and future, as well as recognising the important role of cognitive change in the recovery process. However, as Brewin and Holmes (2003) have pointed out, the theory does not distinguish between flashbacks and ordinary trauma memories nor does it address the role of peri-traumatic reactions. Similarly, Dalgleish
(2004) concluded that although theoretically many core aspects are covered, a more thorough account of trauma related emotions is warranted as is a more detailed account of the actual process involved in schemata change. Additionally, Horowitz assumed that trauma related schemata are incongruent with pre-existing schemata. However, this may not always be the case as epidemiological studies indicate that trauma exposure is relatively common. In the case of individuals who have experienced prior trauma, new trauma information would be congruent with pre-existing schemas, which according to Horowitz would hasten recovery. However, research has shown that this is not the case as experiencing prior trauma and particularly prior PTSD increases the risk for subsequent PTSD (Breslau, 2009; Breslau et al., 1999; Green, et al., 2000).

1.9.3 Behavioural Theory

Mowrer’s (1960) two-factor theory, comprising classical conditioning and instrumental learning (often referred to as operant conditioning), provides a theoretical framework explaining the etiology of PTSD and maintenance of symptoms. It suggests that fear in traumatised individuals is acquired through classical conditioning as neutral trauma stimuli (CS) via associative learning (generalisation) and higher-order conditioning are associated with trauma related stimuli (UCS) resulting in both neutral and trauma stimuli eliciting a fear response (CR). Avoidance behaviours are used to reduce trauma anxiety and fear which in turn reinforcers those behaviours (negative reinforcement). This cyclic process maintains fear and prevents extinction from occurring as avoidance prohibits the realisation that the association between the CS and the UCS no longer exists. Research findings have provided empirical support for the model (Foa, Steketee, & Rothbaum, 1989; Dollinger, O’Donnell, & Staley, 1984; Keane, Zimering, & Caddell, 1985; Kilpatrick, Veronen, & Resick, 1982), with results showing the role of
fear, avoidance and stimulus generalisation in explaining the development and
maintenance of symptoms. Criticisms of the model are that it only accounts for a sub-set
of PTSD symptoms (e.g., hyperarousal and avoidance). Deficiencies regarding the role
of emotions other than fear and the role of cognitions in appraisals as well as in re-
experiencing symptoms have also been highlighted.

1.9.4 Emotional Processing Model

Early models which attempted to explain the aetiology of PTSD primarily focused on
the role of single factors, such as conditioned responses and cognitive processes. This
approach places limitations on the scope of investigations by narrowing the parameters
of inquiry.

The emotional processing model of PTSD (Foa & Riggs, 1993; Foa & Rothbaum, 1998)
is a contemporary cognitive theory which builds upon the earlier work of information-
processing theories (see Creamer, Burgess, & Pattison, 1992; Foa, Steketee, &
Rothbaum, 1989). Based on her earlier fear network model of PTSD, which adopted
Lang’s (1979) cognitive fear structure, Foa and colleagues; model provides a
comprehensive framework designed to explain the acquisition, maintenance, and
treatment of PTSD. It integrates aspects of Mowrer’s two-factor learning theory,
Horowitz’s cognitive model, and personality theories, within an information-processing
framework (Jaycox & Foa, 1998). It postulates that psychopathology emerges after the
event when the frequency and intensity of emotionally re-living the experience fails to
decrease. Consequently, the development of PTSD is influenced by a cognitive fear
structure, and interpretation of information related to the feared stimulus, its meaning,
and subsequent behavioural and cognitive responses. The ensuing pathological
cognitions, that the world is dangerous and uncontrollable and that the self is 
incompetent, are pervasive as well as interactional, and replace previous functional 
benign and competent beliefs (cf., Janoff-Bulman’s shattered assumptions, 1992).

Furthermore, the model contends that trauma-related cues often trigger intrusive 
recollections, and when these intrusions are distressing, attempts to avoid or suppress 
them lead to avoidance strategies. Avoidance cognitions and behaviours play an integral 
role in maintaining PTSD symptoms, as they impede the habituation of traumatic 
memories and prevent adequate emotional processing of the traumatic experience; they 
interfere with the restructuring and integration of adaptive trauma-related cognitions 
(Foa & Riggs, 1993; Foa, Steketee & Rothbaum, 1989; Jaycox & Foa, 1998; Steil & 
Ehlers, 2000).

Treatment interventions based on the model work on the premise that PTSD develops 
from inadequate processing of traumatic memories whilst acknowledging the role of 
dissociative reactions impeding emotional processing as trauma memories are 
fragmented and disorganised. Treatment requires a reduction in the avoidance of, and 
emotional engagement with, feared traumatic memories (fear activation via exposure). 
Treatment also involves the correction of erroneous associations and the provision of 
information that is incompatible with the pathological elements of existing fear 
structures/schemas (Foa & Meadows, 1997; Foa & Riggs, 1995; Harvey & Bryant, 
1999a; Jaycox & Foa, 1998; Solomon, 1997; van der Kolk, McFarlane & van der Hart, 
1996). The principle behind the recovery process is similar in nature to that described 
by Horowitz (1976) which requires the activation and integration/modification of 
trauma memories.
It is well established that exposure based treatments are effective psychological interventions in ameliorating symptoms of PTSD. This provides support for the exposure component of the emotional processing theory (Boudewyns & Hyer, 1990; Cooper & Clum, 1989; Foa & Meadows, 1997; Foa, Rothbaum, Riggs, & Murdock, 1991; Jaycox & Foa, 1996; Keane, Fairbank, Caddell, & Zimering, 1989; Keane & Kaloupek, 1996; Resick, Jordan, Girelli, Hutter, & Marhoefer-Dvorak, 1988; Solomon, Gerrity, & Muff, 1992; van der Kolk, McFarlane & van der Hart, 1996; van Minnen & Foa, 2006). More specifically, findings have also supported the notion that the success of exposure treatment in promoting habituation is achieved through fear activation (Foa, Riggs, Massie, & Yarczower, 1995; Jaycox, Foa, & Morral, 1998; Pitman et al., 1996; van Minnen & Hagenaars, 2002).

Further support for the model is provided by evidence of a relationship between fragmentation and disorganisation of trauma memories and dissociative reactions (Engelhard, van den Hout, Kindt, Arntz, & Schouten, 2003; Hagenaars, van Minnen, Hoogduin, & Verbraak, 2009; Halligan, Michael, Clark, & Ehlers, 2003; Harvey & Bryant, 1999a; Murray, Ehlers, & Mayou, 2002). Van Minnen, Wessel, Dijkstra and Roelofs (2002) reported that a decrease in disorganised thoughts was related to a decrease in symptoms. However, Halligan et al. (2003) found no significant relation between changes in memory disorganisation and PTSD symptoms. O’Kearney and Perrott (2006) suggested that findings are contradictory because no valid instruments to assess disorganisation have been developed and different research methodologies have been used. Hagenaars et al. (2009) also concluded that the distinction between disorganisation of voluntary and involuntary trauma memories is not always made and

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1 Introducing correct information about self, world and others as well as the separation of CS and UCS are also important in modifying the fear structure.
perhaps future research may benefit from comparing both memory types when examining disorganisation of trauma memories.

Brewin and Holmes (2003) and Dalgleish (2004) stated that the emotional processing model offers a great deal of explanatory power which provides an extremely comprehensive account of the development, maintenance and, in particular, the treatment of PTSD. For example, a major contribution to PTSD treatment has been the use of in vivo and imaginal exposure interventions. The model is also empirically supported, particularly in relation to therapeutic intervention and recovery processes via exposure treatment. However, the model is not without limitation. For example detailed elaboration on how cognitive change occurs without memory activation and fear habituation is needed. Furthermore, emotions often associated with PTSD such as guilt, shame, anger, and sadness may not respond to exposure treatment in the same way as fear. Brewin and Holmes suggested that theoretical differentiation is required to explain automatic changes in trauma memory between exposure, and deliberate changes occurring through cognitive reappraisal.

1.9.5 Ehlers and Clark Cognitive Model

A more recent cognitive model, proposed by Ehlers and Clark (2000), partly based upon previous theoretical accounts of PTSD, was developed to explain the maintenance of symptoms whilst also providing a cognitive-behavioural treatment framework. Contrary to other anxiety disorders in which anxiety may be anticipatory or situational (i.e., occurring prior to or during an event), the anxiety associated with PTSD occurs after an event. Ehlers and Clark’s model sought to explain how a traumatic event that occurred in the past produces current and future anxiety (threat), months or years post-trauma.
They proposed that persistent PTSD develops from processing the trauma in such a way that it leads to a sense of serious current threat. Viewed as a vital component of the model (see Figure 1) current threat arises from 1) excessively negative idiosyncratic trauma appraisals and/or sequelae and 2) a disturbance of autobiographical memory.

Negative appraisals contribute to the perception of current threat as either external (the world is unsafe) or internal (self beliefs and future). Negative appraisals about traumatic events may lead individuals to overgeneralise, that is, they may overestimate the danger associated with usual activities such as driving to work each day (“the roads are extremely dangerous”) or they may exaggerate the probability of experiencing another traumatic event.

Appraisals of trauma associated feelings or actions can also potentiate the perception of current threat. For example, a driver involved in a fatal MVA may experience feelings of guilt, anger, sadness, and shame and question their ability to avoid future accidents.

Appraisals of trauma sequelae comprise negative idiosyncratic appraisals including initial PTSD symptoms (e.g., emotional numbing; “I’ll never be able to relate to people again” p. 322), interpretation of others’ reactions (e.g., “They think I am too weak to cope on my own” p. 322), and negative appraisals of the consequences of the trauma in areas such as daily functioning (e.g., disfigurement or disability sustained in an MVA could be perceived as a permanent negative change “My body is ruined” p. 322).

The role of negative appraisals is twofold. Firstly they produce a range of negative emotions, predominately fear as well as anger, guilt, shame, and sadness (see Beck, 1976; Scherer, 1999). Secondly, they motivate maladaptive cognitive and behavioural responses (e.g., avoidance removes the aversive stimuli) which in the short term can ameliorate perceived threat. However, in the long term there is a paradoxical effect whereby cognitive and behavioural responses maintain PTSD as they prevent elaboration of both trauma memories and negative appraisals of the trauma and its sequelae. This occurs through several processes including thought suppression, avoidance of trauma reminders, avoiding thinking about the trauma, the use of alcohol and/or medication, rumination, dissociation, a reduction or abandonment of previous social based activities, and the use of safety behaviours. For example, having been involved in an MVA whilst it was raining and subsequently taking excessive precautions and adopting the safety behaviour of not driving in the rain in order to minimise the possibility of having another MVA. The fear of experiencing another
accident is maintained (hypervigilance behaviours to avoid threat), thus preventing reappraisal.

The model also proposes that negative appraisals can be influenced by prior negative experiences and beliefs as well as thought processes during the trauma. To illustrate this, Ehlers and Clark (2000) provided the example of “mental defeat”, a peritraumatic thought process in which a person perceives the loss of all psychological autonomy, a dehumanising experience and feeling of complete and utter defeat. The response, highlighting lack of control over one’s fate, increases the likelihood of negative appraisals in which the person may perceive themselves as being unable to cope and unworthy. Ehlers et al. (1998) explained that mental defeat impedes the recovery process as it prevents the trauma from being perceived as “a single, time-limited, past event which does not necessarily have global implications for oneself or one's future” (p. 466), and thus maintains a sense of impending threat.

The theory further indicates that a disturbance of autobiographical memory also leads to a sense of current threat. Memories of the traumatic event are said to be poorly elaborated and poorly integrated contextually in time and place with existing autobiographical memory. Intentional recall is disjointed and fragmented, with memory of certain aspects of the event missing and with no specific temporal order. There may also be a lack of connection between recent and existing information and a poor inhibition of triggering unintentional trauma memories by physically similar cues. Strong trauma related stimulus-stimulus and stimulus-response associations increase the likelihood of triggering trauma memories, emotional responses and distressing involuntary re-experiencing. For example, after experiencing a severe MVA, the sole
survivor was comforted by a woman whilst waiting for the ambulance to arrive. For several months the MVA survivor noticed that on occasion he became quite distressed for what appeared to be no apparent reason. Over time he came to the realisation that his intrusions were triggered when he detected a particular (though common) woman’s fragrance. The woman who comforted him at the time of the accident was wearing perfume and it was the association with that fragrance that triggered his distressing intrusions.

Given that these associations are cue-driven and unintentional, a lack of awareness can arise where associated emotional causes remain unknown (affect without recollection). This also impedes the realisation that exposure to trauma triggers pose no danger. Additionally it is proposed that strong perceptual priming for temporally associated trauma stimuli (poor stimulus discrimination) triggers re-experiencing symptoms. Ehlers and Clark (2000) provide the example of an MVA survivor who had been involved in an accident at night who noticed a patch of bright sunlight on his lawn which subsequently triggered vivid intrusions of oncoming headlights.

The nature of the trauma memory is said to be influenced by the way peri-traumatic information is processed at encoding. The model draws a clear distinction between conceptual and data driven processing and subsequent influence on trauma memory. It is proposed that data driven processing (confusion and focus on overwhelming sensory impressions) results in a trauma memory that is difficult to retrieve intentionally and leads to strong perceptual priming. Conceptual processing on the other hand involves processing the meaning of the situation in an organised and contextual way. This process facilitates the integration of trauma memories with existing autographical
memory. Unorganised trauma memories may also result from a lack of self-referent processing (inability to encode new self-related information and other autobiographical information), peritraumatic dissociation (memory fragmentation), and emotional numbing (memory formation). The authors note that aspects of dissociation may in fact overlap with cognitive processing and a lack of self-referential perspective when encoding the trauma. These later factors are said to predict the development of re-experiencing symptoms and persistent PTSD (Ehlers & Clark, 2000; Evans, Ehlers, Mezey, & Clark, 2007).

Pre-trauma factors that may influence cognitive processing, trauma appraisals and strategies to control perceived threat are also taken into account within the model. These comprise trauma characteristics, previous experiences and beliefs, and the current state of the individual.

From a treatment perspective the authors suggest that in order to reduce a sense of current threat and put the trauma in the past a change is required in three specific areas. In order to reduce intrusive re-experiencing the trauma memory needs to be elaborated and integrated contextually, negative idiosyncratic appraisals need to be modified, and maladaptive cognitive and behavioural strategies which prevent memory elaboration and exacerbate symptoms need to be abandoned (see Ehlers, Clark, Hackmann, McManus & Fennell, 2005). Research into various aspects of the theory including treatment has provided empirical support for the model.

Numerous studies including prospective longitudinal designs have provided supporting evidence for the role of negative trauma appraisals and trauma sequelae in the

Research has also demonstrated the role of data-driven processing and/or lack of self-referent processing, (Buck, Kindt, van den Hout, van den Steens & Linders, 2007; Ehring, Ehlers, Cleare, & Glucksman, 2008; Evans, Ehlers, Mezey, & Clark, 2007; Kindt, van den Hout, Arntz & Drost, 2008; Murray, Ehlers, & Mayou, 2002; Rosario, Williams, & Ehlers, 2006), for disorganised trauma memories (Evans, et al., 2007; Foa, Molnar, & Cashman, 1995; Jones, Harvey, & Brewin, 2007; Hagenaars, van Minnen, & Hoogduin, 2009; Halligan, Michael, Clark, & Ehlers, 2003; Jelinek et al., 2010; Jones, Harvey, & Brewin, 2007; Michael, Ehlers, Halligan & Clark, 2005; Murray et al., 2002), the role of perceptual priming in re-experiencing (Ehlers, Michael, Chen, Payne, & Shan, 2006; Michael, Ehlers, & Halligan, 2005), state and to a lesser extent trait dissociation (Ehlers, et al., 1998; Halligan et al., 2003; Karatzias, Power, Brown, & McGoldrick, 2010; Murray et al., 2002), mental defeat (Dunmore et al., 2001; Ehlers et al., 1998; Ehlers, Maercker, & Boos, 2000), and rumination about the trauma and its
consequences (Ehlers, Clark et al., 1998; Kleim & Ehlers, 2008; Murray et al., 2002; Steil & Ehlers, 2000).

Similarly the treatment protocol based on the model (Ehlers & Clark, 2000; Ehlers et al., 2005) has also been well supported, providing additional insight into the therapeutic cognitive processes in the treatment of chronic PTSD (Brewin et al., 2008; Ehlers et al., 2003; Duffy, Gillespie, & Clark, 2007; Hackmann, Ehlers, Speckens, & Clark, 2004; Speckens, Ehlers, Hackmann, & Clark, 2006).

In a review of psychological theories of PTSD, Brewin and Holmes (2003) concluded that Ehlers and Clark’s model represented the most detailed explanation for the aetiology of chronic PTSD and description of treatment strategies. Similarly Dalgleish (2004) reported the model provides one of the most comprehensive accounts on the role of cognitions in regard to both maintenance and recovery processes. However, the model is not without limitations. For example, assessment of, and drawing causal inferences from, peritraumatic cognitive processes poses methodological concerns, as retrospective self-report accounts of immediate responses may be influenced by current state and, as the authors recognise, dissociation and processing type (data driven vs. conceptual) may be interrelated. Additionally, elaboration of the role of peritraumatic dissociation and emotional numbing on the formation of trauma memories as well as further descriptive definitions of prior experiences would be elucidative. For example, the model acknowledges the influential role of background factors such as earlier experiences including prior trauma. However, prior (negative) experiences such as feeling anxious or depressed may not necessarily be associated with prior trauma. Past emotional problems have been found to be influential in the development and chronicity of PTSD (Ehlers et al., 1998; Ehring et al., 2008; Ozer, Best, Lipsey, & Weiss, 2003).
However, A. Ehlers (personal communication, March 16, 2010) acknowledged that prior experiences are not restricted to previous trauma. A major criticism put forward by Taylor (2006) concerns the complexity, or rather lack of parsimony, of the model. However, it is widely acknowledged that PTSD is a complex disorder comprising multiple cognitive, behavioural and affective components. Accounting for such complexity and attempts to encapsulate the etiology of symptom maintenance and treatment processes necessitates a comprehensive theoretical approach rather than a reductionist parsimonious approach. One of the aims of the research reported in this thesis is to test components of the cognitive model provided by Ehlers and Clark, (2000).

1.10 Summary

This chapter provides a brief overview of the historical accounts of traumatic events and subsequent psychological reactions, acknowledging the human suffering dating back many centuries. Initial conceptualisations of stress reactions primarily with a psychodynamic emphasis sought to explain responses in terms of individual characteristics. This was most evident in WWI in which 306 British troops were executed by firing squad having been convicted of cowardice and desertion associated with shell shock. By the end of WWII emotional symptoms associated with war syndromes started to be viewed as a normal reaction to exposure to the extraordinary stresses of warfare. The validity of psychological reactions associated with (combat) trauma was officially acknowledged post-Vietnam with the inception of the DSM-III in 1980.
Over time, recognition and understanding of pathological stress responses predominately associated with combat has made significant contributions to the development of the diagnosis which is now known as PTSD. Similarly, theoretical etiologies over time have also advanced. Earlier theories focused on characteristics of the individual and viewed the traumatic experience as incidental. Contemporary theories, whilst partly acknowledging the etiological aspects from earlier accounts, posit more comprehensive inclusive explanations comprising a complex interaction of pre, peri, and post trauma cognitive, emotional, and behavioural factors. Whilst recognizing the limitations of and differences between theoretical perspectives, it is generally acknowledged that psychological intervention requires an emotional engagement with feared traumatic memories (as well as other emotions) and the correction of erroneous dysfunctional cognitions.

This chapter also highlights the theoretical influences on diagnostic changes that have occurred since the inception of the DSM. Changes in criteria and definitions of what constitutes a traumatic event continue to evolve. Currently, diagnostic criteria warrant objective exposure and subjective perception of fear and threat. Hence, although exposure to a traumatic event is required, it is an individual’s subjective peritraumatic emotional response and personal meaning which further determines diagnosis. Furthermore, epidemiological studies have shown that trauma exposure is relatively common, though only a relatively small proportion of individuals exposed to trauma go on to develop PTSD. Individual differences and vulnerability factors play an integral role in the development and maintenance of post-trauma psychopathology. The role of pre, peri, and post-trauma vulnerability factors, and more specifically their influence on psychopathology following an MVA, is the focus of the following chapter.
2. Vulnerability Factors

Identification of variables that contribute to the development and maintenance of post-trauma psychopathology is important from a treatment perspective to reduce the burden on individual and community resources. Additionally, identification also contributes to the theoretical understanding of PTSD and guides future research. Epidemiological studies have shown that only a relatively small number of people exposed to trauma develop PTSD. Kessler et al. (1995) reported a 60.7% trauma exposure rate yet only 20.4% of women and 8.2% of men developed PTSD. Although having experienced a traumatic event is an essential diagnostic criterion, it remains an insufficient explanation for the occurrence of PTSD, as multiple factors determine the response to trauma and an individual’s propensity to pathological outcomes (Bursztajn, Joshi, Sutherland & Tomb, 1995; Butler, Moffic & Turkal, 1999; Paris, 2000). Whether or not individuals who have experienced a trauma go on to develop PTSD can be explained, in part, by examining a range of individual differences that have been shown to be predictive in determining the potential risk of developing PTSD and also the maintenance of symptoms (Alexander, 1999; Bloom, 1999). These factors can be categorised into three interrelated components, comprising pre-trauma, peri-traumatic, and post-trauma variables.

2.1 Pre-Trauma Variables

Pre-trauma vulnerabilities comprise variables that an individual “brings with them” to the trauma. Research has identified several predispositional factors that have been found to increase the likelihood of developing PTSD following trauma exposure.
These include, but are not limited to, the experience of prior traumas (Brewin, Andrews, & Valentine, 2000; Ozer et al., 2003); previous PTSD (Blanchard, Hickling, Taylor & Loos, 1995; Breslau et al., 2008); childhood abuse (Breslau, Chilcoat, Kessler, & Davis, 1999; Brewin et al., 2008); family psychiatric history (Brewin et al., 2000; Ozer et al., 2003); and low general intelligence (Buckley, Blanchard & Neill, 2000; Brewin et al., 2000).

Similarly, female gender, younger age, having experienced past emotional problems and having a neurotic predisposition have also been identified as pre-trauma vulnerabilities. Each of these factors will be described in detail and examination of their role in the development and course of PTSD will be further reviewed.

2.1.1 Gender

Female gender has been shown to be a vulnerability factor in the development of PTSD (Kessler et al., 1995; Norris et al., 2002). In fact, as noted in section 1.6, upon trauma exposure the risk of women developing PTSD is twice that of men. Differences in the type of exposure have been shown, with females more frequently exposed to rape and molestation, and males reporting higher exposure to accidents, combat and physical attacks. Overall, the frequency, duration, and severity of PTSD symptoms for females is greater than that of males after they have been exposed to trauma (Davis & Breslau, 1998; Ditlevsen & Elklit, 2010; Breslau, Davis, Andreski, Peterson & Schultz, 1997; Kessler et al., 1995; Stein, Walker, & Forde, 2000; Tolin & Foa, 2006). However, not all studies have found a gender association. Hapke, Schumann, Rumpf, John, and Meyer (2006) recruited a large \((n = 4075)\) community sample and found that in general females
did not have an increased vulnerability. They concluded that sexually motivated violent trauma and pre-existing anxiety explained the higher PTSD prevalence in women.

Research with MVA samples indicates a similar overall trend with many studies finding a gender difference with a greater proportion of females developing PTSD (Blanchard, et al., 1995, 1996, 2004; Coronas, García-Parés, Viladrich, Santos, & Menchón, 2008; Dougall et al., 2001; Ehlers et al., 1998; Frommberger et al, 1998; Fullerton et al., 2001; Green, 1994; Hickling, Gillen, Blanchard, Buckley & Taylor, 1998; Irish et al, 2008; Matsuoka et al., 2008; Mayou, Ehlers & Bryant, 2002; Ongecha-Owuor, Kathuku, Othieno & Ndetei, 2004; Stallard & Smith, 2006; Ursano et al., 1999; Wrenger, Lange, Langer, Heuft, & Burgmer, 2008; Zatzick et al., 2002). Bryant and Harvey (2003b) reported a number of significant gender differences with females experiencing more depression, Acute Stress Disorder (ASD) (23% vs. 8%), and PTSD at 6-months post-MVA (38% vs. 15%).

Conversely, several MVA studies have found no association between female gender and PTSD (Barth, Kopfmann, Nyberg, Angenendt, & Frommberger, 2005; Beck, Palyo, Canna, Blanchard, & Gudmundsdottir, 2006; Benight, Cieslak, Molton, & Johnson, 2008; Bryant & Harvey, 1995a; Coffey, Gudmundsdottir, Beck, Palyo & Miller, 2006; Ehring et al., 2008; Hamanaka et al., 2006; Hepp et al., 2008; Jeavons & Greenwood, 2000; Koren, Arnon, & Klein, 1999; Shalev et al., 1998).

Breslau et al. (1997) suggested that the difference between men and women in the prevalence of PTSD may be due to characteristics of the individual and of the traumatic experience. Davis and Breslau (1998) suggest that females may experience a greater
sense of helplessness and have more difficulty in reducing arousal and depressive symptoms. Foa and Rothbaum (1998) found that women were more likely to view the world as a dangerous place following a traumatic experience, a cognition associated with PTSD development and symptom severity. Further to this, Olff, Langeland, Draijer, and Gersons (2007) suggested that gender differences in negative threat appraisals may contribute to the risk of PTSD, with females more likely to report threat and loss of control, and to appraise events as stressful. Ehlers et al. (2008) reported that female MVA survivors perceived their accident as more frightening than males survivors did. Similarity, Mayou, Bryant, and Ehlers (2001) found that females reported more fear and emotional distress post-MVA.

Lucas (2003) found that females who had been involved in an MVA in the previous 5-years reported significantly more concern for their personal safety than males, possibly due to gender specific appraisals regarding safety. Tolin and Foa (2002) examined gender differences in trauma appraisals using the posttraumatic cognitions inventory (PTCI; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999) and found that females reported more negative beliefs about themselves, more self-blame, and perceived the world as more dangerous than males did. More recently, Cromer and Smyth (2010) also found significant gender differences with females reporting more self-blame. A dose-response effect was also evident, with greater trauma exposure related to greater negative post-trauma cognitions.

The subjective nature of immediate cognitive perceptions and emotional reactions has been shown to predict the development of PTSD. When they assessed the subjective role of intense fear, helplessness and horror (A2 DSM-IV criterion), Creamer,
McFarlane, and Burgess (2005) found that more women met the criterion than men. Furthermore, women report more peritraumatic dissociation (Bryant & Harvey, 2003b; Fullerton et al., 2001; Irish et al., 2011) following MVAs, subsequent to experiencing peritraumatic emotions (e.g., intense fear and helplessness) (Gershuny, Cloitre & Otto, 2003; Nixon, Resick & Griffin, 2002). In a study which examined gender differences and maintaining factors post-MVA, females reported having experienced significantly more peritraumatic fear than males (Beck et al., 2006). This finding was similar to that of Vaiva et al. (2003) who reported that significantly more women experienced peritraumatic fright (fear of death) in a sample of seriously injured MVA survivors. On the basis of a recent study, Lilly, Pole, Best, Metzler, and Marmar (2009) suggested that peritraumatic emotions (including intense fear) preceding peritraumatic dissociation may explain the gender disparity. The main thrust of these findings is that subjective peritraumatic reactions facilitate psychopathology, and that these cognitive and emotional reactions appear to be more pronounced in females.

Ursano et al. (1999) suggested that gender differences may be attributed to differences in neurobiology, stress hormones, trauma type, and personality factors. The personality construct neuroticism (further explained in section 2.1.3) has been recognised as a risk factor for trauma exposure (Breslau, Davis, & Andreski, 1995), but perhaps more importantly, has often been found to be associated with PTSD (Norris et al., 2002). Gender differences in personality constructs have been consistently found, with women reporting higher scores on neuroticism than men (Lynn & Martin, 1997; Schmitt, Realo, Voracek, & Allik, 2008). The neurotic predisposition in females may help explain the PTSD gender disparity by providing an indirect link between gender, neuroticism, and PTSD. Additionally, Stein et al. (2000) suggested that, since women also show higher
rates of depression and anxiety disorders, this may also contribute to higher rates of PTSD. However, Fullerton et al. (2001) and Breslau et al. (1997) found that prior major depression and anxiety disorders did not explain gender differences in PTSD. Similarly, Bromet et al. (1998) reported that a history of anxiety did not predict lifetime PTSD in females but it did in males, whereas prior depression increased the risk for PTSD in females but not males.

2.1.2 Section Summary

Gender bias in reporting, type of event and pre-trauma psychopathology, as well as prior trauma experience, although plausible explanations, have not provided consistent evidence to explain the gender disparity (Breslau, 2009). Tolin and Foa (2006), in their meta-analysis of gender differences in PTSD, found that irrespective of methodological variation between studies (e.g., type of study, population, age, and psychometric assessment) women were more likely to develop PTSD. They concluded that gender discrepancies are both complex and sensitive, and further research is required to ascertain why female gender is a vulnerability factor. An aim of the research reported in this thesis will be to examine the role of gender and the inter-relationships with pre, peri and post-trauma outcomes.

2.1.3 Age

The findings from adult trauma studies have shown that age is both a risk and protective factor in the development of PTSD. Generally, older age has been found to be a protective factor whereas younger age is a risk factor in the development of posttrauma sequelae (Norris et al., 2002).
Brewin et al. (2000) found that younger age at the time of a trauma was a risk factor, more so for males than females and particularly in military samples. They found that retrospective studies which used a continuous measure (vs. diagnostic-discrete), and those which used interviews showed the strongest effect size for younger age at the time of a trauma.

Norris, Kaniasty, Conrad, Inman, and Murphy (2002), who examined age differences on PTSD in disaster samples from the United States, Mexico, and Poland, found that older age was protective in survivors only from the United States and Mexico. Their results also showed that middle-aged participants (40 to 59 years) from the United States were at greater risk compared to younger and older participants, thus replicating earlier findings (Thompson, Norris, & Hanacek, 1993). The study by Magruder et al. (2004) on Vietnam veterans showed that older participants (≥ 65 years) reported significantly less PTSD symptoms than both younger age groups (≥ 50 years and 50-64 years). More recently Ditlevsen and Elklit, (2010) used data from previous Danish and Nordic trauma studies (N = 6,548) to examine the effect of age on PTSD and found that the lowest prevalence rate was for both men and women aged in their seventies. Similarly, Acierno, Ruggiero, Kilpatrick, Resnick, and Galea (2006) examined PTSD risk and protective factors after the 2004 Florida hurricanes. They found that older (≥ 60 years) participants were less symptomatic than younger participants (18 to 59 years). This suggested that older participants are more resilient post-trauma. It is feasible that older participants, given greater life experience, are more adaptive and possibly desensitised to the effects of trauma and may have developed adaptive coping skills over time and built up (at least potentially) a greater social support network.
Some studies however have not found a significant age association. Martz, Blackwell, and Birks (2005) reported that age and other demographic variables were not predictive of PTSD. Matthews, Harris, and Cumming (2009) found no significant age or gender differences between injured hospitalised participants with and without PTSD. The later finding is in accord with the results of Christiansen and Elklit (2008) who examined risk and protective factors from two trauma samples and found that irrespective of gender, age did not significantly predict PTSD.

Ticehurst, Webster, Carr, and Lewin (1996), when they examined the psychosocial impact of the 1989 Newcastle earthquake, found that older participants (≥ 65 years) reported higher symptom levels and, in a subsequent study, symptoms were found to persist longer in older participants (Lewin, Carr, & Webster, 1998). These results are in direct contrast to studies that have shown older age to be a protective factor. However, older participants may have found the effects of the earthquake more disruptive particularly for those whose homes were damaged or destroyed and had to deal with insurance companies and building contractors for many months after the earthquake.

Age differences in MVA samples are somewhat inconclusive, with numerous studies finding no difference in age between MVA survivors who experienced psychopathology and those who did not (Barth et al., 2005; Beck et al., 2006; Benight et al., 2008; Blanchard et al., 2004; Bryant & Harvey, 1995b; Freedman, Brandes, Peri, & Shalev, 1999; Hickling et al, 1998; Jeavons et al., 2000; Karl et al., 2009; Koren et al., 2001; Kupchik et al., 2007; Mayou et al., 2001; Ursano et al., 1999; Vaiva et al 2003; Zatzick, et al., 2002). Conversely, Conlon, Fahy, and Conroy (1999), who recruited a sample with minor injuries, found that increasing age was a significant independent predictor of
post-MVA morbidity. However, given the small sample size ($n = 40$ at 7-day baseline and $n = 32$ at 3-month follow-up), and the use of regression analysis, these results would appear to lack statistical power, reducing the strength of conclusions.

Other MVA studies have found a significant relationship between younger age and psychopathology. Irish et al. (2008) found that younger age was associated with PTSD symptoms 6-weeks post-MVA, though no age association was found at 1-year follow-up. It may be that younger drivers who are less experienced take greater risks, such as speeding, which increases the likelihood of being involved in an MVA. Younger age has also been associated with peritraumatic dissociation, with younger subjects reporting more symptoms (Fullerton et al., 2000). In one study younger age was found to predict higher acute stress severity though, as Harvey and Bryant (1998) acknowledge, approximately 80% of the MVA sample were aged less than 40-years.

2.1.4 Section Summary

The mixed findings on the influence of age make it difficult to draw any clear conclusions. However, age usually appears to have an influence when it is discretised into separate categories rather than being analysed as a continuous variable. Norris, Kaniasty et al. (2002) concluded that there is no single effect of age as such; rather the influence of age (younger, middle-aged or older) is dependent on the trauma environment as well as social, cultural, economic, and historical contexts. One of the aims of the research reported in this thesis is to examine the influence of age on peritraumatic and post-MVA morbidity.
2.1.5 Personality Factors

Neuroticism is a personality dimension that was conceptualised by Eysenck to explain the dispositional characteristic of trait anxiety. In his theory of personality Eysenck (1967) posited that neuroticism is associated with increased reactivity of the limbic system, as well as a low tolerance for aversive stimuli and stress. Neuroticism is considered to be stable and resistant to modification, reflects emotional instability, and a vulnerability to stress and anxiety proneness. Generally, people with a neurotic predisposition are overly reactive to stimuli, emotionally unstable, with anxiety levels that are disproportionate to the realities of a given situation. Accordingly, Cattell (1965) found that neurotics show extreme and indiscriminate emotional sensitivity to others, display considerable capricious behaviour, are subject to mood fluctuations in addition to being highly anxious, and easily overcome by their emotions.

Personality factors have been associated with an increased risk of exposure to trauma and severity of PTSD symptoms (Lauterbach & Vrana, 2001). Breslau et al. (1995) reported that the predispositional factors of male gender as well as high scores on neuroticism and extraversion were predictive of trauma exposure. In terms of MVAs, driver characteristics including personality have been shown to be related to the incidence of MVAs. Shaw and Sichel, (1971) compared the personality characteristics of bus drivers with good and bad safety records and found that those with bad driving records were more extraverted and more neurotic. Pestonjee and Singh, (1980) reported that participants who were involved in multiple MVAs scored significantly higher on extraversion than participants who had experienced a single MVA and those who had not experienced an MVA. In an international study comprising data from 34 countries, Lajunen (2001) also found that extraversion was associated with increased MVA’s.
However, no association was found with neuroticism, which is consistent with previous findings of Isherwood, Adam, and Hornblow, (1982), Pestonjee and Singh, (1980), and Wilson and Greensmith, (1983).

A neurotic predisposition has been linked to adverse outcomes such as PTSD, depression and general distress following trauma exposure (Borja, Callahan, & Rambo, 2009; Engelhard, van den Hout, & Lommen, 2009). McFarlane (1990) suggested that personality factors and the personal meaning of the trauma are significant determinants of the trauma response. In trauma populations numerous studies have shown that higher scores on neuroticism were predictive of PTSD and/or associated with greater symptom severity (Bunce, Larsen & Peterson, 1995; Casella & Motta, 1990; Davidson, Kudler & Smith, 1987; Fauerbach, Lawrence, Schmidt, Munster & Costa, 2000; Lauterbach & Vrana, 2000; Lewin, et al., 1998; McFarlane, 1989; Morgan, Matthews, & Winton, 1995).

Research on MVA samples which has investigated the influence of neuroticism on PTSD has provided mixed results. Mayou, et al. (1993), who employed the Eysenck Personality Inventory (EPI) (Eysenck & Eysenck, 1992), reported that neuroticism was significantly associated with acute distress. However, no association was found with PTSD at 3-month follow-up. Conlon, Fahy, and Conroy (1999) also found that neuroticism was not associated with PTSD at 3-months post-MVA in a sample with minor injuries. Conversely, Holeva, and Tarrier (2001) found in a prospective study that baseline measures of neuroticism, and to a lesser extent psychoticism, predicted PTSD at 3-month follow-up. Their results also showed that those with PTSD reported significantly lower extraversion scores than non-cases. Dorfel, Rabe, and Karl (2008)
conducted a cross-sectional study with participants who had experienced an MVA at least 6-months prior to involvement of the study. Their results showed that neuroticism was a predisposing risk factor for PTSD and that extraversion was a protective factor. This later finding replicates the earlier results of Nightingale and Williams (2000) who also found a negative relation between PTSD and extraversion at 6-weeks post-MVA. Although extraversion has previously been identified as a risk factor for trauma exposure (given its association with risk-taking adventurous behaviour and impulsiveness), Dorfel et al. (2008) suggested that extraversion may also be associated with an increased threshold for arousal, thereby ‘buffering’ against the impact of trauma. Furthermore, Lawrence and Fauerbach (2003) proposed that people who score high on extraversion may be more inclined to ‘seek out’ social support following trauma exposure, and as such are more likely to experience a reduction of symptoms.

Given that the majority of studies which have examined the role of neuroticism are retrospective, it is difficult to determine causality as measures have been taken post-trauma. For example, is neuroticism a cause or consequence of PTSD or both? It is also possible that trauma exposure and psychopathology (current state) unduly influence self-report measures of pre-morbid (trait) personality, or alternatively that experiencing a traumatic event changes personality characteristics.

Previous studies with military samples, in which measures including personality were taken on or prior to enlistment (pre-trauma/pre-deployment), have found that exposure, and to a lesser extent personality, were significantly associated with PTSD (Bramsen, Dirkzwager, & van der Ploeg, 2000; Schnurr, Friedman, & Rosenberg, 1993). More specifically, O’Toole, Marshall, Schureck, and Dobson (1998) found that Australian
soldiers who later developed PTSD had higher neuroticism scores at enlistment. In a more recent study, higher neuroticism scores were associated with greater PTSD severity and depression symptoms as well as somatic problems after controlling for trauma severity. However, after controlling for pre-trauma symptoms it was found that neuroticism no longer predicted post-trauma symptoms. The increase in symptoms between pre and post-trauma showed no difference in participants who scored high and low on neuroticism. Based on these findings the authors suggested that those with a neurotic disposition are not more reactive to traumatic stressors (Engelhard, van den Hout, & Lommen, 2009).

In an Australian community-based study examining pre-trauma attributes (Parslow, Jorm, & Christensen, 2005), neuroticism was shown to increase reported PTSD symptoms following the 2003 Canberra bushfires. However, exposure to trauma threat and reaction provided a greater contribution in explaining symptoms. Engelhard, van den Hout, and Kindt (2003) assessed pre-trauma measures including neuroticism and arousal symptoms. They found that after they had experienced pregnancy loss (e.g., miscarriage), women who reported more neuroticism also reported more PTSD symptoms, particularly arousal. However, these women also reported more symptoms pre-trauma, with no significant difference in symptoms between high and low neuroticism groups. These findings support their more recent results (Engelhard et al. 2009) which showed that neuroticism does not explain the development of post-trauma symptoms.
2.1.6 Section Summary

Given the mixed findings on the role of neuroticism in the development and maintenance of PTSD in both retrospective and prospective studies, it is difficult to draw clear conclusions. The relationship may not always be linear, and the effect of neuroticism may be confounded by other variables such as gender and event characteristics. Further investigation is needed to ascertain if indeed a neurotic disposition is a vulnerability or protective factor for initial and post-trauma responses. To elucidate the nature of the relationship of these variables is another aim of the research reported in this thesis.

2.1.7 Past Emotional Problems

The role of pre-trauma psychological state as a vulnerability factor in the development and course of post-trauma psychopathology is well documented (Brewin et al., 2000; Ozer et al., 2003). Pre-trauma psychopathology including prior emotional problems, such as anxiety and depression, can often have a detrimental impact on initial emotional reactions, appraisals, and the ability to tolerate fear and threat (McFarlane & Yehuda, 1996). Ehlers and Clark’s (2000) cognitive model of PTSD acknowledges the role of pre-trauma factors and includes background variables such as prior experiences which influence cognitive and behavioural strategies used to control PTSD symptoms. Individuals with past emotional problems may be more vulnerable as a result of poor or impaired coping ability, reduced resilience and lower level of adjustment to stress, that is, they are more sensitive to stressful events (diathesis stress model) and this increases the risk of developing PTSD as their stress threshold is reduced.
Norris et al. (2002) reviewed the role of prior mental health problems from 26 disaster studies. They reported that both prospective and retrospective findings showed that survivors with prior mental health problems appear to be at greater risk for new or renewed psychological problems post-disaster. A meta-analysis by Ozer et al. (2003) found that pre-trauma psychological problems were associated with increased PTSD symptoms and diagnosis irrespective of sample type. However, greater effect sizes were found in studies using interviews, studies involving interpersonal violence and accidents, as well as those in which less time had elapsed between trauma and assessment (1 to 6-months vs. 6-months to 3-years). Overall, they concluded that peritraumatic processes were stronger predictors of PTSD than pre-trauma factors.

Similarly, a meta-analysis by Brewin et al. (2000) found a weak significant association between psychiatric history and the development of PTSD. However, they reported slightly stronger effect sizes for trauma intensity and post-trauma factors compared to pre-trauma factors. Brewin et al. suggested the possibility of shared variance (interrelation) between pre-trauma variables and suggest that aggregating pre-trauma variables may produce stronger effect sizes. They also suggested that the effects of pre-trauma variables may be mediated by either peri-traumatic and/or post-trauma variables.

Hapke et al. (2006) examined trauma, gender and pre-existing psychiatric disorders and found that participants with at least one pre-existing disorder had a threefold increased risk of PTSD, finding that prior anxiety disorders were more important than depressive disorders.

The influence of prior emotional problems is also well documented within the MVA literature. In a prospective study conducted by Ehring, Ehlers, and Glucksman (2008), past emotional problems were found to be associated with travel phobia, depression as
well as PTSD. However, variables derived from Ehlers and Clark’s (2000) cognitive model, including cognitive processing, dissociation, and memory disorganisation, provided a stronger association than past emotional problems. Shalev et al. (1998) found that prior depression was associated with greater risk for major depression post-accident. MVA survivors who reported having experienced anxiety, depression or irritability in the previous 4-weeks had greater symptom severity and a higher chance of chronic PTSD than survivors without past emotional problems (Ehlers, Mayou et al., 1998). Blanchard, Hickling, Taylor, Loos, and Gerardi (1994), Blanchard et al. (1995), and more recently Dischinger, Read, and Kufera (2009) all found that previous depression was associated with PTSD.

Conversely, some MVA studies have not found an association between past emotional problems and PTSD. The results from Coronas et al. (2008), Kupchik et al. (2007), Matsuoka et al. (2008), and Mayou et al.’s (1993) studies showed no significant difference in prior emotional problems between survivors with and without PTSD.

Past Emotional Problems and the Development of PTSD

The National Comorbidity Study found that retrospective measures of prior anxiety and affective disorders were associated with development of PTSD (Bromet, Sonnega, & Kessler, 1998). Schnurr, Lunney, and Sengupta (2004) examined pre-military, military and post-military factors associated with the development and maintenance of PTSD. Their results showed that depression which occurred before, during or after the Vietnam War was associated with the development of PTSD, though not with the maintenance of symptoms. They concluded that pre, peri and post-trauma variables were associated with the development of symptoms and that chronicity was associated with peri and
post-trauma variables. Several MVA studies have also shown an association between
past emotional problems and the development of PTSD. A study by Benight et al.
(2008) found that past emotional problems were significantly correlated with
posttraumatic symptoms at 1-week, and 1-month, but not at 3-months post-MVA.
Blanchard, Hickling, Taylor, et al. (1996) found that prior depression was associated
with the development and severity of PTSD symptoms. Similarly, Blanchard et al.
(2004) reported that depression which occurred prior to an MVA was a risk factor in
developing PTSD.

Past Emotional Problems and Chronic PTSD

The influence of prior emotional problems on chronicity of symptoms has been
identified in a range of trauma populations. When Lewin et al. (1998) examined
morbidity and recovery from the 1989 Newcastle earthquake, they found that prior
emotional problems were associated with chronicity of PTSD symptoms. Kleim et al.
(2007) recruited a sample of adults who had been assaulted, and found that prior anxiety
or depression symptoms were predictive of PTSD 6-months post-trauma. In a sample of
individuals trapped in a ballroom fire, prior anxiety (simple phobia) was found to be an
independent predictor of PTSD 7 to 9-months after the event (Maes, Delmeire, Mylle,
& Altamura, 2001). Dunmore et al. (2001) examined maintenance factors associated
with PTSD after physical or sexual assault and found that previous psychological
difficulties were associated with ongoing symptoms. However, cognitive variables,
including processing and appraisals, were also found to significantly predict
maintenance of PTSD at 6 and 9-months.
Several MVA studies have also illustrated a link between prior emotional problems and chronic PTSD. Koren et al. (1999) found that higher levels of premorbid psychopathology (affective and anxiety disorders) were an important risk factor for PTSD 1-year post-MVA. Ursano et al. (1999) found that a history of a prior anxiety disorder significantly increased the risk of acute and chronic PTSD (6-months). Additionally, it was found that prior depression was associated with a greater risk of developing acute but not chronic PTSD. Ehring, Ehlers, Clear, et al. (2008) investigated predictors of PTSD symptom severity and depression 6-months post-MVA, and found that prior emotional problems were predictive of PTSD symptom severity but not depression.

In a recent study of accident victims, psychiatric condition (axis 1 disorder) prior to trauma exposure was identified as a predictor of psychopathology assessed at 6 and 12-months post-trauma (Wrenger, Lange, Langer, Heuft, & Burgmer, 2008). These researchers suggested two independent pathways in the development of post-trauma psychopathology; PTSD with or without comorbidity, and post-trauma mental disorders due to pre-existing vulnerability. The authors cautioned that the incidence of post-trauma psychopathology may be over-estimated if pre-existing disorders are not differentiated.

Barton, Blanchard, and Hickling (1996) suggested that survivors who have experienced previous psychopathology are more likely to dissociate in response to trauma. This proposed relation between past emotional problems and dissociative reactions has since gained further support. In a sample of MVA survivors, people who had previously suffered major depression, as well as younger aged participants, reported significantly
more peritraumatic dissociation (Fullerton et al., 2000). Similarly, in a study of hospitalised accident survivors (22.9% MVA survivors), Wittmann, Moergeli, and Schnyder (2006) reported that retrospective pre-accident psychiatric diagnosis was associated with higher peritraumatic dissociation scores. More recently, Regambal and Alden (2009) found that, in an experimental design with undergraduate university students using structural equation modelling, pre-existing emotion (depression and anxiety) indirectly influenced intrusions mediated by cognitive processing. The authors contended that a deficit in conceptual processing results from cognitive demands due to experiencing distress. In participants with pre-existing emotional problems these cognitive demands are excessive and more likely to result in sensory (data-driven) processing.

2.1.8 Section Summary

The detrimental influence of prior emotional problems on post-trauma psychopathology appears to be fairly consistent. What remains unclear is the pathway and strength of the relationship. Meta-analyses have reported that peri and post-event factors are more influential than prior emotional problems on PTSD. Some studies have found no relation, some findings report an association with the development of PTSD whereas others have found an association between past emotional problems and chronic PTSD. Differences in trauma type, study design and assessment method (interview, questionnaire or self-report likert format) have yielded similar results. However, it is possible that differences relating to the recency and severity of past emotional problems, for example, lifetime, previous 4-weeks or current may have a bearing on post-trauma outcomes.
Brewin et al. (2000) proposed a meditational relationship between past emotional problems, peri and post-trauma variables on psychopathology. Barton et al. (1996) also suggested a meditational link between past emotional problems and peritraumatic dissociation. Furthermore, Regambal and Alden (2009) found that past emotional problems were associated with cognitive processing which directly influenced intrusive symptoms. These results imply that past emotional problems facilitate cognitive trauma responses that have been shown to be predictive of posttraumatic symptoms. It is also possible that past emotional problems may also be indirectly associated with other trauma variables that can potentiate the development and chronicity of symptoms. Further research examining specific pathway(s) of past emotional problems on post-trauma on initial trauma responses and post-trauma psychopathology may provide additional knowledge and a clearer understanding of the mechanisms involved which is an aim of this study.

2.1.9 MVA Fatigue

Driver fatigue has been identified as a major contributing risk factor for MVA’s (Conner et al., 2002; Cummings, Koepsell, Moffat, & Rivara, 2001; Dinges, 1995; Fell & Black, 1997; Johns, 2000; Lal & Graig, 2001; Philip et al., 2005; Philip, Verialle, Breton, Taillard, & Horne, 2001: Smith & Trinder, 2000). It is conservatively estimated that fatigue was a contributing factor in at least 15% of all fatal MVA’s in NSW (NSW RTA, 2011). This estimate is based upon police reports and/or crash assessments where the vehicle performed a manoeuvre which suggests a loss of concentration due to fatigue. Others have estimated that up to 30-33% of MVA’s can be attributed to fatigue (Moore & Brooks, 2000; Pierce, 1999; Robertson, 2003).
The terms fatigue, sleepiness, and drowsy are often used interchangeably though each have distinguishable precise definitions with some overlap. In the current context psychophysiological symptoms of fatigue include; feeling tired or drowsy with decreased alertness and inattentiveness, as well as loss of concentration, which impair elements of performance that are essential for safe driving. Fatigue also reduces vigilant attention and promotes sleepiness. As fatigue gradually develops, levels of alertness fluctuate which masks the decline in performance capacity. This is particularly relevant for drivers who may lose awareness of the decline in their driving performance (Horne & Rayner, 1999). Reduced levels of alertness and increased sleepiness impair the ability to process and integrate information, the accuracy of short term memory decreases, attention-based performance declines and reaction time increases (Dinges, 1995). As information processing is impaired the ability to anticipate and detect crash risk situations are reduced (Swan, 2000). Decreased physiological arousal, reduced sensorimotor function, impaired judgement and a gradual withdrawal of attention from the demands of driving affects the ability to control the vehicle (Lal & Craig, 2001; Mascord & Heath, 1992).

Conner et al. (2002) used the Stanford sleepiness scale (SSS; Hoddes, Zarcone, Smythe, Philips, & Dement, 1973) to assess levels of alertness and found that decreased levels of alertness in MVA drivers were associated with increased risk of being involved in an accident. The association between level of alertness and subsequent risk of MVA is well documented (see Connor, Whitlock, Norton, & Jackson, 2001). However, the role of alertness and its influence on post-accident psychopathology is unknown. It is plausible that reduced levels of alertness influence peritraumatic responses. Given that cognitive impairments (impaired ability to process and integrate information as well as deficits in
memory function) are involved in dissociation, it is possible that reduced alertness may also influence peritraumatic dissociation. Giesbrecht, Smeets, Leppink, Jelicic, and Merckelbach (2007) conducted an experimental study to explore the relationship between sleepiness and acute dissociation. Their sample comprised 25 undergraduate students who, during a 32-hour period of wakefulness, were assessed periodically using a Dutch translated modified version of the peritraumatic dissociative experiences questionnaire (PDEQ; Marshall, Orlando, Jaycox, Foy, & Belzberg, 2002) and the Stanford sleepiness scale. Results indicated that disruptions in circadian rhythms, which affect wakefulness and level of arousal, had a detrimental effect on attentional control and memory. Giesbrecht et al. also found that reduced levels of alertness (fatigue) predicted acute dissociative tendencies. The authors suggested that fatigue increases acute dissociation thus impairing conscious cognitive control which increases the risk of an accident. Mellman, David, Kulick-Bell, Hebding, and Nolan, (1995) reported that sleep disturbance in the 4-weeks before Hurricane Andrew was a vulnerability factor for posttrauma morbidity. Despite limitations such as small sample size ($N = 54$), subjective reports and potential reporting bias as well as a retrospective design (6 to 12-months post-event), this appears to be the first study to examine the role of sleep related problems prior to trauma exposure and provides tentative results of the potential influence of reduced arousal on (post) exposure morbidity.

### 2.1.10 Section Summary

Clearly reduced alertness increases the risk of an MVA since dissociative responses may influence an individual’s ability to circumvent an accident. What is not clear is the role of fatigue on cognitive and emotional reactions during and immediately after an MVA. It may be that MVA survivors who are less alert are more prone to dissociate.
Reduced cognitive functions associated with decreased alertness may also impair the ability to process the event in a conceptual way. Peritraumatic dissociation has been identified as a risk factor in the development of PTSD following an MVA (Murray et al., 2002). An exploratory component of the research reported in this thesis is to examine the role of alertness and immediate trauma responses and post-accident psychopathology.

2.2 Peri-Trauma Variables.

Peri-trauma refers to factors occurring during or immediately after the trauma. They comprise event-related characteristics, such trauma severity and injury severity, cognitive reactions including peritraumatic dissociation, as well as emotional responses including feelings of fear, helplessness, and horror.

2.2.1 Trauma Severity

Numerous studies have found a significant association between the severity of a traumatic event and PTSD severity. A dose-response effect where the magnitude of the stressor directly influences the degree of psychopathology has been reported in a variety of trauma samples as well as cross-culturally (Brewin et al., 2000; March, 1993; Norris et al., 2002). Defining trauma severity can be problematic as there is no global operational definition, and what may be deemed to constitute severity in one trauma type may not be applicable across a range of different traumas. For example, trauma severity of combat may comprise experiencing torture, prolonged and/or intense exposure (duration), being wounded, and number fatalities, whereas measures of severity in an earthquake sample may include proximity, personal loss and/or relocation as well as community destruction.
Green (1990) identified eight trauma severity ‘dimensions’ that she suggests are generic across trauma types. They comprise: 1) threat to life and body; 2) severe physical harm or injury; 3) intentional injury or harm; 4) exposure to the grotesque; 5) witnessing or learning of violence to loved ones; 6) violent sudden loss of loved ones; 7) learning of exposure to a noxious agent; and 8) causing death or severe harm to another. Similarly, with some overlap, Alexander (1998) outlined several factors that are associated with post-MVA psychopathology including, death, mutilation, prolonged exposure (e.g., being trapped inside a vehicle), perceived threat, as well as injury severity and resultant impaired physical functioning.

Within the MVA literature several different trauma characteristics and various assessment methods have been used to ascertain MVA severity. Several researchers have used a self-report likert scale (5 or 10-point format) rating of perceived accident severity (e.g., 1 = not at all bad to 10 = extremely bad) with mixed results. Hepp et al. (2008), Schnyder et al. (2001), Green et al. (1993), and Jeavons (2000) found that self-report ratings of perceived MVA severity were not associated with PTSD. However, Bryant and Harvey (1995a) found that perceived MVA severity was significantly correlated with psychological distress.

Frommberger et al. (1998) determined accident severity by the extent of injuries to others and did not find a direct association with PTSD. Dorfel et al. (2008) assessed MVA severity using objective injury scores as well as subjective severity ascertained using an unpublished MVA severity interview comprising questions relating to fear, loss of control, probability of being killed and general impairment. Results showed that subjective severity predicted PTSD severity, noting that subjective appraisals were more
important than objective characteristics, such as injury severity, which were not associated with PTSD severity. Ehlers et al. (1998) recruited an MVA sample, 26% of whom were admitted to hospital, and used objective injury scores, hospital admission, as well as persistent medical and financial problems at 3 and 12-months post-accident, to assess trauma severity. They found that persistent health and financial problems were predictive of chronic PTSD. These findings suggest that ongoing physical problems resulting from injury and financial difficulties represent ongoing stressors which are more important than objective ratings of initial injuries.

In two retrospective MVA studies with an approximate time since MVA of 6-years, Steil and Ehlers (2000) used objective characteristics of the accident, including information on injuries sustained and injury severity, length of hospitalisation, number of people killed or injured, and witnessing death or injury. Subjective severity was determined using perceived threat, probability of death or severe injury responses rated at 2-time points, (1) during the MVA and (2) from current perspective. In study 2, perceived probability of death or severe injury were assessed from current perspective. Results showed that 48% in study one and 54% of participants in study 2 met the criteria for PTSD but only weak and mainly non-significant correlations between PTSD and subjective and objective accident severity were found. Severity of injury in study 1 was significantly related to PTSD severity. This result is not surprising given that only 6% of participants in study 1 were uninjured compared to 23% in study 2, which may have restricted the range of (injury) effect.

Shalev et al. (1996) assessed trauma severity using the severity of event scale which comprises eight items based on Green’s (1990) eight trauma dimensions. Their results
showed that event severity did not differentiate participants with and without PTSD. O’Donnell et al. (2004) assessed trauma severity using measures including objective injury severity and the severity of event scale, and found that event severity differentiated PTSD from no diagnosis. Shalev et al. (1998) ascertained trauma severity using 12 professional raters who were blind to participants’ diagnostic status. Each rater listened to participants’ audio-taped scripts, which described their traumatic event, recorded 1-week post-trauma. Trauma severity ratings were then determined on a scale of 1 (not severe at all) to 10 (extreme severity) and averaged across the 12 raters. Their results indicated that participants with PTSD and PTSD comorbid depression rated the accident significantly more severe than participants with no PTSD and no comorbid depression and participants with depression only.

2.2.2 Section Summary

The findings on the role of MVA severity and subsequent PTSD severity are somewhat inconsistent, suggesting that a linear dose-response effect may be confounded by a number of factors. Methodological differences such as time-frame between MVA and assessment, the differing assessment methods and the use of different trauma characteristics used to determine trauma severity, prevent direct comparisons from being made. Brewin et al. (2000) reported that the effect size of trauma severity was significantly greater in retrospective studies (vs. prospective) and noted that if subjective assessment of severity occurs too soon after the trauma, when survivors may still feel numb, they may not be able to fully appraise the event and this could lead to an underestimation of trauma severity. Conversely, it could be argued that current psychological state and a reliance on retrospective recall of severity appraisal also presents bias potential with greater chronic symptomatology associated with an
amplification of memory for both trauma severity and injury severity (Harvey & Bryant, 2000; Southwick, Morgan, Nicolaou, & Charney, 1997). An aim of the research reported in this thesis was to examine the subjective influence of accident severity in relation to initial post-trauma responses and psychopathology over the longer term.

2.2.3 Injury Severity

The most widely used indicator of trauma severity in MVA studies is injury severity. Objective assessment of injury severity is predominately conducted using the Injury Severity Score (ISS; Baker et al., 1974) or the Abbreviated Injury Scale (AIS; American Association for Automotive Medicine, 1980). Subjective rating of injury severity is also used and generally comprises a self-report rating using likert format (e.g., 1 = minor injuries to 5 = life threatening injuries). Numerous MVA studies have found significant associations between objectively assessed injury severity and PTSD (Blanchard, Hickling, Barton, et al., 1996; Blanchard, Hickling, Mitnick et al., 1995; Blanchard, Hickling, Taylor, et al., 1996; Coronas et al., 2008; O’Donnell et al., 2004). Similar findings were evident in a study by Blanchard, et al. (1997) in which the severity of injury, and perhaps more importantly the degree of recovery from injury, was predictive of PTSD 6-months post-MVA. More recently Hamanaka et al. (2006) reported that physical injury severity and persistent physical disability were predictive of PTSD.

Jeavons (2000) found that subjective appraisal of injury severity was more influential in the longer term than initial objective ratings. Similarly, Murray et al. (2002) reported a significant association between subjective appraisals of injury severity and PTSD severity at 4 and 6-weeks, whereas objectively measured injury severity showed no significant association with symptom severity.
Conversely, numerous MVA studies have found that injury severity (predominately objective) was not associated with PTSD (Bryant & Harvey, 1995a, 1996; Ehring et al., 2008; Epstein, 1993; Green et al., 1993; Irish et al., 2008; Jones et al., 2005; Koren et al., 1999; Matthews et al., 2001; Schnyder et al., 2001; Ursano et al. 1999; Vaiva et al., 2003).

Within the MVA literature the influence of injury severity on PTSD is somewhat inconsistent. In an earlier review of trauma research, Green (1994) stated that objectively assessed injury severity was not always the best predictor of psychopathology, explaining that the influence of injury severity on PTSD is more likely to be observed when only a subset of participants are injured. However, this does not always seem to be the case within the MVA trauma literature. Irrespective of injury heterogeneity the findings remain inconclusive, as studies in which all participants were severely injured have produced mixed findings, as have studies where the majority of participants sustained very minor injuries.

The disparity between the results of studies on the impact of injury severity and poor psychological outcome may be better understood by examining the appraisal of severity and impact of injury from a subjective perspective rather than objectively. In essence, it is the appraisal and personal significance of injury, which is the degree to which the injury affects physical functioning, self-esteem, occupational impairment and relationships over the long term, which may ultimately determine the influence of injury, rather than an initial objective medical measurement (Alexander, 1998). O’Donnell et al. (2004) found that cognitive appraisals (anxiety about injuries) were associated with PTSD at 3 and 12-months and PTSD and comorbid depression at 12-
months, and appraisal regarding the anticipated impact of injury was associated with PTSD and comorbid depression at 12-months. In line with Ehlers and Clark’s (2000) cognitive model of PTSD, negative appraisals of the consequences of trauma, such as injury, pain, financial problems, physical disability and physical appearance, serve to produce a sense of current threat. This maintains symptoms by directly producing negative emotions such fear, anger, guilt, and sadness, which in turn promotes dysfunctional cognitive and behavioural coping strategies that prevent cognitive change from occurring.

Jeavons, (2000), used subjective and objective injury ratings, and reported that length of time spent in hospital, emotion-focused coping and severity of injury were predictive of PTSD at 12-months. She found that victims’ own appraisals of injury were more important than objective injury assessment, and that over time serious injury and resultant disability, served as a constant reminder of the event and were predictive of PTSD. Mayou et al. (1997) concluded from a five-year follow-up study that both minor and severe injuries sustained in MVAs contribute to psychological problems in the longer term. They found that continuing physical problems, pain, and disability were predictive of PTSD five-years after the accident had occurred. Mayou and Bryant (2002) found that survivors with serious injury had more financial problems, worse physical outcomes and were more likely to report depression at 3-years post-accident than participants with less severe injuries.

Ehlers et al. (1998) found a significant relationship between PTSD and severity of symptoms at one-year post-accident in survivors who reported persistent medical and financial problems. They concluded that participants who suffered from continuing
medical and financial problems had a higher chance of a PTSD diagnosis and greater symptom severity. Furthermore, they suggested that these factors may represent chronic stressors that exceed the coping resources of individuals, which may impede the emotional process of overcoming the accident. This is consistent with other researchers who have found that chronic pain, physical disability, health problems, and financial difficulties are maintaining factors as they act as constant reminders of the event and appear to make psychological recovery more difficult by triggering posttraumatic intrusions (Blanchard et al., 1997; Green et al., 1993; Mayou & Bryant, 2001; Mayou et al., 2001).

Barth et al. (2005) in a five-year follow-up found that no MVA survivors with PTSD had completely recovered from physical injury. Their results showed the importance of both physical impairment and chronic pain in maintaining long term PTSD, and thus supported previous findings. Contrary to these findings, Koren et al. (2001) found that injury-related medical complications were not associated with PTSD at 12-months post-MVA. Instead, Koran et al. found that severity of initial posttraumatic symptoms were a ‘key’ risk factor for long term PTSD, though the small sample size ($N = 58$) somewhat limits the strength of their results.

2.2.4 Section Summary

Initial objective measures of injury do not readily capture the personal significance of injury, which may not be apparent for many months or even years post-accident. Injuries similar in nature may be appraised quite differently depending on the (individual) personal meaning assigned to them. For example, a finger injury may have great significance to a pianist but have little impact for a truck driver. Ongoing stressors,
such as pain and physical disability, may prevent re-appraisal, and subsequent psychosocial implications, including social, occupational, and financial difficulties, may serve to reinforce negative interpretations of perceived permanent change. Overall, it appears that injury severity is most influential when assessed subjectively. Appraisal of the injury, as well as the long term psychosocial implications associated with the injury, can maintain ongoing psychopathology, potentially for many years post-MVA. Another of the aims of the research reported in this thesis was to assess the effect of injury severity on the development and maintenance of PTSD.

2.2.5. Fear of Dying

The DSM-IV A2 diagnostic criterion specifies that in order to meet diagnosis following trauma exposure an emotional response comprising subjective feelings of intense fear, helplessness or horror must be experienced (APA, 2000). It is assumed that peritraumatic emotional reactions are essential for the development of PTSD. Theoretical accounts, such as those of Ehlers and Clark (2000) and Foa and Riggs, (1995), also acknowledge the importance of acute cognitive appraisals and affective reactions in the development and maintenance of PTSD.

During immediate and overwhelming traumatic threat, fear induced arousal is associated with the release of stress hormones (e.g., cortisol and catecholamines) which are said to interfere with encoding and impairment of intentional recall of trauma memories (Ehlers & Clark, 2000). At this time, trauma memories are thought to be ‘overconsolidated’ due to a heightened level of distress. In a highly aroused state the sympathetic nervous system becomes hyperactive and the formation of distressing
trauma memories is said to occur (Pitman, 1989; see also Yehuda, McFarlane & Shalev, 1998).

The importance of initial emotional reactions has also been identified within the MVA trauma literature. Thought to arise from intense immediate life threat, fear is considered fundamental to PTSD and is an emotional reaction frequently reported (Amstadter & Vernon, 2008). Mayou and Bryant (2001) found that MVA survivors who reported feeling very frightened immediately after the accident had significantly worse outcomes at both 3 and 6-months. Green, McFarlane, Hunter, and Griggs (1993) used a 4-point scale to assess perceived life-threat at 4-weeks and 18-months post-MVA, and found that participants with PTSD had significantly higher initial fear mean scores compared to those without PTSD. They also reported that event characteristics (injury and accident severity) had no effect on psychopathology. Similarly, Jeavons et al. (2000) and Wrenger et al. (2008) reported that, irrespective of event characteristics, threat to life was associated with chronic PTSD. Ehlers et al. (1998) in a prospective longitudinal study found that perceived threat (how frightening), independent of injury severity, was predictive of chronic PTSD. In a subsequent follow-up study Mayou et al. (2002) reported that variables including initial threat and dissociation were predictive of symptom severity at 3-years post-MVA.

Other studies have found significant associations between event characteristics, immediate threat and PTSD. Blanchard et al. (1995, 1996) reported that fear of dying was predictive of PTSD. Dorfel et al. (2008) reported results which also highlight the importance of subjective trauma appraisals; they found that fear of dying was predictive of PTSD symptom severity. Similar to the findings of Blanchard et al., Wu and Cheung
(2006) reported significant associations between perceived threat and posttraumatic symptoms.

The results published by Wu and Cheung (2006) showed that high levels of perceived threat predicted symptom remission over time (3 and 6-months). It is suggested that over time initial threat is re-appraised and the reality that the accident was not as severe or threatening as initially thought parallels a reduction in anxiety. However, ongoing medical problems and physical disability associated with an accident may impair such re-appraisals as they may confirm initial emotion reactions in MVA samples if the injuries are perceived as life-threatening. Interestingly, in a sample of injured MVA survivors with an average injury severity score (ISS; Baker, O’Neill, Haddon, & Long, 1974) of 2.12 (SD = 2.34), Ehring, Ehlers et al. (2008) found that the association between fear and PTSD symptom severity decreased over time. The initial correlation at 2-weeks ($r = .55$) gradually reduced over the course of 6-months ($r = .28$). Amstadter and Vernon (2008) examined peri and post-trauma emotional reactions (fear, shame, guilt, anger and sadness) within different trauma types (24% sexual assault, 22% physical assault, 24% transport accident and 30% illness/injury). A high level of peritraumatic fear was elicited in all trauma groups but this decreased post-trauma, whereas anger, shame, guilt, and sadness tended to increase post-trauma, particularly with interpersonal trauma (physical and sexual assault). The authors contended that, given time to process the event, some emotional responses tend to increase though fear decreases as the immediate threat is thought to have passed. Though the study did not assess the relation between emotional responses and psychopathology it did provide further evidence that trauma exposure can elicit a range of emotional responses which can vary and change over time according to trauma type.
Vaiva et al. (2003) assessed the influence of peritraumatic responses on PTSD using a sample of hospitalised MVA survivors. Their results showed that 57% of participants experienced intense fear, 32% reported helplessness, and 12% reported feeling horror during the MVA. At 2-months post-accident 46% of survivors had developed PTSD. Peritraumatic fright, comprising being confronted with death, a momentarily or complete absence of affect or thought, or being spaced out, was also assessed. A fright reaction was endorsed by 47 participants, 42 of whom developed PTSD. The A2 criterion was endorsed by 64 participants, 47 of whom developed PTSD. Though these results highlight the importance of intense emotional reactions, they also show that experiencing acute emotional reactions is not always a precursor for psychopathology.

It has been found that peritraumatic dissociation occurs in response to extremely intense and overwhelming emotional and physiological arousal (Gershuny & Thayer, 1999; van der Kolk & van der Hart, 1989). Bernat, Ronfeldt, Calhoun, and Arias (1998) recruited a sample of college students and found that the relationship between peritraumatic fear and dissociation was mediated by panic. Similarly, Fikretoglu et al. (2003) in a study ($M \geq 6$ years post-trauma) also found that panic partially mediated the relationship between peritraumatic fear and dissociation, and therefore proposed that peritraumatic dissociation is an epiphenomenon of peritraumatic distress. Gershuny et al. (2003), recruited a sample of female university students and found that the indirect effect of peritraumatic dissociation on PTSD was explained by fear of dying and losing control. They suggested that fear and loss of control may elicit dissociation but whether fear is a cause or consequence of peritraumatic dissociation remained unclear.
In contrast, a mediation analysis published by Nixon et al. (2004) failed to support the fear-arousal-dissociation model. Their results from two studies with female crime victims showed that fear predicted arousal and dissociation; however, fear irrespective of arousal remained an independent predictor of peritraumatic dissociation. Overall, these findings support the theorised relationship between experiencing intense peritraumatic emotional reactions (e.g., fear) and peritraumatic dissociation. However, methodological limitations, such as the use of college students, all female samples, and retrospective trauma assessment, limit both the conclusion and the generalisability of the results.

2.2.6 Section Summary

Intense emotional reactions are a common response to trauma with a range of emotions experienced. However, experiencing emotions such as fear, helplessness, and horror do not always necessarily lead to the development of PTSD. The role of fear and its association with PTSD is well documented, though the relationship may not be direct. Studies have shown that intense peritraumatic fear elicits dissociation which has been identified as a strong predictor of PTSD. Additionally, given that there is much variability in how MVA’s are experienced, fear appraisals may not always be immediate. For example, a collision may be unexpected and sudden (hit from behind) or may be anticipated over a period of time, such as an approaching truck on the wrong side of the road (head on collision). Similarly, MVA survivors who are trapped inside a vehicle and/or who sustain severe injuries may fear they are going to die. In this sense, fear could be immediate or occur after the collision has occurred upon (re) appraisal of the severity of the accident and/or injury. Additionally, the predominant use of self-report likert scales and retrospective assessments of fear and other acute emotional responses can pose
methodological problems as current state and personal meaning may influence subjective recall of distressing emotions. Another of the aims of the research reported in this thesis was to examine the relationships between peritraumatic fear, peritraumatic dissociation, and post-MVA psychopathology.

2.2.7 Dissociation

The term ‘dissociation’ dates back to 1812 when Benjamin Rush used it to describe characteristics of patients who were possibly suffering from mania or schizophrenia. In 1845 the term, similar to contemporary conceptualisations, was employed by Jacques-Joseph Moreau de Tours when he examined the psychological effects of hashish. He noted a “dissociation of ideas” and “disintegration of associations” (Carlson, 1986; van der Hart & Horst, 1989). However, Pierre Janet, in the late 19th century, in his studies of hysteria, was the first to link dissociation with overwhelming psychological stress and trauma, hence the development of the term psychological dissociation (Gershuny & Thayer, 1999). He theorised that dissociation of affect and memory of trauma experiences were adaptive defence mechanisms of hysteria, although ongoing dissociation and related emotional constriction were said to result in various forms of psychopathology. Similar to contemporary cognitive theories of PTSD, Janet considered the recovery and integration of fragmented trauma memories to be an essential component in the treatment of psychological trauma (van der Hart, Brown, & van der Kolk, 1989)

In recent years there has been resurgence in the study of trauma-related dissociative processes, in response to trauma and influence on post-trauma psychopathology. There is no single definitive description of the construct of dissociation. However,
contemporary conceptualisations, though somewhat inconsistent, present descriptive definitions with some similarities. According to the DSM-IV, dissociation is described as “a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment” (APA, 1994, p. 477). Van der Kolk, van der Hart, and Marmar (1996) described dissociation as a compartmentalisation of experience in which “elements of the trauma are not integrated into a unitary whole or an integrated sense of self” (p. 306). Similarly, Bernstein and Putnam (1986) stated that dissociation is “the lack of normal integration of thoughts, feelings, and experiences into the stream of consciousness and memory” (p. 727). Hence, dissociation could be described as a parallel state of consciousness in which perception of time, self, space, and awareness of reality become distorted (Scaer, 2001).

Dissociation, though often viewed as pathological, comprises a continuum of symptoms some of which are viewed as part of a normal experience (APA, 2000). For example day-dreaming or fantasising (aspects of absorption) are common experiences and not considered pathological (compared to depersonalisation\(^1\) or derealisation\(^2\)). Dissociative experiences varying in frequency and intensity have been found to occur in both clinical and non-clinical samples (Bernstein & Putnam, 1986; Ross, Joshi, & Currie, 1990; van Ijzendoorn & Schuengel, 1996). Non-pathological dissociation, which is considered ubiquitous and part of everyday life, comprises absorption and fantasy proneness. These experiences involve alterations in consciousness such as a reduced awareness of external events, problems associated with reality monitoring, daydreaming, intense

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1 Depersonalisation refers to feeling detached or estranged from one’s self, a sensation of being an observer of one’s body, feeling like an automaton, as if watching a movie or living a dream (APA, 2000).

2 Derealisation refers to an alteration in the perception of one’s surroundings so that a sense of reality of the external world is strange or unreal (APA, 2000).
imaginative activities, and a sense of functioning on ‘automatic pilot’ (Eisen & Lynn, 2001; van der Hart, Nijenhuis, & Steele, 2005). Hence, dissociation may be viewed as either state/situational (in response to intense acute overwhelming emotions) or as a trait, in which an individual may be predisposed and have a greater propensity to dissociative in everyday life.

Trauma-related dissociation is thought to arise in response to an event that is perceived as intense and overwhelming and could be construed as a form of mental escape when physical escape is not possible (Gershuny & Thayer, 1999). From this perspective dissociation is thought to be a defensive response, functional in nature, which safeguards one’s psychological integrity by reducing the psychological impact of trauma (Cardena, 1994).

Dissociation which occurs during or immediately after trauma is referred to as peritraumatic dissociation. It involves a complex array of acute alterations in both perceptual and cognitive functioning which leads to a sense of unreality (Marmar, Weiss, & Metzler, 1998). Peritraumatic dissociative reactions include somatoform and psychoform dissociative symptoms (defined by physical and psychic symptoms respectively) and, though phenomenologically distinct, both have a similar traumatogenic etiology (van der Boom, van den Hout, & Hunjtens, 2010). Peritraumatic dissociation symptoms include depersonalisation, derealisation, time distortion (slowed down or accelerated), temporal confusion where the sequence of events is incomplete, lack of pain perception, and disorientation (Marmar et al.,).
The effect of peritraumatic dissociation and its relation with psychopathology appears to be mixed. On the one hand it is proposed that acute dissociative reactions are adaptive in the short term, initially serving as a protective avoidance function by reducing conscious awareness of overwhelming aversive emotions such as an imminent fear of death during the trauma experience. This diverts conscious attention away from threatening stimuli and in the process limits the ability to encode the situation. This in turn reduces aversive trauma memories (Horowitz, 1986; van der Kolk, 1987). Alternatively, it is proposed that peritraumatic dissociation potentiates posttraumatic symptoms since processing and subsequent memory recall is impaired by restricted awareness (van der Kolk & van der Hart, 1989). Encoding of trauma memory is disrupted such that vivid sensory-perceptual event information (rather than contextual information) forms the basis of trauma memories. Deficits in trauma memory, including memory fragmentation, impede the recovery process as the incoherent memory of the trauma cannot be elaborated and integrated contextually as well as temporally (Ehlers & Clarke, 2000; Foa & Hearst-Ikeda 1996). The later perspective supports Janet’s original account that dissociative trauma responses increase the risk of psychopathology.

In a meta-analysis which examined predictors of PTSD, Ozer et al. (2003) found that peritraumatic psychological responses, predominantly peritraumatic dissociation (effect size $r = 0.35$), and to a lesser extent peritraumatic emotions (e.g., fear, helplessness, horror, guilt, & shame; $r = 0.26$), as well as perceived threat ($r = 0.26$), were the strongest predictors of PTSD. However, methodological concerns such as the retrospective assessment time-frame, which ranged from 1-month to over 20-years post-trauma, as well as differing psychometric instruments (8 different measures were used to assess peritraumatic dissociation), and different outcome measures (PTSD symptoms,
or PTSD diagnosis) limit the conclusion of any clear predictive and temporal relationships. Breh and Seidler (2007) in their meta-analysis also reported a significant positive relation between peritraumatic dissociation and PTSD with an overall effect size of $r = 0.36$. Further analysis of retrospective studies, deemed to show a relationship between peritraumatic dissociation and PTSD, produced an effect size of $r = 0.37$. Quasi-prospective studies (peritraumatic dissociation assessed prior to that of PTSD) showed an effect size of $r = 0.35$. Surprisingly they found that their results were homogenous, indicating no significant differences between studies. This is in contrast to the findings of Ozer at al. who found that the strength of the relationship between peritraumatic dissociation and PTSD varied significantly with the time that had elapsed between event and assessment, type of sample, and method of assessment. Another meta-analysis, conducted by Lensvelt-Mulders et al. (2008), found a significant positive association between peritraumatic dissociation and posttraumatic stress ($r = .401$). They also found significant variability between effect sizes of studies which depended on sample type, assessment time, and design of the study. Similarly, van der Hart, van Ochten, van Son, Steele, and Lensvelt-Mulders (2008) conducted a critical review using 53 empirical studies comprising both cross-sectional and longitudinal designs. They found that 34 studies (64.1%) that included community, medical, and, psychiatric samples provided evidence that the experience of peritraumatic dissociation increased the risk of posttraumatic stress.

Critical reviews and meta-analyses ‘generally’ support the association between acute trauma dissociative reactions and post-traumatic stress responses. However, as noted by Lensvelt-Mulders et al. (2008), and van der Hart et al. (2008), differences in study designs and samples with varied results across studies preclude clear conclusions from
being drawn. What is clear is that not all trauma survivors experience acute dissociative reactions. However, this does not necessarily preclude the development of PTSD, as results (overall effect sizes) indicate that peritraumatic dissociation is neither sufficient in identification of risk nor necessary for the development of PTSD, with much variance remaining unexplained. Bryant (2007) proposed that the relationship between peritraumatic dissociation and PTSD is not linear and that potential variables that may contribute to the association between peritraumatic dissociation and PTSD need to be considered.

Examination of known predictive factors that may share variance and combine in an additive manner and/or mediate or moderate the relationship between trauma exposure and experiencing peritraumatic dissociation may help to disentangle the complex interactions that lead to PTSD. The personality construct of neuroticism has been shown to be associated with peritraumatic dissociation and has also been found to be a risk factor for PTSD. When they examined personality factors which predict dissociative tendencies in a clinical sample of out-patients, Groth-Marnat and Jeffs (2002) found that the strongest most robust predictor of dissociation was neuroticism whereas extraversion and conscientiousness were associated with low levels of dissociation. Similarly, Jaycox, Marshall, and Orlando (2003) when they examined predictors of acute distress in injured victims of physical assault, reported significant positive correlations between peritraumatic dissociation and neuroticism, as well as prior major depression, prior dysthymia, and injury severity. However, only neuroticism and injury severity were shown to be significant predictors of peritraumatic dissociation. Marshall and Schell (2002) used a cross-lagged panel analysis and found that neuroticism and injury severity were significantly related to peritraumatic dissociation as well as PTSD.
symptom severity in a sample of injured victims of physical assault. Holeva and Tarrier (2001) in a study of MVA survivors reported a significant positive correlation between neuroticism and peritraumatic dissociation. They proposed that the magnitude of recalled dissociative symptoms may be the result of high levels of neuroticism rather than the result of the trauma. This view is consistent with previous researchers who have suggested that the extent to which dissociation occurs is dependent on personality style (Goldberg, 1999: Spiegel & Greenleaf, 1992).

Female gender (Bryant & Harvey, 2003b; Christiansen & Elklit, 2008; Fullerton et al., 2001; Groth-Marnat & Jeffs, 2002; Olff et al., 2007), younger age (Fullerton et al., 2000; Groth-Marnat, Roberts, & Ollier, 1999; Irwin, 1994; Marmar, Weiss, Metzler, & Delucchi, 1996; Olff et al., 2007), and prior affective and anxiety disorders (Fullerton et al., 2000; Jaycox et al., 2003; Regambal & Alden, 2009; Wittmann et al., 2006) have been shown to be associated with increased peritraumatic dissociation but have also been identified as risk factors for PTSD. Similarly, trait dissociation has been found to independently predict peritraumatic dissociation (McCaslin et al., 2008; Engelhard et al., 2003). McCaslin and colleagues reported that peritraumatic dissociation partially mediated the relationship between trait dissociation and PTSD symptoms. Murray et al. (2002) found significant positive correlations between trait, peritraumatic, and persistent dissociation in a sample of MVA survivors, with each of the dissociation measures predicting PTSD. However, persistent dissociation displayed the strongest effect in predicting PTSD severity at 6-months post-MVA. Both Briere, Scott, and Weathers (2005) and Halligan, Michael, Clark, and Ehlers (2003) also found that persistent dissociation was predictive of PTSD beyond the influence of peritraumatic dissociation. These results indicate that upon trauma exposure a predisposition to dissociate
influences peritraumatic dissociation which contributes in part to the development of PTSD. However, the strongest dissociative influence is persistent dissociation which contributes to the maintenance of symptoms by impeding the access, integration, and resolution of trauma memories over time. Hence, the influences of trait and peritraumatic dissociation are reduced in the presence of persistent dissociation.

Controlling for variables that mediate or moderate the relationship between peritraumatic dissociation and PTSD has produced mixed results with some studies finding that peritraumatic dissociation remained an independent predictor of PTSD (Marmar et al., 1994; Shalev et al., 1996; Tichenor, Marmar, Weiss, Metzler, & Ronfeldt, 1996), whereas others have found that the influence of peritraumatic dissociation on PTSD was significantly reduced or completely diminished when other variables were taken into account (Engelhard et al., 2003; Gershuny et al., 2003; Holeva & Tarrier, 2001). In a recent review of prospective studies, van der Velden and Wittmann (2008) examined the independent predicative value of peritraumatic dissociation on chronic PTSD. They found that in most cases peritraumatic dissociation assessed within 4-weeks did not independently predict PTSD symptomatology assessed at least 3-months post-trauma. The authors questioned the predictive value of peritraumatic dissociation on PTSD since it may be confounded by other factors. They suggested that variables including initial psychological problems occurring days or weeks post-trauma are better predictors which may play a mediational role between peritraumatic dissociation and chronic PTSD.

A limited number of studies have examined the frequency of specific peritraumatic dissociative symptoms. Shalev et al. (1996), Fullerton et al. (2000), and Sterlini and
Bryant (2002), found that time distortion was the most frequently reported dissociative item using the Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Marmar, Weiss, & Metzler, 1997). The PDEQ is the most widely used measure of peritraumatic dissociation. It comprises 10 (psychoform) items and is presumed to be a unidimensional construct. Bryant et al. (2009) examined the factor structure of the PDEQ with initial results failing to confirm a single-factor structure. Subsequent exploratory and confirmatory factor analysis revealed an eight-item two-factor structure comprising altered awareness and derealisation. Cross-validation using a more traumatised sample (PDEQ mean scores 7.43 vs. 23.09) provided only partial support for the initial findings as four items were found to cross-load on each of the two factors. Regression analysis using data from the least traumatised sample showed that the derealisation factor was predictive of ASD, anxiety as well as depression. The authors proposed that peritraumatic dissociation may comprise two separate factors and acknowledged that further research is needed to ascertain which peritraumatic dissociative factors are pathologic and lead to PTSD.

2.2.8 Section summary

The evidence to date indicates that peritraumatic dissociation is a risk factor for PTSD. However, the nature of this relationship is far from simple; it appears to be a complex interaction between pre, peri, and post-trauma variables. Additionally, criticism of the use of predominantly psychoform measures, a lack of a clear conceptualisation of the construct, and subsequent variability in the assessment of different acute dissociative symptoms subsumed as a collective phenomenon, hinder any clear conclusions from being drawn. Furthermore, assessment of dissociative symptoms as a global construct prevents evaluation of specific factors. Bryant (2007) suggested that peritraumatic
dissociation needs to be deconstructed into particular factors (e.g., time distortion, derealisation, and reduced awareness) which would provide valuable information on specific dissociative mechanisms and associated psychopathology. One of the aims of the research reported in this thesis was to examine the factor structure of the PDEQ as well as the relationships between pre, peri, post-trauma dissociation and PTSD.

2.3 Post-Trauma Factors

Post-trauma factors such as coping (including the use of alcohol), disability, and social support play a significant role in the prediction and the maintenance of PTSD. However, in comparison to the assessment of pre and peri-traumatic characteristics, post-trauma factors have received considerably less attention. The role of social support as a protective factor against PTSD has received consistent empirical support particularly amongst combat samples. Meta-analyses examining predictors of PTSD have reported social support overall effect sizes of $r = -0.28$, (Ozer et al., 2003) and $r = -0.40$, (Brewin et al., 2000). Although having access to a social network provides the opportunity of immediate and extended positive support, availing oneself of, and actively seeking, such support is an adaptive coping strategy.

2.3.1 Coping

Lazarus and Folkman (1984) describe coping as a reaction to the process of evaluative appraisal, which determines both cognitive and behavioural reactions when internal and external demands exceed resources. In essence, when an event is perceived as stressful or threatening, strategies are utilised to reduce the stress and moderate the tension that accompanies it.
Although various typologies of coping exist, those proposed by Lazarus and Folkman (1984), which incorporate problem and emotion-focused coping, and more recently by Endler and Parker (1990), which emphasise avoidance-focused coping, have gained considerable empirical support. Lazarus and Folkman described problem-focused coping as problem solving or attempting to alter a situation in order to reduce the stress impact. Emotion-focused coping involves the regulation of emotional distress associated with a stressor being circumvented through intra-psychic activity such as re-appraisal, denial, and distancing. Additionally, the particular use of coping strategies are influenced by the nature of the stressor, personality factors and, depending on the context of the stressor, coping strategies may be combined (Lazarus, 1993).

Endler and Parker (1990) emphasised avoidance-focused coping, which encompasses a range of strategies that involve cognitive and behavioural activities that reduce the level of distress. These include distraction, wishful thinking, social diversion, and consumption of substances to alleviate stressor-related anxiety. They explained that although people have a preferred coping style (trait), their choice of style is governed by the interaction of situational factors (state). For example, when it is perceived that if something could be done to alter the stressor or change the situation, then problem-focused coping is utilised, and when an individual perceives that nothing can be done to change the situation, emotion-focused and/or avoidance-focused coping predominates.

Coping plays a significant role in posttraumatic adjustment since it mediates the relationship between trauma exposure and psychopathology (Jeavons et al., 2000; Joseph, Williams, & Yule, 1995; Keane, Marshall, & Taft, 2006; Steil & Ehlers, 2000). Ehlers and Clark’s (2000) cognitive model of PTSD emphasises the role of maladaptive
cognitive and behavioural coping strategies in maintaining PTSD symptoms. Avoidance strategies such as thought suppression, rumination, safety behaviours, persistent dissociation and the use of alcohol prevent emotional processing of the event. This interferes with the integration and restructuring of dysfunctional trauma cognitions (Clohessy & Ehlers, 1999; Dunmore et al., 2001; Ehlers et al., 1998; Steil & Ehlers, 2000). Evidence from a range of trauma populations, including survivors of natural disasters (Carr, et al., 1997), sexual abuse (Johnson, Sheahan, & Chard, 2004), physical and sexual assault (Dunmore et al., 2001), domestic violence (Street, Gibson, & Holohan 2005), burns (Lawrence & Fauerbach, 2003), and combat (Wolf, Keane, Kaloupek, Mora, & Wine, 1993), have shown that coping strategies which result in avoidance of the trauma experience, significantly contribute to poorer outcome and the chronicity of PTSD symptoms.

2.3.2 Coping and MVA’s
Jeavons et al. (2000) found that MVA survivors who relied on emotional and avoidance-focused coping displayed higher levels of trauma symptoms. They concluded that participants who avoid coming to terms with the accident may be more vulnerable to trauma symptoms. Bryant and Harvey’s (1995b) study showed that the main predictor of intrusion symptoms was avoidant coping style, which explained more variance than injury severity. Avoidant coping was also found to be the main predictor of PTSD symptom severity 6-months after hospital discharge in a sample that sustained severe traumatic brain injury (Bryant, Maroszeky, Crooks, Baguley, and Gurka, 2000). Nightingale and Williams (2000) found that avoidance/escape coping was most strongly predictive of PTSD. Similarly, Steil and Ehler’s (2000) results indicated that avoidance coping strategies were related to the severity of PTSD. They suggest that avoidance
strategies are likely to impede a change in maladaptive cognitions and prevent emotional processing of the traumatic experience, thereby obstructing the recovery process. Dougall et al. (2001) in a study with injured MVA survivors found that the use of the avoidance strategy ‘wishful thinking’ distinguished between participants with and without PTSD symptoms. They concluded that wishful thinking could be used to identify MVA survivors who are at risk of developing chronic posttraumatic stress.

When they assessed coping as a predisposing factor for PTSD following an MVA, Dorfel et al. (2008) found that the general coping strategies used in everyday life (trait), lack of situational control and avoidance, were associated with the development and maintenance of PTSD. Similar findings were shown from a prospective study in which trait coping was assessed 2-weeks before trauma exposure. Results showed that high levels of trait avoidance and trait emotion focused coping, as well as high levels of state avoidance coping and low levels of state problem focused coping, were predictive of PTSD at 6-months post-trauma (Gil, 2005). These findings show that trait coping styles are influential not only in the development and maintenance of PTSD but also influence post-trauma coping.

2.3.3 Section Summary

The use of avoidance coping pre and particularly post-trauma supports the findings of other trauma studies indicating that cognitive and behavioural strategies intended to distance or disengage from distressing reminders of the trauma are a central factor in maintaining symptoms. Avoidance strategies impede emotional processing of traumatic experience, reduce habituation to traumatic memories and interfere with integration and restructuring of dysfunctional of trauma cognitions (Ehlers & Clark, 2000; Foa,
Steketee & Rothbaum, 1989; Foa & Riggs, 1993; Horowitz, 1976; Jones & Barlow, 1990; van der Kolk & van der Hart, 1991). A component of the research reported in this thesis will be to examine the role of pre-trauma coping on the development and maintenance of PTSD.

2.3.4 Alcohol

Alcohol and other substances that act as depressants on the central nervous system (CNS) are common among individuals with PTSD (Jacobsen, Southwick & Kosten, 2001). Self-medication with alcohol is an effective short-term coping mechanism in reducing PTSD symptoms. It acts as a CNS depressant and provides a feeling of distraction, its anxiolytic properties ameliorate the effects of anxiety, reducing muscle tension, and it decreases the severity and number of nightmares by suppressing REM sleep (Blanchard, Magee, Veniegas, & Blanchard, 1993; Jelinek & Williams, 1984). However, long-term alcohol abuse often leads to physiological dependence and associated problems such as social isolation, depression, irritability, and anger (APA, 2000). Individuals with PTSD and comorbid alcohol use disorders are more likely to have poor social skills, occupational difficulties and experience financial problems which can hinder accessing treatment (Mills, Teesson, Ross, & Peters, 2006). Moreover, the continual avoidance of traumatic memories through the use of alcohol exacerbates PTSD symptoms, adds considerably to the chronicity of the disorder, and reduces the effectiveness of treatment (Peterson, Prout, & Schwarz, 1991; Watts & de L Horne, 1994).

General population lifetime prevalence rates of substance abuse disorders range from 8% to 24% for people without PTSD, with estimations for individuals with PTSD
ranging between 21% to 43% (Breslau et al., 1991; Breslau et al., 1997; Kessler et al., 1995). Similarly, findings from the Australian national survey of mental health and well-being reported comorbid PTSD and alcohol abuse/dependence rates of 37.2% for males and 12.4% for females (Creamer et al., 2001).

Research on Vietnam veterans has found that in addition to comorbid depression, many veterans also displayed high levels of comorbid substance abuse, particularly alcohol (McCann & Pearlman, 1990; Niles, 1991). Green (1994) and Green, Lindy, Grace and Leonard (1992) reported that comorbidity (depression and substance abuse) was common, evident in over three-quarters of the Vietnam veterans sampled. Research on both combat veterans and civilians has found that alcohol abuse or dependence is the most common comorbid condition for men with PTSD, followed by comorbid depression. Rates of comorbidity among women showed that depression is most common followed by alcohol abuse (Kessler, et al., 1995; Kulka et al., 1990). Green (2003), who examined PTSD symptom profiles in men and women, reported that men were significantly more likely to consume excessive amounts of alcohol compared to women.

It is commonly recognised that the use of alcohol compromises driving ability, which consequently increases the risk of being involved in an MVA (Bloom, 1998; Vingilis, Larkin, Stoduto, Parkinson-Heyes, & McLellan, 1996; Vingilis, Stoduto, Macartney-Filgate, Liban & McLellan, 1994). In 2010, the use of alcohol was attributed to at least 19% of MVA related deaths on NSW roads (NSW Roads & Traffic Authority, (RTA), 2011). Rosman, Ferrante, and Marom, (2001), who examined the association between drink driving and MVAs in Western Australia, reported that drink-driving crashes
tended to be more severe than those that did not involve the use of alcohol. They found that serious MVAs involving fatalities or hospitalisations accounted for 20% of alcohol-related crashes.

Few MVA studies have examined the use of alcohol on post-accident adjustment. Hickling and Blanchard (1992) reported that only one MVA survivor in a sample of 20 developed alcohol and substance abuse as a result their MVA. Blanchard, Hickling, Barton, et al. (1996) assessed a sample \((N = 158)\) of MVA survivors and found that alcohol abuse, assessed 1 to 4-months post-MVA, accounted for 32% of the variance in posttraumatic stress symptoms at 12-months.

An Australian study conducted by Bryant and Harvey (1995a) on a sample of MVA survivors \((N = 56)\), found that substance abuse (including alcohol) was evident in 37% of participants who reported significant psychological impairment \((n = 23)\) 12-months post-accident. More recently, McFarlane et al. (2009) investigated pre and post-trauma alcohol consumption and risk for PTSD in a sample \((N = 1045)\) of hospitalised injury survivors comprising 64% MVA. Results showed that moderate levels of alcohol consumption (up to 4 glasses a week) prior to and post-trauma had a protective role, predicting lower levels of psychopathology (depression, anxiety, and PTSD symptoms) at 1 week and 3-months. In a sub-group of participants, PTSD predicted alcohol abuse and those participants who reported dependence and problem drinking generally experienced greater symptomatology. Conversely, minimal drinking was also associated with PTSD. Though they require further replication, these findings indicate that the moderate use of alcohol without dependence provides pharmacological benefits in relieving the effects of initial distress and reducing symptom development. Conversely,
minimal consumption may increase risk as it removes the opportunity of self-
medication in coping with the effects of the trauma. However, excessive consumption
contributes to poorer outcomes as an over reliance on self-medication prevents adequate
processing of trauma related memories.

Blanchard et al. (1994) found a complete absence of alcohol abuse in a sample \((N = 50)\)
of MVA survivors in which 23 participants had PTSD and comorbid depression. Mayou
et al. (1997), in a follow-up study of PTSD assessed at five-years post-accident, found
no effect of alcohol use either prior to the MVA or at 12-months. More recently,
Blanchard et al. (2004) examined psychological morbidity in two MVA samples.
Sample one comprised participants who had been involved in a MVA within the last 2-
years and were seeking psychological treatment. Results showed that 2\% of participants
with PTSD reported current alcohol or drug abuse and 17.6\% reported lifetime alcohol
or drug abuse. No significant differences on current or lifetime abuse were found
between those with and without PTSD. Sample two comprised hospital attendees who
were re-assessed at 12-months post-accident. Results showed that 10.5\% of participants
with PTSD reported current alcohol or drug abuse and 5.3\% reported lifetime alcohol or
drug abuse. Similar to the first sample, no significant differences were found on alcohol
measures between MVA survivors with PTSD, those who had remitted, and those who
had sub-threshold PTSD/no PTSD.

In examining the results on post-trauma alcohol use there is no clear single explanation
to account for the mixed findings. Results that have shown no effect of alcohol between
participants with and without PTSD may indicate that for some participants trauma
alone (without PTSD) may lead to alcohol abuse (McFarlane, 1998). Additionally, it is
also important to note that the effects of alcohol may mask symptoms of PTSD, which makes diagnosis more difficult. Alcohol use is a contributing causal factor in the occurrence of MVAs. However, the finding that post-accident alcohol abuse is more likely to occur in MVA survivors who display high levels of psychological impairment may not necessarily be attributed to a causal relationship between alcohol abuse and the occurrence MVAs (Vingilis et al., 1994). For example, unless pre-trauma alcohol abuse is assessed, temporal causality cannot be determined, that is, whether alcohol abuse was pre-existing or secondary to PTSD.

Alternatively, alcohol abuse may be attributed to the degree of post-accident morbidity as individuals with PTSD and comorbid alcohol use disorders suffer from more severe avoidance and arousal symptoms compared to individuals with PTSD alone (Saladin, Brady, Dansky & Kilpatrick, 1995). Moreover, post-MVA alcohol abuse and dependence may be an avoidance coping strategy used to alleviate the symptoms associated with the unpleasant traumatising experience, in which case the onset of alcohol abuse may parallel the onset and, perhaps more importantly, the severity and chronicity of PTSD symptoms (Bremner, Southwick, Darnell, & Charney, 1996). However, if assessment is conducted at early post-trauma then detection of comorbid relationships may not always be apparent.

2.3.5 Section Summary

The occurrence rate of comorbid PTSD and alcohol abuse is concerning. Alcohol has been identified a contributing factor in the occurrence of MVAs but it is often assumed that alcohol abuse occurs secondary as a form of maladaptive post-trauma coping. Identified as a contributing factor in the chronicity of PTSD, it is important to assess the
use of alcohol post-trauma to help circumvent ongoing psychological problems. Similarly, it is also important to take into account pre-trauma alcohol use as post-trauma assessment is likely to be influenced by previous drinking patterns. For these reasons it is important to determine if alcohol abuse contributes to post-trauma psychopathology after controlling for pre-trauma alcohol use, which is one of the aims of the research reported in this thesis.

2.3.6 Physical Disability

As outlined in section 2.2.3, appraisal of injury severity plays an important role on post-MVA adjustment. Perhaps equally important, though with less empirical support, is assessment of the longer-term implications of injury. For some MVA survivors physical disability is permanent, therefore learning to cope with and adapt to physical change, including ongoing pain, represents a significant challenge. For some MVA survivors these post-MVA stressors exceed coping resources. Similarly, the appraisal and the personal meaning of disability and how it impacts on quality of life also play an important role in post-trauma adjustment. Within the MVA trauma literature the assessment of disability has been in association with the maintenance of symptoms and long-term morbidity.

When they assessed one-year outcomes in MVA survivors, Mayou and Bryant (2001) noted that in participants’ verbal and written responses, musculoskeletal complaints were reported as restricting and a cause of frustration in participants whose leisure or work activities were physically demanding. They suggested that psychiatric complications affect the perception of pain and physical impairment and that physical impairment contributes to the maintenance of PTSD symptoms. Mayou et al. (1997)
found that physical problems, pain and disability, when assessed five-years post-accident, acted as reminders of the event and contributed significantly to long term maintenance of symptoms. Mayou et al. (2001) found that persistent health problems at 3-months were predictive of psychopathology at one-year post-MVA. The authors noted that anxiety and financial difficulties are common and that ongoing physical problems as well as disability restrict not only everyday activities but also longer-term ambitions. Ehlers et al. (1998) found that persistent physical and financial problems following an MVA presented an increased risk for PTSD and symptom severity at one-year post-accident.

More recently Barth et al. (2005) reported that ongoing physical impairments place considerable demands on coping resources, finding that at 5-years post-MVA no participants with PTSD had fully recovered from physical injury. In a sample of severely injured MVA survivors, persistent physical disability was predictive of PTSD at 6-months post-accident. Physical disabilities were significant secondary stressors and a risk factor for chronic PTSD, hindering daily functioning in areas including occupation, income, and relationships (Hamanaka et al., 2006). Jenewein et al. (2009) in a sample of severely injured accident survivors comprising 59% MVA, found that at 3-years post-accident individuals who were experiencing chronic pain had significantly more PTSD symptoms, depression and more disability and spent more days away from work. Similarly, Clapp, Masci, Bennett, and Beck (2009), when they examined physical and psychosocial functioning following MVA, found that pain severity was strongly associated with functional impairment.
Ehlers and Clark (2000) suggested that negative appraisals of trauma sequelae, including chronic pain, physical disability, impaired occupational and social functioning, serve to maintain symptoms since the emotions associated with negative appraisals promote dysfunctional coping strategies which in turn prevent cognitive change. Matthews et al. (2009) who recruited a sample of hospitalised injury survivors comprising 59% MVA, found significant negative correlations between avoidance coping, negative appraisals of the self and the world, and work potential. After controlling for PTSD symptoms, negative appraisals were associated with poor adjustment and poor work potential, which represented a significant barrier in returning to work following injury. O’Donnell et al. (2004) found that appraisal of the anticipated impact of injury was associated with PTSD and comorbid depression at 12-months post-injury. Similarly, appraisals and fear of pain contribute to injury-related avoidance activities whereby the avoidance of movement and other activities can lead to, as well as enhance, the perception of disability (Sharp & Harvey, 2001). Furthermore, Waddell, Newton, Henderson, Somerville, and Main (1993) reported that the strongest predictor of disability was pain-related fear-avoidance, that is, fear of pain and avoidance of pain-related activity. Pain and disability, in addition to causing discomfort and distress, also act as reminders of the trauma, and trigger arousal and re-experiencing symptoms. Avoidance of distressing intrusive reminders in turn escalates distress and avoidance, which maintains symptoms (Blanchard et al., 1997; Ehlers and Clark, 2000; Kennedy, Duff, & Beedie, 2003; Martz, 2004). One of the aims of the research reported in this thesis was to examine the role of physical disability in maintaining PTSD.
2.3.7 Section Summary

Physical disability is an unfortunate yet common outcome of experiencing an MVA. For many MVA survivors the implications of physical disability affect various facets of everyday life. Coming to terms and coping with disability is strongly influenced by personal meaning and subsequent appraisals. For some MVA survivors these appraisals are negative which has been shown to have long-term implications particularly in relation to psychological recovery. An aim of the research reported in this thesis was to examine the influence of physical disability in relation to the maintenance of PTSD and depressive symptoms.

2.4 Chapter Summary

This chapter has reviewed a range of pre, peri, and post-traumatic predisposing and vulnerability factors associated with the development and maintenance of PTSD. Identifying variables that pose an increased risk of PTSD may help prevent undue post-MVA distress. Similarly, given that PTSD has been diagnosed in MVA survivors up to five-years post-accident, recognition of variables that maintain the disorder will no doubt help in reducing the long term burden on individual and community resources through identification and earlier psychological intervention.

Female gender, younger age, having experienced past emotional problems, and having a neurotic predisposition are pre-trauma variables which have been identified as influential in the development of PTSD. However, findings have been somewhat inconsistent and different causal explanations have been sought to describe the relationship between pre-trauma factors and PTSD. According to Ehlers and Clark’s (2000) cognitive model, pre-trauma variables are associated with PTSD as they
influence cognitive processing, trauma appraisals and coping strategies used to control perceived threat. Meta-analyses have reported significant though small effect sizes for pre-trauma factors in predicting PTSD. It has been suggested that aggregating pre-trauma factors may produce stronger effect sizes and that the effects of pre-trauma variables may be mediated by either peri-traumatic and/or post-trauma variables.

Subjective emotional fear responses, peritraumatic dissociative reactions and injury severity as well as accident severity (event characteristics) have received considerable empirical support which has highlighted the important role of subjective experience in trauma exposure in the prediction of PTSD. For example, assessment of event characteristics, particularly injury severity, provides the strongest effect when it incorporates subjective appraisals. Though peri-traumatic variables (particularly dissociation) have generally shown strong associations with the development and maintenance of PTSD, recent findings have questioned the predictive value and assessment of peritraumatic dissociation as well as the utility of a fear response in predicting PTSD. It has been suggested that the relationship between acute dissociative reactions and PTSD may not be linear and that the assessment of peritraumatic dissociation as a global construct prevents evaluation of specific dissociative factors. Though peritraumatic dissociation has been identified as a risk factor for PTSD, the nature of this relationship is far from simple and appears to be a complex interaction between pre, peri, and post-trauma factors.

Post-trauma coping strategies play an integral role in the relationship between trauma exposure and psychopathology. The use of maladaptive cognitive and behavioural coping strategies has been found to maintain PTSD symptoms. In particular, emotion-
focused and avoidance coping strategies, persistent dissociation and the use of alcohol prevent emotional processing of the event, which interferes with the integration and restructuring of dysfunctional trauma cognitions.

Sustaining injury, particularly serious injury, is not uncommon following an MVA. Learning to cope with and adapt to physical changes can present significant challenges. Appraisal of the physical implications and disability play an important role in post-trauma adjustment as negative appraisal of disability maintains symptoms by preventing cognitive change. The personal meaning that is assigned to disability rather than the disability itself, influences coping strategies and subsequent adjustment over the longer term.

Variations in study design, particularly cross-sectional and retrospective measures of pre-morbid and peritraumatic variables, somewhat limits the strength of causal conclusions as current psychological state is likely to influence subjective recall. However, research using MVA samples is retrospective and, unlike combat samples for example, accurately measuring pre-trauma variables before the event poses significant logistical and methodological problems given the unpredictable and frequent occurrence of MVAs. Variation in assessment time-frames, the use of different psychometric instruments and demographic variations all influence to some extent the outcome and interaction of variables of interest as well as the strength of conclusions. Notwithstanding methodological variations, from a clinical perspective there can be no doubt that the identification of factors that aid in the recognition of MVA survivors who are at risk of developing PTSD is extremely important. Early identification and treatment intervention in the initial stage of symptom onset can help circumvent
chronicity of the disorder and comorbidity. Motor vehicle accidents will continue to occur in Australia as in most areas of the world. Recognition and awareness of the psychological impact of MVAs and procedural screening during hospitalisation as well as psychological aftercare would help to reduce the burden on individuals, families and societies at large.
Chapter 3

3.1 Motor Vehicle Accidents

Accidents, injury, and fatalities are adverse consequences of road transport activity. In 1896 the first recorded MVA death occurred in London when a 44-year old female passenger died after the vehicle she was travelling in was hit by another vehicle (RoadPeace, n. d). In 2009 it was estimated that (globally) 1.2 million MVA deaths occur annually with up to 50 million MVA survivors who sustained injury. Motor vehicle accidents were the 8th leading cause of death in 2004, and by 2030 are predicted to become that 5th leading cause. The annual financial losses associated with MVAs are estimated at 518 billion (U.S.) dollars (World Health Organisation, 2009).

Since 1925 when MVA statistics started to be recorded in Australia there have been over 178,000 MVA related deaths. In 2010, 1,352 people died on Australian roads. In NSW there were 42,299 reported MVAs with 405 deaths and 24,623 individuals sustained physical injury. The estimated annual cost associated with MVAs is 18 billion dollars nationally with NSW accounting for approximately 3.7 billion (Department of Infrastructure, Transport, Regional Development and Local Government, 2009; RTA NSW, 2011).

In addition to the financial costs, damage to property and infrastructure, physical injuries, as well as physical disability, MVA survivors often experience a range of psychological responses. Given the ubiquitous nature and potential severity of MVAs it is not surprising to find that some individuals may experience psychological distress. Motor vehicle
accidents have been consistently found to be one of, if not the most common traumatic occurrences an individual may experience (Bull, 1979; Norris, 1992; Richter, 1981).

The potential of severe MVAs to elicit a stress response of significant magnitude is recognised in the PTSD diagnostic features in the DSM-IV (APA, 1994). Prior to this change in diagnostic criteria, MVAs received comparatively little empirical investigation as they did not “lie outside the usual range of human experience”.

Research on the psychological impact of experiencing a MVA over the past 20-years has provided a wealth of information which has helped shape current understanding. Some of the earlier studies conducted (2nd and 3rd quarter) last century on injury samples that included MVA survivors were often associated with examination of psycho-neurological aspects of head injury, a neurotic predisposition and post-accident neurosis. Various forms of neurosis including anxiety, hysterical, phobic, and depressive forms as well as malingering and compensation neurosis were assessed post-injury (Culpan & Taylor, 1973; Miller, 1961; Ruesch & Bowran, 1945).

As noted in the previous chapter, irrespective of the severity of the accident, the experience of an MVA can elicit a range of psychological responses. For example, emotional responses such as guilt, anger, frustration, grief, sadness, and fear. For most MVA survivors these responses will be transient and dissipate over time as individuals come to terms with their experience of being involved in a MVA. For others ongoing fear and anxiety associated with travelling in a motor vehicle, fear of being involved in another accident, apprehension and avoidance of driving as well as avoidance of the accident scene can continue long after the MVA has occurred. Accident phobias
(specific and generalised MVA-related anxiety), also referred to as driving and travel phobia, have been documented by several researchers who found that up to 5-years post-accident MVA survivors suffered from severe and disabling phobic anxiety (Blanchard, Hickling, Taylor, et al., 1995; Lucas, 2003; Mayou, Simkin, & Threlfall, 1991; Taylor & Deane, 2000; Taylor & Koch, 1995).

3.2 Incidence of PTSD in MVA Samples

It is not uncommon for MVA survivors to experience both acute and chronic psychological problems, the most common of which is PTSD (Blanchard & Veazey, 2001). In a review of mental disorders following MVAs, Blanchard and Veazey reported that on average approximately 24% of MVA survivors at 3-months and 15% at 12-months post-MVA met DSM-IV diagnostic criteria for PTSD. These PTSD rates are similar to those in Table 3.1, illustrating the incidence of PTSD from a number of studies dating back to 1991, showing a weighted average of 27% of participants at 1-month and 16% at 12-months post-accident suffered from PTSD.

The PTSD percentage rates from the studies shown in Table 3.1 highlight the considerable variability between studies, even when those (noted) studies that included multiple traumas are excluded. For example, Green et al. (1993) and Nishi et al. (2010) reported that, at 1-month post-MVA, 8% of participants had developed PTSD compared to 61% reported by Dougall et al. (2001). Percentage rate variations are also evident at 3-months, 8% vs. 44% (Mayou et al., 1997; Blanchard et al., 1997), 6-months, 9% vs. 45% (Ehring, Ehlers, Cleare, et al., 2008; Dougall et al., 2001), and at 12-months, 7% vs. 46% (Jeavons & Greenwood 2000; Bryant & Harvey, 1995b).
### Table 3.1

**PTSD Incidence Rates in MVA Samples**

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<tr>
<th>Authors</th>
<th>Population</th>
<th>N</th>
<th>Diagnostic Instrument</th>
<th>% PTSD in Months Post-MVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Barth et al. (2005)</td>
<td>Hospital attendees</td>
<td>70</td>
<td>DIA-X</td>
<td>-</td>
</tr>
<tr>
<td>Blanchard et al. (1996)</td>
<td>Treatment seeking or self-referred</td>
<td>158</td>
<td>CAPS</td>
<td>39</td>
</tr>
<tr>
<td>Blanchard et al. (1997)</td>
<td>Seeking medical attention</td>
<td>145</td>
<td>CAPS</td>
<td>54</td>
</tr>
<tr>
<td>Blanchard et al. (2003)</td>
<td>Treatment-seeking</td>
<td>75</td>
<td>CAPS</td>
<td>-</td>
</tr>
<tr>
<td>Bryant &amp; Harvey (1995b)</td>
<td>Hospital attendees</td>
<td>56</td>
<td>IES</td>
<td>-</td>
</tr>
<tr>
<td>Coronas et al. (2008)</td>
<td>Hospital attendees</td>
<td>60</td>
<td>SCID</td>
<td>-</td>
</tr>
<tr>
<td>Creamer et al. (2004)</td>
<td>Hospital attendees</td>
<td>307b</td>
<td>CAPS</td>
<td>-</td>
</tr>
<tr>
<td>Delahanty et al. (2003)</td>
<td>Hospital attendees</td>
<td>55</td>
<td>SCID</td>
<td>16</td>
</tr>
<tr>
<td>Dougall et al. (2001)</td>
<td>Hospital attendees or police referral</td>
<td>108</td>
<td>SCID</td>
<td>61</td>
</tr>
<tr>
<td>Ehlers et al. (1998)</td>
<td>Hospital attendees</td>
<td>967</td>
<td>PSS</td>
<td>-</td>
</tr>
<tr>
<td>Ehring et al. (2008)</td>
<td>Hospital attendees</td>
<td>53</td>
<td>SCID</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note. CAPS = Clinician-Administered PTSD Scale. DIA-X = Diagnostic Expert System for DSM-IV. IES = Impact of Event Scale. PSS = Posttraumatic Stress Symptom Scale. SCID = Structured Clinical Interview for PTSD.  
a assessed at 8-weeks.  
b sample comprised 74% MVA survivors  
c Follow up study conducted by Mayou et al. (2002).
### PTSD Incidence Rates in MVA Samples (continued)

<table>
<thead>
<tr>
<th>Authors</th>
<th>Population</th>
<th>N</th>
<th>Diagnostic Instrument</th>
<th>% PTSD in Months Post-MVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Feinstein &amp; Dolan (1991)</td>
<td>Hospital attendees</td>
<td>48&lt;sup&gt;d&lt;/sup&gt;</td>
<td>CIS</td>
<td>25&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td>Frommberger et al. (1998)</td>
<td>Hospital attendees</td>
<td>179</td>
<td>PSS</td>
<td>-</td>
</tr>
<tr>
<td>Green et al. (1993)</td>
<td>Hospital attendees</td>
<td>24</td>
<td>DIS</td>
<td>8</td>
</tr>
<tr>
<td>Hamanaka et al. (2006)</td>
<td>Hospital attendees</td>
<td>100</td>
<td>SCID</td>
<td>-</td>
</tr>
<tr>
<td>Harvey &amp; Bryant (1998)</td>
<td>Hospital attendees</td>
<td>92</td>
<td>CIDI</td>
<td>-</td>
</tr>
<tr>
<td>Hepp et al. (2008)</td>
<td>Hospital attendees</td>
<td>90&lt;sup&gt;f&lt;/sup&gt;</td>
<td>CAPS</td>
<td>-</td>
</tr>
<tr>
<td>Irish et al. (2008)</td>
<td>Hospital attendees</td>
<td>188</td>
<td>CAPS</td>
<td>12&lt;sup&gt;g&lt;/sup&gt;</td>
</tr>
<tr>
<td>Jeavons &amp; Greenwood (2000)</td>
<td>Hospital attendees</td>
<td>72</td>
<td>PTSD-1</td>
<td>24</td>
</tr>
<tr>
<td>Jones et al. (2007)</td>
<td>Hospital attendees</td>
<td>131</td>
<td>PSS</td>
<td>30&lt;sup&gt;h&lt;/sup&gt;</td>
</tr>
<tr>
<td>Koren et al. (1999)</td>
<td>Hospital attendees</td>
<td>74</td>
<td>SCID</td>
<td>-</td>
</tr>
<tr>
<td>Koren et al. (2002)</td>
<td>Hospital attendees</td>
<td>102</td>
<td>SCID</td>
<td>-</td>
</tr>
<tr>
<td>Matthews et al. (2001)</td>
<td>Hospital attendees</td>
<td>40</td>
<td>CIDI</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note.* CAPS = Clinician-Administered PTSD Scale, CIDI = Composite International diagnostic Interview, CIS = Clinical Interview Schedule, DIS = Diagnostic Interview Schedule, PSS = Posttraumatic Stress Symptom Scale, PTSD-I = Post Traumatic Stress Disorder Interview, SCID = Structured Clinical Interview for PTSD.

<sup>d</sup> sample comprised 56% MVA survivors.  <sup>e</sup> assessed at 6-weeks.  <sup>f</sup> sample comprised 59% MVA survivors.  <sup>g</sup> assessed at 6-weeks.  <sup>h</sup> assessed at 6-weeks.
### PTSD Incidence Rates in MVA Samples (continued)

<table>
<thead>
<tr>
<th>Authors</th>
<th>Population</th>
<th>N</th>
<th>Diagnostic Instrument</th>
<th>% PTSD in Months Post-MVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Mayou et al. (1997)</td>
<td>Hospital attendees</td>
<td>111</td>
<td>PSS</td>
<td></td>
</tr>
<tr>
<td>Mayou &amp; Bryant (2001)</td>
<td>Hospital attendees</td>
<td>1148</td>
<td>PSS</td>
<td></td>
</tr>
<tr>
<td>Mayou &amp; Bryant (2002)</td>
<td>Hospital attendees</td>
<td>507</td>
<td>PSS</td>
<td></td>
</tr>
<tr>
<td>Murray et al. (2002)</td>
<td>Hospital attendees</td>
<td>27 (176)</td>
<td>PDS</td>
<td>32 (28)</td>
</tr>
<tr>
<td>Nishi et al. (2010)</td>
<td>Hospital attendees</td>
<td>64</td>
<td>CAPS</td>
<td>8</td>
</tr>
<tr>
<td>O’Donnell et al. (2004)</td>
<td>Hospital attendees</td>
<td>363</td>
<td>CAPS</td>
<td>-</td>
</tr>
<tr>
<td>Schnyder et al. (2001)</td>
<td>Hospital attendees</td>
<td>106</td>
<td>CAPS</td>
<td>-</td>
</tr>
<tr>
<td>Shalev et al. (1998)</td>
<td>Hospital attendees</td>
<td>211</td>
<td>CAPS</td>
<td>30</td>
</tr>
<tr>
<td>Smith et al. (2007)</td>
<td>Hospital attendees</td>
<td>50</td>
<td>SRS-PTSD</td>
<td>14</td>
</tr>
<tr>
<td>Ursano et al. (1999)</td>
<td>Hospital attendees or police referral</td>
<td>122</td>
<td>SCID</td>
<td>34</td>
</tr>
<tr>
<td>Vaiva et al. (2003)</td>
<td>Hospital attendees</td>
<td>123</td>
<td>CAPS</td>
<td>51</td>
</tr>
</tbody>
</table>

**Note.** CAPS = Clinician-Administered PTSD Scale, PDS = Posttraumatic Diagnostic Scale, PSS = Posttraumatic Stress Symptom Scale, SCID = Structured Clinical Interview for PTSD, SRS-PTSD = Self-Rating Scale for PTSD.

1 Results from an initial study Mayou et al. (1993). 1 Study was conducted with inpatient and out-patients, numbers in parentheses represent out-patient results. 1 sample comprised 74% MVA survivors. 1 sample comprised 60% MVA survivors. 1 sample comprised 85% MVA survivors. assessed at 16-weeks. assessed at 16-weeks. assessed at 8-weeks.
The disparity of PTSD percentage rates between MVA samples may be influenced by methodological differences. For example, participants in the study by Dougall et al. (2001) who had either threshold or sub-threshold diagnoses of PTSD were combined into one group. This gave over-inflated percentage rates of 61% and 45% at 1 and 3-months respectively. The high percentage rate (50%) reported by Coronas et al. (2008) at 3-months may have been influenced by the recruitment procedure since half of the sample had either sought psychological help or were thought to be experiencing post-trauma psychological problems. The use of different psychometric instruments used to determine PTSD may also influence incidence rates as the use of questionnaires as opposed to diagnostic interview limits the reliability of classification.

Additionally, the use of different diagnostic criteria (DSM III-R vs. DSM-IV), study design (cross-sectional vs. longitudinal), sample characteristics (demographic differences), as well as trauma characteristics (accident and injury severity) may also influence PTSD incidence rates. Furthermore, methodological variations in the assessment and conceptualisation of variables associated with the development and maintenance of PTSD (e.g., accident severity) make direct comparisons between studies problematic.

In addition to showing the percentage variations of PTSD, Table 3.1 also illustrates a general trend of symptom remission over time. This is also evident when the weighted average percentage rates of PTSD for 1 (27 %), 3 (20%), 6 (15%), and 12-month (16%) time periods are examined. However, the course of PTSD can vary, symptoms may fluctuate over time, and reactivation (including Delayed Onset) may occur in response to reminders of the trauma. A protracted course can also occur with some MVA survivors suffering from PTSD 5-years post-accident. This highlights the importance of early
identification and treatment intervention. One of the aims of this research is to assess the incidence of PTSD and to examine variables related to its development and maintenance.

### 3.3 Comorbidity

As was reviewed in section 1.6.2, PTSD is a highly comorbid condition. Predominantly, major depression is the most frequently occurring comorbid condition with PTSD, followed by other anxiety disorders (Bursztajn, et al., 1995; Davidson & Fairbank, 1993; Green, 1994). Blanchard and Veazey (2001) estimated that approximately 40% of MVA survivors with PTSD also experience comorbid depression. Similarly, examination of a number of MVA studies shown in Table 3.2 revealed a PTSD comorbid depression weighted average of 41.57%.

Several MVA studies have found that participants with PTSD had significantly higher depression scores than participants without PTSD (Barth et al., 2005; Frommberger et al., 1998; Hepp et al., 2008; Karl et al., 2009; Kupchik et al., 2007; Shalev et al., 1996). Though depression and related symptoms are frequently reported among trauma survivors, for individuals with PTSD, depression is more common and depressive symptoms appear to be more intense than among survivors without PTSD.

Shalev et al. (1998) found, in a sample comprising 85% MVA survivors, that PTSD and depression comorbidity was associated with greater symptom severity and lower levels of functioning. Similarly, Blanchard, Buckley et al. (1998) reported that the co-occurrence of PTSD and depression in MVA survivors resulted in greater role impairment, less remission of symptoms and increased (subjective) distress in comparison to participants with a single diagnosis of PTSD.
Table 3.2

**Comorbid Incidence rates of PTSD and Depression in MVA Samples**

<table>
<thead>
<tr>
<th>Authors</th>
<th>N</th>
<th>Percentage of PTSD &amp; Comorbid Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blanchard, Buckley et al. (1998)</td>
<td>107</td>
<td>53%</td>
</tr>
<tr>
<td>Blanchard et al. (1994)</td>
<td>50</td>
<td>48%</td>
</tr>
<tr>
<td>Blanchard et al. (1995)</td>
<td>158</td>
<td>53%</td>
</tr>
<tr>
<td>Blanchard et al. (2004)</td>
<td>132</td>
<td>53%</td>
</tr>
<tr>
<td>Frommberger et al. (1998)</td>
<td>152</td>
<td>39%</td>
</tr>
<tr>
<td>Kuch et al. (1995)</td>
<td>54</td>
<td>41%</td>
</tr>
</tbody>
</table>

Depression has also been identified as a significant predictor of PTSD. Dorfel et al. (2008) reported that depression was predictive of avoidance and hyperarousal symptoms. Freedman et al.’s (1999) results showed that depressive symptoms assessed at 1-week post-MVA were predictive of PTSD at 4-months and also at 12-months post-accident. Similarly, Irish et al. (2008) found that depression assessed at 6-weeks post-MVA was predictive of PTSD symptoms at 12-months post-accident.

The findings on the role of depression present a multifaceted influence. Prior depression is a risk factor for both PTSD and depression post-trauma (Blanchard et al., 1994; Shalev et al., 1998). A number of studies have reported that depression occurred secondary to PTSD (Ginzburg et al., 2010; Wittmann et al., 2008). However, this is not always the case (Blanchard, Buckley et al., 1998). Some studies have found that, although highly correlated, PTSD and depression are independent sequelae that interact in a reciprocal manner which increased the level of distress and dysfunction (Blanchard, Buckley et al.,
1998; Shalev et al., 1998; Wolf, King, King, & Sharkansky, 2001). Whereas others have reported that at 12-months post-trauma, PTSD and comorbid depression was indistinguishable and represented a shared vulnerability with the same predictor variables (O’Donnell et al., 2004). One aim of the research reported in this thesis was to examine the influence of depression in relation to the development and chronicity of PTSD and to assess the incidence of comorbidity over time.

3.4 Chapter Summary

Motor vehicle accidents are an unexpected yet common occurrence. Since the late 19th century the number of MVA related fatalities has increased significantly with an estimated 1.2 million deaths occurring annually. The financial cost associated with MVAs equates to many billions of dollars each year. Physical injury and disability are unfortunate consequences of MVAs, which for some survivors are often permanent.

The experience of an MVA can elicit a range of psychological responses. For most survivors these responses are transient, though for others psychological responses and associated problems can persist long after the MVA has occurred. Posttraumatic Stress Disorder is the most frequent psychological sequelae following an MVA, with approximately 24% of MVA survivors suffering from PTSD at 3-months and 10% at 5-years post-accident. In addition to PTSD, concurrent depression is also common; approximately 40% of PTSD sufferers also experience comorbid depression.
Chapter 4

4. General Aims of the Study

The overall aim of the research presented in this thesis was to examine the role of pre-trauma, peritraumatic, and post-trauma characteristics on post-MVA psychopathology. A further aim was to examine the inter-relationships between variables which influence the development and maintenance of PTSD. Additionally, this research was intended to provide empirical support for aspects of contemporary cognitive PTSD models. This thesis comprises a 4-phase longitudinal study on MVA survivors assessed at approximately 1, 3, 6, and 9-months post-accident (see Table 4.1). The following section describes the methodology including participant recruitment, procedure, and materials used in each of the phases.

4.1 Phase 1 Methodology

4.1.2 Participants

Participants comprised members of the general community who had recently survived an MVA (as a driver or passenger of a motor vehicle, motorcyclist or pillion passenger, pedal cyclist or pedestrian) and attended the Accident and Emergency department one or another of two local general hospitals. As shown in Table 4.1 the Phase 1 sample comprised 157 males (46.6%) and 180 females (53.4%) who ranged from 18 to 88 years of age. Additional sample information including hospital attendance type (see Table 5.1) and detailed demographic information (see section 5.2.1) are described in the following chapter.
4.1.3 Materials

The Phase 1 survey was a specifically designed 4-page self-report package which was deliberately kept short to increase the response rate. Participants were asked to provide demographic information, including date of birth, gender, yearly gross income and educational attainment. Additional required information comprised accident status (e.g., driver, passenger, motorcyclist/pillion passenger, pedal cyclist, or pedestrian), date of the accident and survey completion date. Subjective measures of accident severity (1 = very slight to 5 = very severe), injury severity (1 = none to 5 = life threatening), and fear of dying (1 = not at all to 5 = certain I would die) were rated using a 5-point likert format. Post accident distress comprising; sad or depressed and anxious or fearful, and pre-morbid status: feeling depressed or anxious (in previous 4-weeks) was rated on a 5-point likert scale, (1 = not at all to 5 = extremely). These unstandardised questions (initial emotional responses) were used rather than standardised scales to reduce the length of the survey to improve the response rate. Participants were also asked to indicate if they had been involved in any other MVAs and, if so, how many, as well as rating this MVA (not as bad, the same, or worse) in comparison to previous MVAs.

Two standardised questionnaires, to assess peri-traumatic dissociation and the level of alertness were used, comprising:

1. Stanford Sleepiness Scale (SSS; Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973). The SSS is a 7-item scale that has become the most widely used subjective instrument for sleepiness evaluation (Curcio, Casagrande, & Bertini, 2001). Participants were asked to rate their level of alertness by choosing one of seven hierarchical statements that best described
their level of alertness immediately prior to the MVA. (e.g. "I felt active, wide awake" was
given a score of 1 and "I could not stay awake, sleep onset was imminent" was given a
score of 7). Conner et al. (2002) suggested that a score of 4 or higher indicates non-
alertness. Hoddes, Dement and Zarcone (1972) reported an alternate form reliability of $r =
.88$. The same scoring method as used by Conner et al. (2002) to categorise alertness levels
was applied, with scores $\geq 4$ categorised as sleepy/not alert.

2. The Peritraumatic Dissociative Experiences Questionnaire-Self Report (PDEQ-SR;
Marmar, Weiss, & Metzler, 1997). The PDEQ-SR is a 10-item scale which assesses
retrospective dissociative symptoms (e.g. "I felt as if things were not real"). Participants
responded to items using a 5-point likert rating scale in reference to their MVA from 1= not
at all true to 5 = extremely true. Total score is the sum of the 10-items, with higher scores
indicating greater peritraumatic dissociative symptomatology. Scores ranged from 10 to 50
with greater than 10 categorised as experiencing a dissociative response. Marmar et al.
(1997) reported high internal consistency ($\alpha = .80$) as well as good divergent and
convergent validity.

4.1.4 Procedure

The John Hunter Hospital (JHH) provides primary and tertiary trauma services to a
population of approximately 840,000 people, covering a geographical area of
approximately 130,000 square kilometers. At the time of the study the JHH Accident and
Emergency Department was the busiest in NSW, treating 54,318 people in 2005, of which
1,771 were MVA presentations with 523 admissions; including adult and paediatric as well as MVA fatalities (Hunter New England Health Service, 2006).

The Maitland Hospital is a rural referral facility for the upper and lower Hunter regions. In 2005, 32,810 people were treated at the hospital, including 430 MVA presentations; including adult, paediatric and MVA fatalities. Seventy-six of these were either admitted to the Maitland Hospital, or those who were severely injured or required specialist treatment were transported to JHH.

Following consent from both hospitals to be involved in the study, ethics approval (Appendix A) from Hunter New England Area Health Service Human Research Ethics Committee and (Appendix A1) The University of Newcastle Research Ethics Committee was granted.

In order to increase the potential number of MVA survivors available for inclusion in the study, permission was subsequently sought to recruit MVA survivors from two additional regional hospitals. Approval was granted from the hospital’s director of trauma services, the Central Coast Area Health Service ethics committee (Appendix A2) and from the Clinical Nurse Consultant (CNC) of trauma services who had agreed to indentify and recruit potential participants for inclusion in the study. Combined, these two hospitals on average have over 85,500 accident and emergency attendances each year (Northern Sydney Central Coast Health, 2009). However, due to unforeseen circumstances no participants were recruited from either hospital as recruitment for the current study conflicted with the recruitment procedure of a study running concurrently at both hospitals.
To ensure MVA survivors’ anonymity, the Clinical Nurse Consultants (CNCs) of Trauma Services at both JHH and The Maitland hospital identified and mailed potential participants a Phase 1 survey package (see Appendix B) within 2-weeks after hospital discharge. MVA survivors were categorised as either being admitted to hospital or as being treated and discharged from hospital the same day. Each survey sent was coded in order to enable identification of the hospital (Admitted or Treated) and MVA category type. The code number, age and gender of each MVA survivor who was sent a survey were recorded. This provided information for comparison of demographic characteristics to determine the representativeness of the sample. The code numbers also provided a means of linking returned surveys from later phases of the study.

The Phase 1 survey package comprised a cover letter on hospital letterhead signed by the CNCs explaining why they (MVA survivors) had received an invitation to participate in the study and how they had been selected. An information sheet which outlined the nature of the study, a Phase 1 survey and a results summary request form were also included in the package. Using the pre-paid return addressed envelope provided in the survey package, participants could return the survey anonymously. Also included in the Phase 1 survey package was an invitation to participate in a follow-up study (Phase 2) and a follow-up study information sheet. Participants who volunteered for the follow-up study or requested a summary of results provided their name and postal address for future correspondence. Return of the survey was considered as consent. A total of 1410 surveys were distributed and 367 were returned, which equated to a 26.02% response rate (see Table 4.1).
The initial selection criteria were being aged 18-years of age or over and having experienced an MVA. It was anticipated that conscious impairment, assessed with the Glasgow Coma Scale (Teasdale & Jennett, 1974), and if MVA survivors were proficient in English language would be able to be identified. Interpreter services were not available so participants had to be conversant in English language in order to understand and complete the survey. Being unconscious is a possible confounding variable, which may impair memory recall of accident details and immediate reactions as well as possible symptom overlap (memory and concentration difficulties) with PTSD. However, the CNCs when they identified MVA survivors, could not determine if English was a second language. Therefore, it is unknown if any surveys were not returned because of inadequate English language skills. Furthermore, the CNCs were not able to ascertain from computer records if MVA survivors had been unconscious. Given that this could not be accurately assessed, it poses as a potential confounding variable and a limitation of the study. It was also anticipated that the Injury Severity Scores (ISS) of each MVA survivor who received a Phase 1 survey would be recorded. However, only those MVA survivors who were admitted to hospital had ISS scores recorded.

4.2 Phase 2 methodology

4.2.1 Participants

Participants comprised MVA survivors from Phase 1 who had volunteered for the follow-up study and provided their contact details (mailed back with their initial survey) in order to be mailed the Phase 2 questionnaire to be completed at 3-months post-MVA. The Phase 2 sample ($n = 128$) comprised 48 males (37.5%) and 80 females (62.5%) ranging from 18 to
87 years of age. Detailed descriptions of sample characteristics including demographic information are shown in section 6.2.1.

4.2.2 Materials

The Phase 2 survey package (see Appendix C\textsuperscript{1}) comprised self-report items as well as standardised questionnaires. Participants were asked to indicate whether they had received any treatment following the accident for \textit{emotional problems}, \textit{physical problems} or both and if so the duration of treatment (1 day, \leq 1-week, \leq 4-weeks, 1 to 3-months, or \geq 3 months).

Seven standardised questionnaires were used to assess PTSD diagnostic status and symptom severity, depression symptom severity, physical disability, and alcohol consumption. Participants were also asked to indicate if their level of alcohol use had increased, decreased or was the same compared to before the accident. In order to assess pre-MVA personality, coping and trait dissociation participants were instructed \textit{“When answering the questions on this page and the following pages please think back to the time before the accident occurred”}.

Standardised Questionnaires

1. The Posttraumatic Stress Diagnostic Scale (PDS; Foa, Cashman, Jaycox & Perry, 1997) is a 49-item scale that assesses both PTSD diagnosis and symptom severity (range 0 to 51) based on the DSM-IV diagnostic criteria. Symptom severity items comprise 5

\textsuperscript{1} Standardised copyright questionnaires are not included in Appendices.
reeexperiencing symptom statements (e.g., *having bad dreams or nightmares about the traumatic event*), 7 avoidance symptom statements (e.g., *Trying not to think about, talk about, or have feelings about the traumatic event*), and 5 arousal symptom statements (e.g., *Having trouble falling or staying asleep*). Participants respond by indicating how often they had been bothered by each of the symptom statements with corresponding scores of:

0 = *Not at all or only one time*, 1 = *Once a week or less/once in a while*, 2 = *2 to 4 times a week/half the time*, 3 = *5 or more times a week/almost always*). In order to assess PTSD in relation to the MVA, participants were prompted to answer the PDS questions in relation to their recent MVA as opposed to other trauma(s) they may have previously experienced. The PDS has a reported test-retest reliability of \( r = .74 \), internal consistency \( \alpha = .92 \) and also demonstrated a high level of agreement (kappa = .65, and agreement 82%) on the diagnosis of PTSD compared with Structural Clinical Interview (SCID).

2. The Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Brown, 1996) is a widely used measure of depression symptom severity. It consists of 21 multiple-choice self-rating statements with item scores ranging from 0 to 3. Participants respond to each of the items based on how they have been feeling in the past 2-weeks by circling the statement that is most applicable to them. A total score is calculated by summing each of the item scores, with scores ranging from 0 to 63. The BDI-II has high internal consistency \( \alpha = .93 \) and high test-retest reliability \( r = .93 \). Based on the authors scoring recommendation the cut-off total score for moderate levels of depressive symptoms \( \geq 20 \) was used to categorise symptomatic levels of depression.
3. The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993) identifies harmful, hazardous and dependence patterns of alcohol consumption. The AUDIT comprises 10 items with scores ranging from 0 to 4. Total score (range 0 to 40) is the sum of all items. Participants respond to each item by circling the response that is correct for them (e.g., *How often do you have a drink containing alcohol? 0 = never, 1= monthly or less, 2 = 2-4 times a month, 4 = 4 or more times a week*). Using the author’s recommendation, a total score of $\geq 8$ was categorised as problematic drinking. The AUDIT has high test-retest reliability ($r = .88$) and internal consistency ($\alpha = .81$) (Bohn, Babor, & Kranzler, 1995).

4. The Brief Disability Questionnaire (BDQ; von Korff, Ustun, Ormel, Kaplan, & Simon, 1996) is an 11-item self-report scale which assesses the degree of recent physical disability. Participants indicate whether they have been limited in performing different physical activities (e.g., *The type or amount of vigorous activity you can do, e.g., lifting objects, running or sports*) during the last month by ticking the appropriate response rating from $0 = not at all, 1 = yes, sometimes or a little, to 2 = yes, moderately or definitely$. Total score is the sum of all items, with a score range of 0 to 22, with higher scores indicating greater disability. The BDQ has high internal consistency ($\alpha = .88$).

5. The short Eysenck Personality Inventory (EPI; Duncan-Jones, 1983) consists of 8 neuroticism items and 8 extraversion items. Participants were instructed to tick either *Yes* or *No* to each of the items indicating whether or not it described them in general (e.g., *Have you often got a restless feeling that you want something but don’t know what?*). Total scores were calculated by summing the number of *Yes* endorsed items for both of the
sub-scales, with items 4 and 15 reverse scored. Correlations between the short scale and the full EPI have been found to be \( r = .90 \) for neuroticism and \( r = .85 \) for extraversion with good temporal stability \( r = .75 \) for neuroticism and \( r = .63 \) for extraversion.

6. The Coping Inventory for Stressful Situations (CISS; Endler & Parker, 1990) assesses emotion-focused, problem-focused, and avoidant coping styles. The CISS comprises 48-items which are rated on a 5-point likert scale. On each item participants rate the degree to which they engage in a particular coping strategy from 1 = not at all to 5 = very much, when encountering stressful situations (e.g., Focus on the problem and see how I can solve it). Total scores for each of the sub-scales are the sum of the 16 items, with scores ranging from 16 to 80. The reported test-retest reliability for the subscales ranged between, \( r = 0.51 \) to 0.73, and internal consistency ranged from, \( \alpha = 0.80 \) to \( \alpha = 0.90 \).

7. The Trait Dissociation Questionnaire (TDQ; Murray, Ehlers, & Mayou, 2002) is a 38-item scale which measures habitual dissociative tendencies comprising detachment, sense of split-self, emotional numbing, amnesia for important life events, confusion/altered sense of time, lability of mood, and inattention (e.g., Things seem to go faster or slower than they really do). Scores range from 0 to 190 with item ratings of 0 = never, 1 = rarely, 2 = sometimes, 3 = often, 4 = mostly, 5 = always. Total score is the sum of all items. The reported internal consistency was \( \alpha = .92 \) and test-retest reliability was \( r = .82 \).

4.2.3 Procedure

An information sheet was included in the initial phase of the study (Phase 1 survey package) which outlined the nature of a follow-up study (Phase 2). Participants could
volunteer for the follow-up study by providing their contact details (mailed back with their initial survey) in order to be mailed the Phase 2 questionnaire to be completed at 3-months post-MVA. From the 337 participants who returned the Phase 1 survey, 223 (66%) volunteered to be included in the Phase 2 follow-up study. However only 134 (60%) of the 223 surveys that were mailed were returned, as shown in Table 4.1.

4.3 Phase 3

4.3.1 Participants

The sample \((n = 58)\) comprised 20 males (34.4%) and 38 females (65.6%) who ranged from 18 to 81 years of age who had volunteered to participate in a second (6-month post-MVA) follow-up study. Additional Phase 3 sample characteristics are displayed in Table 7.1 and are further described in section 7.2.1.

4.3.2 Materials

The Phase 3 survey was identical to Phase 2 with the exception of the instructions in response to personality, coping, and trait dissociation measures. In Phase 2, participants were instructed to think back to before their MVA, but in Phase 3 participants were instructed “The questions on this page and the following pages refer to how you are currently feeling and behaving”.

In an attempt to increase the number of surveys returned participants who had been mailed a Phase 2 survey and had not returned it were sent a reminder letter (Appendix D) thanking them for their previous contribution and asking if they had not yet returned the survey could they please do so.
4.3.3 Procedure

Each of the 128 participants who had returned a completed Phase 2 survey was mailed an information package which sought volunteers for a second follow-up survey and interview (Phase 4). The information package contained an invitation letter, Phase 3 and Phase 4 information sheets, consent form, and pre-paid returned addressed envelope (Appendix E). From the 128 invitations sent, 65 participants (50.78%) agreed to participate in Phase 3 and were posted the second follow-up survey to be completed at 6-months post-accident. From the 65 surveys sent a total of 63 were returned which equated to a response rate of 96.9% (see Table 4.1).

4.4 Phase 4

4.4.1 Participants

As shown in Table 4.1 the Phase 4 sample \( n = 51 \) comprised 19 males (37.3%) and 32 females (62.7%) who ranged from 18 to 81 years of age. All participants had previously completed the Phase 3 survey. Details of the Phase 4 sample characteristics are displayed in Table 8.1 and further described in section 8.2.1

4.4.2 Materials

Phase 4 comprised computerised administration of the PTSD, depression, and alcohol modules of the Composite International Diagnostic Interview-Auto, version 2.1 (WHO, 1997; CIDI-A). The CIDI-A is a standardised structured interview which allows for classification of diagnosis based on the DSM-IV criteria. Prior to commencing interviews, familiarization in the administration of the CIDI-A was undertaken and numerous practice sessions were conducted under the supervision of an experienced Clinical Psychologist (Dr.

Participants also completed the Posttraumatic Cognitions Inventory (PTCI; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999). The PTCI is a 36-item scale that assesses negative cognitions about self, negative cognitions about the world and self-blame. Participants answer each statement using a 7-point likert response format from 1= totally disagree to 7 = totally agree. The test-retest reliability of the total score is good ($r = .85$), as is the internal consistency for the 3 factors ($\alpha = 0.97$, $\alpha = 0.88$, and $\alpha = 0.86$) respectively.

### 4.4.3 Procedure

As outlined in section 4.3.3, after receiving an invitation which sought volunteers to participate in Phase 3 and Phase 4 (interview), 58 participants initially agreed to be interviewed. However, when contacted to arrange a suitable interview time, seven participants declined to be interviewed. Participants who volunteered for Phase 4 were given the opportunity to have the interview conducted at a Psychology Clinic located in a regional university or in their home (see Appendix A3 for ethics approval letter) by indicating their preference on the returned consent form. Participants who were unable to travel to the interview were interviewed via the telephone after interview-related material was posted to them. Interviews were conducted after completion of Phase 3 at approximately 9-months post-MVA. Those participants who incurred a cost associated with interview travel expenses were offered $20.00 to cover costs.
Table 4.1

Summary of Longitudinal Sample Recruitment Information

<table>
<thead>
<tr>
<th>Sample Characteristics</th>
<th>1 Month Post-MVA (Phase 1)</th>
<th>3 Months Post-MVA (Phase 2)</th>
<th>6 Months Post-MVA (Phase 3)</th>
<th>9 Months Post-MVA (Phase 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>337</td>
<td>128</td>
<td>58</td>
<td>52</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males n (%)</td>
<td>157 (46.6%)</td>
<td>48 (37.5%)</td>
<td>20 (34.4%)</td>
<td>19 (37.3%)</td>
</tr>
<tr>
<td>Females n (%)</td>
<td>180 (53.4%)</td>
<td>80 (62.5%)</td>
<td>38 (65.6%)</td>
<td>32 (62.7%)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>18-88 years</td>
<td>18-87 years</td>
<td>18-81 years</td>
<td>18-81 years</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>39.33 years (17.75)</td>
<td>42.10 years (18.01)</td>
<td>45.19 years (18.12)</td>
<td>45.33 years (17.73)</td>
</tr>
<tr>
<td>Surveys Sent</td>
<td>1410</td>
<td>223</td>
<td>65b</td>
<td>-</td>
</tr>
<tr>
<td>Surveys Returned</td>
<td>367</td>
<td>134</td>
<td>58</td>
<td>-</td>
</tr>
<tr>
<td>Response Rate (%)</td>
<td>26.02%</td>
<td>60.00%</td>
<td>89.23%</td>
<td>87.93%a</td>
</tr>
<tr>
<td>Interviews conducted</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>51</td>
</tr>
</tbody>
</table>

Note. a 58 participants initially volunteered to be interviewed however when contacted to arrange the interview only 51 agreed.
b a total of 128 Phase 3/Phase 4 invitations were mailed and 63 were returned equating to a 49.21% response rate.
Chapter 5

5.1 Phase 1 Study

5.1.1 Aims of the Study

The overall aim of this study was to examine the extent of post-accident psychological distress and in the process describe the accident and demographic characteristics of the sample. Further aims were to identify predictors of peritraumatic dissociation as well as post-MVA distress. The literature reviewed in chapter two, highlights the role of both pre-trauma and trauma related characteristics in the development of psychopathology. However, given that findings have not always been consistent, a further aim was to examine the role of pre and peritraumatic characteristics and their relation to post-MVA distress. The influence of pre-trauma factors: past emotional problems; gender, and age; trauma-related factors: accident and injury severity; and also peritraumatic variables: fear of dying and peritraumatic dissociation, were each examined as were the associations between variables and their relationship to post-MVA distress. An exploratory component of this study was to examine survivors’ level of alertness prior to their MVA according MVA category types and immediate as well as post-MVA sequelae.

5.1.2 Hypotheses

On the basis of theory and past research it was hypothesised that (1) pre-trauma characteristics, traumatic event characteristics and peritraumatic psychological characteristics would predict post-MVA psychological morbidity. More specifically, it was hypothesised that (2) peritraumatic psychological characteristics would be more influential
than pre-trauma characteristics on post-MVA distress. It was also hypothesised that (3) the influence of traumatic event characteristics on post-MVA distress would be mediated by dissociation. Furthermore, it was hypothesised that (4) fear would be a cause rather than a consequence of dissociation and that (5) age (younger) and gender (female) would be vulnerability factors for dissociative responses as well as post-MVA distress.

5.2 Results

Statistical Package for the Social Sciences (SPSS) 16 was used for statistical analyses with an alpha level of 0.05. For t-tests, when Levene’s test of homogeneity indicated a significant difference between the variances of groups, the Welsch-Satterthwaite corrected degrees of freedom and significance values are reported. For chi-square tests, where more than 25% of the cells have an expected frequency of 5 or less, Fisher’s Exact Test probabilities are reported. Additionally, tests of analysis of variance (ANOVA) and Multiple Linear Regression (MLR) assumptions of homogeneity (using Levene’s Test) as well as normality (using the Kolmogorov-Smirnov Test (K-S) are not reported unless the assumptions were violated. Since only one participant was a Pillion Passenger on a Motorcycle, their data were included in the Motorcyclist group.

5.2.1 Demographics

A total of 1,410 surveys were distributed but only 367 were returned, which is a response rate of 26%. However, 30 surveys were returned blank or incomplete and therefore were not included in statistical analyses. Not all participants answered every question; therefore, the sample size in some analyses may vary slightly from 337. As shown in Table 5.1, the
sample \((N = 337)\) comprised 286 participants \((85\%)\) from JHH and 51 participants \((15\%)\) from the Maitland Hospital. Collectively, there were 157 males and 180 females who ranged from 18 years to 88 years of age \((M = 39.33, SD = 17.75)\).

In terms of educational attainment, 111 participants \((33\%)\) reported that they achieved Some Secondary Schooling, 53 participants \((16\%)\) Completed Year 12, 104 \((31\%)\) reported that they had completed TAFE or College, and 67 participants \((20\%)\) completed University. One hundred and ten participants \((33\%)\) reported an income level of Less than $15,000, 77 \((23\%)\) reported earning Between $15,000 and $30,000, 79 \((24\%)\) earned Between $30,000 & $50,000, and 69 participants \((20\%)\) reported earning More than $50,000.

A comparison of participants’ demographic characteristics between hospitals showed that there were no significant differences in educational attainment, \(p = .516\) (Fisher’s Exact Test), income level, \(\chi^2 (9, N = 335) = 4.98, p = .849\), and age \(F (3,336) = 0.23, p = .995\). However, results showed a significant gender difference between the hospitals, \(p = .001\) (Fisher’s Exact Test). The adjusted standardised residuals\(^1\) revealed that significant differences were in the JHH Admitted group, with 53 males accounting for 64\%, and with the Maitland Hospital Treated group, with 14 males accounting for 31\%, as shown in Table 5.1. Further demographic comparisons between participants who received treatment and those admitted to hospital revealed no significant differences in age, \(t (335) = .25, p = .806\), education, \(t (333) = 1.34, p = .179\), or income level, \(t (333) = .78, p = .438\).

---

\(^1\) Adjusted (standardised) residuals provide a test for the significance of individual cells. Adjusted residuals that are greater than the absolute value of 2.00 are deemed to make a significant contribution to the overall chi-square value.
Table 5.1

*Hospitals Age and Gender Composition and Attendance Type*

<table>
<thead>
<tr>
<th>Sample Characteristics</th>
<th>JHH Treated</th>
<th>JHH Admitted</th>
<th>JHH Total</th>
<th>Maitland Treated</th>
<th>Maitland Admitted</th>
<th>Maitland Total</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>n 88</td>
<td>53</td>
<td>141</td>
<td>14</td>
<td>2</td>
<td>16</td>
<td>157 (47%)</td>
</tr>
<tr>
<td>Mean age</td>
<td>38.23</td>
<td>40.70</td>
<td>39.16</td>
<td>36.29</td>
<td>33.50</td>
<td>35.94</td>
<td>38.83</td>
</tr>
<tr>
<td>SD</td>
<td>16.73</td>
<td>17.88</td>
<td>17.15</td>
<td>20.16</td>
<td>21.92</td>
<td>19.63</td>
<td>17.38</td>
</tr>
<tr>
<td>Range</td>
<td>18 - 81</td>
<td>18 - 79</td>
<td>18 - 81</td>
<td>20 - 87</td>
<td>18 - 49</td>
<td>18 - 87</td>
<td>18 - 87</td>
</tr>
<tr>
<td>Female</td>
<td>n 115</td>
<td>30</td>
<td>145</td>
<td>31</td>
<td>4</td>
<td>35</td>
<td>180 (53%)</td>
</tr>
<tr>
<td>Mean age</td>
<td>40.36</td>
<td>35.87</td>
<td>39.43</td>
<td>41.48</td>
<td>41.25</td>
<td>41.17</td>
<td>39.82</td>
</tr>
<tr>
<td>SD</td>
<td>17.27</td>
<td>18.26</td>
<td>17.51</td>
<td>19.80</td>
<td>28.15</td>
<td>20.63</td>
<td>18.06</td>
</tr>
<tr>
<td>Range</td>
<td>18 - 80</td>
<td>18 - 69</td>
<td>18 - 80</td>
<td>18 - 88</td>
<td>18 - 69</td>
<td>18 - 88</td>
<td>18 - 88</td>
</tr>
<tr>
<td>Total</td>
<td>N (%) 203 (60%)</td>
<td>83 (25%)</td>
<td>286 (85%)</td>
<td>45 (13%)</td>
<td>6 (2%)</td>
<td>51 (15%)</td>
<td>337 (100%)</td>
</tr>
</tbody>
</table>

*Note.* JHH = John Hunter Hospital.
There were no significant gender differences in educational attainment, $\chi^2 (3, N = 335) = 2.96, p = .393$, nor age, $t (335) = -0.48, p = .631$. However, results showed a significant association between gender and income level, $\chi^2 (3, N = 335) = 18.36, p = .001$. The adjusted standardised residuals revealed that significant differences were in the Less than $15,000$ group with 73 females accounting for 65% and the More than $50,000$ group with 24 females accounting for 35%.

In order to ascertain if the sample was representative of the hospital MVA population, the demographic information (obtained by JHH and The Maitland hospital) and Injury Severity Score (ISS) of those admitted to JHH who received a survey over a 1-year period were compared. The proportion of females in the study sample, 53.4% ($N = 337$), was compared to the population proportion, 42.6% ($N = 1,094$), and found to be significantly higher, $p = .001$ (one sided)$^2$. A comparison between the mean age of the sample ($M = 39.3$) and that of the population ($M = 35.4$) also revealed a significant difference, $t (336) = 4.06, p = .001$. Therefore, females and slightly older people were more likely to volunteer to participate in the research. There was no significant difference between the ISS in the study sample compared to the hospital ISS.

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$^2$ Significance testing to determine if sample proportions were different to the population proportions was carried out using SPSS’s binomial test for a single proportion. Due to the sample sizes involved, SPSS reported in each case a one-sample $Z$ test using the normal approximation to binomial. As the differences in all cases (except JHH Admitted) were very significant, the finite population correction to the variance was not carried out for those, as the correction would only have made the test more significant.
5.2.2 Accident Characteristics

Pre-Accident Information

One hundred and eighty six participants (55%) reported that they had previously experienced an MVA. The range of previous MVAs varied between 1 and 10 ($M = 1.91$, $SD = 1.30$), with 93 (50%) participants reporting having experienced 1 previous MVA, 50 participants (27%) experiencing 2 previous MVAs, 26 (14%) reporting 3 previous MVAs and the remaining 17 participants (9%) reported that they had experienced four or more previous MVAs. Compared to previous MVAs 48 participants (26%) rated the current MVA as *not as bad* as previous accidents, 25 (13%) rated it *the same*, and 113 (61%) rated this MVA as *worse* than previous accidents. A gender comparison between those who had experienced previous MVAs revealed a significant difference, ($\chi^2 (1, N = 337) = 5.16$, $p = .023$), with males ($n = 97$) accounting for 62%. The number of previous MVAs experienced by males ($M = 2.14$) was greater than females ($M = 1.65$) and the difference ($M = .49$) was significant, $t (148) = 2.68$, $p = .008$ (Welsch-Satterthwaite corrected). There was no significant difference between gender and participants’ MVA comparative rating (*not as bad, the same, worse*) $\chi^2 (2, N = 186) = 2.17$, $p = .331$). No other significant differences were found between participants who had previously experienced an MVA and those who had not on the following variables; age, past emotional problems, level of alertness, accident severity, injury severity, fear of dying, peritraumatic dissociation, anxious or fearful, and sad or depressed.
5.2.3 Past Emotional Problems

Amongst this sample, 213 participants (63%) reported that they had been not at all bothered by emotional problems (feeling depressed or anxious) in the 4-weeks prior to their MVA while 124 participants (37%) reported, to varying degrees, that they had been bothered by emotional problems, with 74 participants (22%) reporting a little, 20 (6%) somewhat, 25 (7%) a lot, and 5 participants (2%) reported that they had been extremely bothered by emotional problems in the 4 weeks leading up to their MVA. Females (n = 78, 63%) experiencing past emotional problems were significantly over-represented ($\chi^2 (1, N = 337) = 7.10, p = .008$) compared to males (n = 46). However, there was no significant gender difference in the severity rating of past emotional problems, $t (122) = .29, p = .771$. Further analysis revealed that participants who reported having experienced past emotional problems compared to those who did not were younger ($M = 36.13$ vs. $M = 41.19$), $t (295) = -2.66, p = .008$ (Welsch-Satterthwaite corrected), were less alert at the time of the accident ($M = 2.10$ vs. $M = 1.69$, $t (335) = 2.69, p = .007$), and reported more peritraumatic dissociative symptoms ($M = 23.98$ vs. $M = 19.94$, $t (332) = 3.81, p = .001$). No significant differences were found between participants who had experienced past emotional problems and those who had not in terms of accident severity, injury severity, and fear of dying.

5.2.4 Level of Alertness

Level of alertness immediately prior to the MVA was rated from 1) felt active/wide awake to 7) could not stay awake/sleep onset was imminent. Demographic comparisons revealed no gender difference. Age comparisons between alertness categories showed that participants who were categorised as sleepy (less alert) were significantly younger
(M = 29.25 years) than participants who were alert (M = 40.53 years), t (50) = - 4.50, p = .001, (Welsch-Satterthwaite corrected). Similarly, as a continuous variable level of alertness showed a significant relationship with age, F (6, 336) = 4.92, p = .001, though due to heteroscedacity of variance, F (6, 330) = 2.64, p = .016, and a violation of the normality assumption, K-S (337) = .07, p = .001, the significance level may need to be interpreted with a degree of caution. Post-hoc Comparisons using Tukey’s test, with a familywise Type I error rate of .05, revealed that the mean age of participants in level 1 (felt active/wide awake) was significantly greater than those in level 2 (was functioning at a high level but not at peak), as illustrated in Figure 5.1.

![Figure 5.1. Mean age range and associated levels of alertness.](image-url)
A comparison of alertness mean scores between MVA category types (illustrated in Figure 5.2) revealed a significant main effect, $F(4, 336) = 5.38, p = .001$. However, due to heteroscedacity of variance, $F(4, 332) = 11.41, p = .001$, and a violation of the normality assumption, $K-S(337) = .24, p = .001$, the significance level may need to be interpreted with a degree of caution. Post-hoc comparisons using Tukey’s test, with a family-wise Type I error rate of .05, revealed that passengers were significantly less alert than drivers, motorcyclists and pedal cyclists. Sleepy participants’ peritraumatic dissociation scores ($M = 27.28$) were significantly greater than alert participants’ scores ($M = 20.72$), $t(332) = 3.99, p = .001$.

Further comparisons between participants categorised as sleepy and alert revealed no significant differences on accident severity, injury severity, and fear of dying nor feeling sad nor depressed and anxious nor fearful.

![Figure 5.2](image-url)  
*Figure 5.2. MVA category types and associated levels of alertness.*
5.2.5 Accident Information

Drivers ($n = 190$) constituted 56% of the sample, passengers ($n = 70$) 21%, motorcyclists ($n = 48$) 14%, pedal cyclist ($n = 20$) 6%, and pedestrians ($n = 9$) accounted for 3% of the sample. It was anticipated that the time of the MVA to completion of the survey would be less than 2-weeks but it was not always possible for the CNC`s to mail out surveys 2-weeks after hospital discharge and in some cases MVA survivors would have been admitted to hospital for a period greater than 2-weeks. Subsequently, the timeframe varied considerably, ranging from 5 days to 157 days, with the mean time between the MVA and completion of the survey being 37.17 ($SD = 21.24$). Overall, 50% of participants had completed the survey within 1-month and by 2-months 90% of participants had completed the survey.

5.2.6 Accident Severity

Accident severity was rated by 11 participants (3%) very slight, 94 (28%) as moderate, 127 (38%) as serious, 75 (22%) as severe, and 30 participants (9%) rated their MVA as very severe. Males rated the accident more severe compared to females ($M = 3.17$ vs. $M = 2.97$ respectively) and this difference approached significance at $t (315) = 1.91, p = .057$.

Accident severity ratings differed according to age, $F (4, 332) = 2.84, p = .024$. Post-hoc Comparisons using Tukey’s test, with a familywise Type I error rate of .05, revealed that participants who rated their MVA as very slight were significantly older ($M = 56$ years) than participants who rated their MVA as moderate ($M = 39.81$ years), serious ($M = 39.74$ years), severe ($M = 37.07$ years), and very severe ($M = 36.17$ years). Analysis between MVA category type revealed a significant main effect, $F (4, 336) = 3.62, p = .007$, though it was noted that although the homoscedacity assumption was supported, $F (4, 332) = 1.65, p = .160$, the distribution of the residuals indicated a departure from normality, $K-S (337) = .18$, 


Post-hoc Comparisons revealed that accident severity ratings of drivers, passengers and motorcyclists was significantly greater than those of pedal cyclists, as displayed in Figure 5.3.

![Figure 5.3. Accident severity ratings and MVA category types.](image)

### 5.2.7 Injury Severity

Injuries were sustained by 317 (94%) participants with 96% of males (n = 150) and 93% of females (n = 167) reporting injuries. Injury severity was rated by 89 participants (28%) as mild, 148 (46%) as moderate, 75 (24%) as serious, 75 (22%) and 5 participants (2%) rated their MVA as life threatening. Age comparisons revealed no significant differences on injury status or injury severity and though there was no significant difference between injury status and gender, males rated their injuries as more severe than did females ($M = 3.08$ vs. $M = 2.90$ respectively), $t (315) = 2.05, p = .040.$
Significant though somewhat weak correlations were found between injury severity and accident severity, fear of dying, sad or depressed, and anxious or fearful (see Table 5.2). A comparison between MVA category types as illustrated in Figure 5.4 showed that motorcyclists ($M = 3.27$) rated their injuries as most severe followed by passengers ($M = 3.01$), pedal cyclists ($M = 3.00$), drivers ($M = 2.91$) and pedestrians ($M = 2.78$). The differences between these groups approached significance, $F (4, 316) = 2.35, p = .053$.

*Figure 5.4. Injury severity ratings and MVA category types.*
Table 5.2

*Pearson Correlation Coefficients of Pre, Peri, and Post-MVA Variables*

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>-</td>
<td>.03</td>
<td>-.23**</td>
<td>-.13*</td>
<td>.03</td>
<td>-.26**</td>
<td>-.09</td>
<td>-.11*</td>
<td>-.06</td>
<td>-.05</td>
</tr>
<tr>
<td>2. Gender</td>
<td>-</td>
<td>-.09</td>
<td>-.11</td>
<td>-.10</td>
<td>.13*</td>
<td>.14*</td>
<td>.11*</td>
<td>.13*</td>
<td>.22**</td>
<td></td>
</tr>
<tr>
<td>3. Level of Alertness</td>
<td>-</td>
<td>.04</td>
<td>.05</td>
<td>.22**</td>
<td>.02</td>
<td>.18**</td>
<td>.06</td>
<td>.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Accident Severity</td>
<td>-</td>
<td>.33**</td>
<td>.24**</td>
<td>.44**</td>
<td>.04</td>
<td>.24**</td>
<td>.20**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Injury Severity</td>
<td>-</td>
<td>.09</td>
<td>.24**</td>
<td>-.02</td>
<td>.18**</td>
<td>.12**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Peritraumatic Dissociation</td>
<td>-</td>
<td>.40**</td>
<td>.19**</td>
<td>.32**</td>
<td>.37**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Fear of Dying</td>
<td>-</td>
<td>.07</td>
<td>.30**</td>
<td>.30**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Past Emotional Problems</td>
<td>-</td>
<td>.31**</td>
<td>.27**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Sad or Depressed</td>
<td>-</td>
<td>.31**</td>
<td>.27**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Anxious or Fearful</td>
<td>-</td>
<td>-</td>
<td>.75**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

*Note.* N = 337.

*p < .05. **p < .01.*
5.2.8 Fear of Dying

The degree to which participants feared for their lives during the MVA varied with 85 participants (25%) reporting not at all, 71 (21%) reported a little, 67 (20%) somewhat, 76 (23%) a lot, and 35 participants (11%) reported certain I would die. Females ($n = 142$) accounted for a significantly greater proportion (57%) of participants who feared for their lives compared to males, $\chi^2 (1, N = 334) = 4.64, p = .031$. However, the extent to which females feared for their lives ($M = 2.79$) was not significantly different to males ($M = 2.63$). Similarly, there was no significant effect of age on either experiencing or the degree of fear.

Analysis between MVA category types and fear of dying (illustrated in Figure 5.5) revealed a significant main effect, $F (4, 333) = 5.78, p = .001$. Post-hoc Comparisons using Tukey’s test, with a familywise Type I error rate of .05, revealed that the extent to which drivers, passengers and pedestrians feared for their lives was significantly greater than that of pedal cyclists.

![Figure 5.5. MVA category types and fear of dying mean scores.](image)
5.2.9 Peritraumatic Dissociation

Having experienced peritraumatic dissociation was reported by 299 participants (89.5%) with scores ranging from 11 to 50 ($M = 22.77$, $SD = 9.15$). A greater proportion of females experienced peritraumatic dissociation compared to males ($\chi^2 (1, N = 334) = 4.41, p = .036$), the strength of peritraumatic dissociative symptoms was also greater for females, $t (332), = -2.51, p = .012$. Participants (95%) who reported having past emotional problems were more likely to have experienced peritraumatic dissociation ($\chi^2 (1, N = 334) = 6.51, p = .011$). Similarly, the mean dissociative scores of those participants ($M = 23.98$) was greater than participants who had not experienced past emotional problems ($M = 19.94$), $t (332), = 3.81, p = .001$. Analysis of age and peritraumatic dissociation revealed a significant main effect, $F (9, 333) = 1.51, p = .034$) as displayed in Figure 5.6. Given that age was a continuous variable post-hoc analysis were not conducted. An independent samples $t$-test showed that the mean age of participants who reported dissociation ($M = 37.86$) was significantly less than those who did not dissociate ($M = 51.03$) at the time of the accident ($t (332) = - 4.24, p = .001$). Analysis of peritraumatic dissociation and MVA type showed a significant main effect, $F (4, 333) = 2.64, p = .034$, however, Tukey’s test with a familywise Type I error rate of .05 for post-hoc comparisons, revealed that there were no significant differences between the MVA groups on peritraumatic dissociation scores.
5.2.10 Post Accident Distress

Post-MVA distress, as shown in Table 5.3, was reported by approximately two-thirds of the sample. Participant response ratings of feeling sad or depressed and anxious or fearful were somewhat similar. The representation of females who reported post-MVA distress compared to males was significant: $\chi^2 (1, N = 337) = 5.30, p = .021$, sad or depressed; $\chi^2 (1, N = 337) = 11.03, p = .001$, anxious or fearful. Similarly, females’ (mean) rating of distress was also greater than males’: $t (335), = - 2.51, p = .015$, sad or depressed; $t (334), = - 4.19, p = .001$, anxious or fearful. There were no significant age effects in relation to post-MVA distress.

Figure 5.6. Mean age range and associated peritraumatic dissociation scores.
There was a significant association in the proportion of participants who reported post-MVA distress with 85% of participants who felt anxious or fearful also reported feeling sad or depressed, ($\chi^2 (1, N = 337) = 101.79, p = .001$). There was also a significant association between having experienced past emotional problems in the 4-weeks prior to their MVA and post-MVA distress with 77% of participants who reported feeling sad or depressed and 73% who reported feeling anxious also reporting past emotional problems ($\chi^2 (1, N = 337) = 14.52, p = .001$, ($\chi^2 (1, N = 337) = 4.92, p = .026$, respectively). Participants who experienced past-emotional problems in comparison to those who did not, were also more inclined to endorse greater levels of post-MVA distress ($t (232) = 4.88, p = .001$, sad or depressed, $t (216) = 4.26, p = .001$, anxious or fearful, Welsch-Satterthwaite corrected).
5.3 Predictors of Peritraumatic Dissociation and Post-MVA Distress

5.3.1 Path Analysis

In order to simultaneously analyse the influence and interaction of variables associated with both peritraumatic dissociation and post-MVA distress, a statistical approach beyond the capabilities of Multiple Linear Regression was required. Path analysis (the original form of structural equation modelling using observed variables) was chosen as the most appropriate method of analysis. A path analytic approach allows for the prediction of simultaneous dependant variables and analysis between groups would potentially differentiate the effects of age and gender.

Path analysis (an application of SEM) was conducted using AMOS (Arbuckle, 2005 version 6.0) maximum-likelihood method. Multiple criteria for evaluating model fit were used. Kline (2005) recommends a Pearson chi-squared divided by the degrees of freedom (\(\chi^2/df\)) with values of less than 3.0 and a nonsignificant chi-squared goodness-of-fit test suggesting a good fitting model. Hu and Bentler (1999) recommend that the comparative fit index (CFI) should be greater than 0.95 and a root mean square error of approximation (RMSEA) value less than 0.06 considered desirable. Modification indexes were used to guide model re-specification based on theoretical considerations and previous findings. Modification indexes are a statistical application used to test if the addition of paths not included in the original model improve model fit. Akaike’s information criterion (AIC) was used to evaluate competing models. As recommended by Kline, a lower AIC value is favoured. When comparing nested models (multiple group comparisons) both the standardised and unstandardised path coefficients have been presented as standardised...
indicators can have different variances and error terms in which case unstandardised comparisons are preferred (see Kline, 2005). To develop an initial model the following process was adopted. Pearson correlations were computed between variables (see Table 5.2), and there was no evidence of multicollinearity as all correlations were found to be less than .85 (Kline, 2005).

As shown in Table 5.2 the relationships between pre and accident variables were modest with significant correlations between peritraumatic dissociation and age, gender, level of alertness, accident severity, past emotional problems, whilst fear of dying showed a moderately strong relationship with peritraumatic dissociation. Also noted in Table 5.2 is the strong positive relationship between measures of post-accident distress: feeling sad or depressed and anxious or fearful. Though these two variables share a common variance they will be considered separately in order to assess their independent effects which will provide information on their unique aspects in this and in the following phases of the study.

Based on the hypotheses an initial model was developed whereby variables of interest that were significantly correlated were included in the model. Skew and kurtosis levels were within acceptable limits (< 2.0 & 5.0 respectively), however the initial model did not meet the goodness-of-fit criteria, $\chi^2 = 46.60, p < .001$ (df = 10; N = 333), $\chi^2/df = 4.66$, CFI = .936; RMSEA = .105, AIC = 114.60. Examination of the standardized path coefficients showed that five paths were not significant: between injury severity and anxious or fearful ($\beta = .06, p = .490$); between injury severity and sad or depressed ($\beta = .11, p = .327$); between accident severity and sad or depressed ($\beta = .08, p = .096$); between accident
severity and anxious or fearful ($\beta = .05, p = .411$) and between accident severity and peritraumatic dissociation ($\beta = .07, p = .162$). To improve model fit, these paths were excluded and, based on the recommendations of Modification Indices, the covariance between injury severity and accident severity was added. The respecified model (see Figure 5.7) significantly improved model fit ($\Delta \chi^2 = 29.14, df = 4, p = .001$) resulting in an excellent fit, $\chi^2 = 17.46, p = .232$ ($df = 14; N = 333$), $\chi^2/df = 1.24$, CFI = .994; RMSEA = .027, AIC = 77.46. An alternate model was evaluated whereby the direction of the path coefficient from fear of dying to peritraumatic dissociation was reversed (fear of dying $\rightarrow$ peritraumatic dissociation vs. peritraumatic dissociation $\rightarrow$ fear of dying). However, the revised model did not meet all goodness of fit criteria $\chi^2 = 35.98, p = .001$ ($df = 14; N = 333$), $\chi^2/df = 2.57$, CFI = .962; RMSEA = .069, AIC = 95.98, indicating the former model provided a better fit of the data.

The final model illustrated in Figure 5.7 shows that past emotional problems ($\beta = .21$ and .26) fear of dying ($\beta = .18$ and .21), and dissociation ($\beta = .26$ and .19) predicted both anxious or fearful and sad or depressed respectively. However, neither level of alertness, nor perception of injury, nor accident severity had any direct effect on emotional state post MVA. Past emotional problems ($\beta = .21$ and .26) had more effect than fear of dying ($\beta = .18$ and .21) on both anxious or fearful and sad or depressed respectively (see Table 5.4 for the direct, indirect and total effects). Past emotional problems ($\beta = .21$) had less effect than dissociation ($\beta = .26$) on anxiety or fear but more effect ($\beta = .26$ vs. .19) on sad or depressed. The effect of accident and injury severity on anxious or fearful and sad or depressed is complex since they have direct effects on fear of dying ($\beta = .41$ and .11)
Figure 5.7. Path analysis model with standardized path coefficients. All paths are significant ($p < .05$). Dashed lines represent correlations.
respectively and fear of dying has direct effects ($\beta = .18$ and .21) on anxious or fearful and sad or depressed respectively, but also has a direct effect on dissociation ($\beta = .39$). Fear of dying fully mediated the relationship between both injury severity (standardized indirect effects .04 $p = .036$) and accident severity (standardized indirect effects .16 $p = .007$) with dissociation, which had direct effects on anxious or fearful and sad or depressed responses post-MVA.

Table 5.4

*Standardised Direct, Indirect, and Total Effects of Variables on Anxious/Fearful and Sad/Depressed*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Anxious/Fearful</th>
<th>Sad/Depressed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Direct</td>
<td>Indirect</td>
</tr>
<tr>
<td>Past Emotional Problems</td>
<td>.21*</td>
<td>.03</td>
</tr>
<tr>
<td>Alertness</td>
<td>-</td>
<td>.05</td>
</tr>
<tr>
<td>Injury Severity</td>
<td>-</td>
<td>.07</td>
</tr>
<tr>
<td>Accident Severity</td>
<td>-</td>
<td>.12</td>
</tr>
<tr>
<td>Fear of Dying</td>
<td>.18*</td>
<td>.10</td>
</tr>
<tr>
<td>Peritraumatic Dissociation</td>
<td>.26*</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note. N = 333.*

*p < .05.

The results showed there were four sets of (partial) mediated relationships (past emotional problems or fear of dying $\rightarrow$ dissociation $\rightarrow$ anxious or fearful and sad or depressed) within the overall model where dissociation is the mediator. The direct effects of past emotional problems were greater than the indirect effects for both anxious/fearful and sad/depressed
respectively. Similarly, the direct effects of fear of dying was greater than the indirect effect for both anxious/fearful and sad/depressed, as displayed in Table 5.4.

### 5.4.1 Demographic Comparisons

Further analysis examining demographic effects of both gender and age within the model were conducted. A median split for age (37 years) differentiated young from old. In order to ascertain if there are significant group differences, simultaneous multiple-sample path analysis was conducted. Following the recommendations of Kline (2005), cross-group equality constraints on the direct effects were imposed and based on the comparison between the constrained and unconstrained models. A chi-square difference statistic determined if the parameters were equal in the population from which the samples were drawn.

The results of gender group comparisons indicated that both the constrained and unconstrained models showed an adequate fit of the data (see Table 5.5). Inspection of the standardised and unstandardised path coefficients revealed that although some path coefficients were significant for females and not for males and vice versa (see Figure 5.8), results of nested model comparisons showed that the models were not significantly different from one another, indicating that there was no gender effect in the specified model.
Figure 5.8. Gender comparisons of unconstrained path coefficients. Females participants path coefficients are presented in parenthesis. 
a represents standardised paths, b represents unstandardised paths, and an * indicates that the larger path coefficient was significant for one 
gender but not for the other (p = < 0.05). Dashed lines represent correlations.
Table 5.5

*Gender Nested Model Comparisons and Fit Indices*

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$p$</th>
<th>$\chi^2$/df</th>
<th>CFI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconstrained</td>
<td>48.45</td>
<td>28</td>
<td>.010</td>
<td>1.73</td>
<td>.965</td>
<td>.047</td>
</tr>
<tr>
<td>Constrained</td>
<td>64.71</td>
<td>39</td>
<td>.006</td>
<td>1.65</td>
<td>.956</td>
<td>.045</td>
</tr>
<tr>
<td>$\Delta \chi^2$</td>
<td>16.25</td>
<td>11</td>
<td>.132</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note. N = 333.*

The results of age group comparisons indicated that both the constrained and unconstrained models showed an excellent fit to the data (see Table 5.6). Similar to the male and female comparisons, inspection of standardised and unstandardised path coefficients revealed some differences. Some path coefficients which were significant for older participants were not significant for younger participants and vice versa (see Figure 5.9). However, results of nested model comparisons showed that the young and old models were not significantly different from one another, which indicated that ‘overall’ there was no age effect within the specified model.

Table 5.6

*Age Nested Model Comparisons and Fit Indices*

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$p$</th>
<th>$\chi^2$/df</th>
<th>CFI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconstrained</td>
<td>26.78</td>
<td>28</td>
<td>.530</td>
<td>.957</td>
<td>1.00</td>
<td>.000</td>
</tr>
<tr>
<td>Constrained</td>
<td>35.09</td>
<td>39</td>
<td>.469</td>
<td>.900</td>
<td>1.00</td>
<td>.000</td>
</tr>
<tr>
<td>$\Delta \chi^2$</td>
<td>8.30</td>
<td>11</td>
<td>.685</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note. N = 333.*
Figure 5.9. Age comparisons of unconstrained path coefficients. Older participants path coefficients are presented in parenthesis. 

\(^a\) represents standardised paths, \(^b\) represents unstandardised paths, and an \(^*\) indicates that the larger path coefficient was significant for one age group but not for the other (\(p = < 0.05\)). Dashed lines represent correlations.
5.5 Discussion

5.5.1 Overall Findings

This study showed that having had prior emotional problems was a vulnerability factor for developing post-MVA psychological distress. This finding provides partial support for the first hypothesis and is consistent with past research (Benight et al., 2008; Blanchard et al., 2004; Blanchard, Hickling, Taylor et al., 1995; Ehlers et al., 1998; Ursano et al., 1999). Individuals with past emotional problems were also more likely to experience peritraumatic dissociation following an MVA. These results are consistent with the findings of previous MVA studies (Barton et al., 1996; Fullerton et al., 2000; Wittmann et al., 2006) and are also in accord with the cognitive model of PTSD presented by Ehlers and Clark (2000) which acknowledged that background factors influence initial emotional reactions, processing, and trauma appraisals.

Individuals who feared they were going to die and experienced dissociative symptoms during the MVA also developed psychological distress after the accident. This finding also provides partial support for the first hypothesis and is consistent with past research (Blanchard, Hickling, Mitnick et al., 1995; Jeavons et al., 2000; Ehlers et al., 1998; Murray et al., 2002) which has shown these phenomena are vulnerability factors. This supports the perspective that high levels of fear and dissociation potentiate posttraumatic symptoms through incomplete (data driven) processing and subsequent memory recall (van der Kolk & van der Hart, 1989) and resulting deficits (poor elaboration and integration) in trauma memories (Ehlers & Clarke, 2000; Foa & Hearst-Ikeda, 1996). However, there was no support for the perspective that dissociative reactions were adaptive and therefore
protective by reducing aversive trauma memories (see Horowitz, 1986; van der Kolk, 1987).

There was no evidence that level of alertness, perception of injury and accident severity had any direct effect on anxiety and depression symptoms post-MVA. Therefore, there was no support for this component of the first hypothesis. Instead level of alertness had an effect on dissociation and perceptions of injury, and accident severity had direct effects on fear. These findings are inconsistent with past research (Blanchard et al., 1996; 1997; Keane et al., 2006; March, 1993; Wittmann et al., 2006) which has suggested that both accident and injury severity affect psychological morbidity. However, they are consistent with the findings of other researchers (Bryant & Harvey, 1995; Bryant & Harvey, 1996; Ehlers et al., 1998; Mayou et al., 1997) who did not find this association. The current results are not completely surprising given inconsistent findings in past research which have resulted from different conceptualisations and assessment methods of trauma severity. Similarly, my assessment of injury may have been premature as the full extent of injury (appraisal) and implications on social and occupational functioning may not have been apparent at 1-month post-MVA. The results of this study also suggest that the relationships are complex since the results suggested that these factors indirectly influence psychological morbidity via fear and dissociative responses.

Since alertness has not previously been explored the findings regarding this variable are provisional but it seems that any influence is immediate and only contributes to dissociative symptoms. It is possible that a reduced level of alertness may contribute to a state of unreality and possible perceptual distortions (features of dissociation) that may result in
deficits in trauma memory such as poorly elaborated and integrated memories (see Ehlers & Clarke, 2000; Foa & Hearst-Ikeda, 1996). Given that fatigue is a contributing risk factor for MVA’s it is important that future studies assess this relationship which, if substantiated, may indicate that level of alertness is an additional vulnerability marker for peritraumatic dissociative responses following an MVA.

Past emotional problems had a greater direct effect than fear of dying on both measures of post-MVA psychological distress. Furthermore the direct effects of past emotional problems were greater than the indirect effects (via dissociation). However, the total effect of fear of dying and peritraumatic dissociation on anxious/fearful was greater than past emotional problems, but not for sad/depressed for which past emotional problems had the strongest effect. These findings provide partial support for the second hypothesis and are consistent with previous studies that have found peritraumatic factors to have the strongest effect on posttraumatic stress (Ozer et al., 2003), and with others which have found significant associations between past emotional problems and posttraumatic stress as well as depression (Ehring, Ehlers, & Glucksman, 2008; Shalev et al., 1998). One explanation for these findings is that the analyses used in the current study (which incorporated two outcome variables) allowed the individual effects of each variable to be partialled out while controlling for every other variable. Path analysis is a powerful statistical method that allows a clear differentiation of the unique contributions of each variable. Overall, these findings suggest that past emotional problems interact with acute trauma responses which increases the risk of post-MVA distress. The relationships between pre, peri, and post-trauma factors are consistent with the meditational model proposed by Brewin et al. (2000) and Barton et al. (1996).
It was found that higher levels of exposure (more severe accident and more severe injuries) contributed to psychological distress predominantly in the presence of extreme levels of fear responses and subsequent dissociative experiences. These findings provide partial support for the third hypothesis, that the influence of traumatic event characteristics on post-MVA distress would be mediated by dissociation, but demonstrate the complexity of the relationships. Previous research has shown that exposure is associated with dissociation (Bryant & Panasetis, 2005; Marmar et al., 1996) and that perception of life threat (fear) is associated with dissociation (Bryant & Panasetis, 2005; Fikretoglu et al., 2007; Nixon et al., 2002). However, in the current study both fear and dissociation had direct effects on levels of distress. This is not consistent with previous findings that fear is a mediator between dissociation and PTSD (Gershuny et al., 2003). Previous research has shown elements of these relationships but this is the first study that has incorporated all these variables in a path analysis to demonstrate the inter-relationships.

The current study found that fear was a cause not a consequence of dissociation, which provides partial support for the fourth hypothesis that fear would mediate the effects of the traumatic event on dissociation and thereby influence ongoing psychological distress. Again the findings are complex since fear directly influenced psychological distress but also indirectly through dissociation. Since no previous studies have explored these relationships with this level of complexity it is not possible to make direct comparisons with previous findings. However, contrary to past research (Gershuny et al., 2003), this study did not find any evidence that fears of death mediated the relationship between dissociation and posttraumatic stress. These findings are more consistent with the results
published by Nixon et al. (2002) who reported that life threat predicted dissociation. Thus they support the notion that intense peritraumatic fear elicits peritraumatic dissociation.

5.6 Demographics

5.6.1 Age

Individuals who were younger were more likely to be fatigued. Given that the potential causes of fatigue were not assessed it is difficult to ascertain why age had such an effect. It may be that older drivers are more aware of the dangers of driving whilst fatigued whereas younger less experienced drivers may be more inclined to take unnecessary risks and continue to drive when initially feeling tired and less alert. Future studies assessing fatigue may benefit from examining potential causes (e.g., number of hours driving, previous sleeping pattern, and use of medication and/or influence of psychotropic substances) which would help to educate drivers.

This study found that younger participants were more likely to experience greater peritraumatic dissociative symptoms, which is consistent with the results of previous studies (Fullerton et al., 2000; Groth-Marnat et al., 1999; Irwin, 1994; Marmar et al., 1996; Olff et al., 2007) and supports the age component of the fifth hypothesis. However, age was not associated with post-MVA distress. These results do not support the age component of the fifth hypothesis and are consistent with some previous MVA studies (Barth et al., 2005; Beck et al., 2006; Benight et al., 2008; Blanchard et al., 2004; Bryant & Harvey, 1995b; Freedman, Brandes, Peri, & Shalev, 1999; Hickling et al, 1998; Jeavons et al., 2000; Karl et al., 2009; Koren et al., 2001; Kupchik et al., 2007; Mayou et al., 2001; Ursano et al., 1999; Vaiva et al 2003; Zatzick, et al., 2002), but inconsistent with others which reported an age
effect (Conlon et al., 1999; Irish et al., 2008; Ongecha-Owuor et al., 2004). These findings
tend to support the conclusions by Kaniasty et al. (2002) who stated that there is no single
age effect as such, rather the effect of age is dependent upon multiple factors. It was noted
that in isolation age did have an effect on either fatigue or peritraumatic dissociation,
however in the presence of other variables, as revealed by the path analytic approach, there
was no overall age effect.

5.6.2 Gender

This study showed that females experienced a greater frequency and severity of post-MVA
distress compared to males. These results support the gender component of the fifth
hypothesis and are also consistent with the results of previous MVA studies (Blanchard., et
al., 1995, 1996, 2004; Coronas, García-Parés, Viladrich, Santos, & Menchón, 2008;
Dougall et al., 2001; Frommberger et al, 1998; Fullerton et al., 2001; Green, 1994;
Hickling, Gillen, Blanchard, Buckley & Taylor, 1998; Irish et al, 2008; Matsuoka et al.,
2008; Mayou, Ehlers & Bryant, 2002; Ongecha-Owuor, Kathuku, Othieno & Ndetei, 2004;
Stallard & Smith, 2006; Ursano et al., 1999; Wrenger, Lange, Langer, Heuft, & Burgmer,
2008; Zatzick et al., 2002).

It is well documented that females are more vulnerable to developing psychopathology
following trauma. However, explanations as to why females are at greater risk remain
inconclusive. The current findings indicated that females experienced more past emotional
problems which were found to directly influence acute dissociation, as well directly
influence post-MVA distress. Peritraumatic dissociation in turn was also found to directly
influence distress. From this it may be inferred that females are predisposed to post-trauma
psychopathology given their premorbid psychological state which affects dissociative and post-trauma outcomes. However, it is acknowledged that the influence of past emotional problems on dissociation (in comparison to the direct effect of both past emotional problems and dissociation on distress) is relatively weak.

Females experienced greater dissociative symptoms, which provides support for the gender component of the fifth hypothesis and is consistent with previous MVA studies (Bryant & Harvey, 2003b; Fullerton et al., 2001; Irish et al., 2011). Peritraumatic dissociation was strongly associated with post-MVA distress, however, factors influencing dissociation, such as, accident characteristics and particularly fear of dying, were not influenced by gender. These results pose more questions than they answer. For example, why do females have a greater tendency to experience acute dissociation, particularly given that fear is thought to elicit such a response? It was noted in section 5.2.8 that more females reported fear. However, females’ ratings of fear intensity were similar to those of males. Clearly other factors involved in acute dissociative reactions warrant further investigation. Gender affected aspects of the pre, peri, and post-MVA experience, but as evident from the nested path analysis comparisons, the overall the patterns of responses are similar for males and females. In the following chapter additional factors that have been shown to influence acute trauma reactions will be examined which may provide more meaningful results in relation to the influence of gender.

5.7 Limitations and Summary

The low response rate poses some concern as the demographic variables of age and gender do not represent the population of hospital MVA survivors. However, demographic biases
are not uncommon in the MVA literature. For example, Ehring, Ehlers, and Glucksman’s (2008) sample were significantly older than the hospital MVA population. Wu and Cheung’s (2006) sample comprised significantly younger and more severely injured participants compared to non-participants, and Mayou and Bryant, (2001) reported a greater proportion of females and older participants compared to non-MVA participants in their sample. Though perhaps more importantly, in terms of objectively assessed injury severity, which is often used as a measure of accident severity, it was found that there was no bias as injury severity ratings of the sample were quite similar to that of the population.

Reasons for non-participation are unknown. Following an MVA quite often a lot of paperwork needs to be completed. Treatment and hospital administration, police accident statements, and insurance related paperwork needs to be completed. Paperwork seen as non-essential (such as completing a research survey) may be avoided for this reason. Similarly, MVA survivors who are traumatised may not want to be reminded of their accident by answering survey items. Furthermore, MVA survivors who perceived their MVA as minor or who sustained very mild or no injury and who were not distressed by their MVA experience may have felt that the survey was not relevant to them.

The response rate was calculated by dividing the number of returned surveys by the number distributed. However, this does not take into factors that could not be controlled for. For example, it is unknown how many MVA survivors who received a survey were not proficient in English. Excluding these MVA survivors from the participant pool would have increased the response rate; unfortunately without access to this information the extent to which the response rate would have increased remains unknown. Additionally, the cross-
sectional and retrospective design somewhat limits conclusions about causal relationships and the use of unstandardised pre and post-accident morbidity measures preclude conclusions regarding diagnoses. The assessment of previous emotional problems was based on the simple rating scale used by Ehlers et al. (2008). It is acknowledged that the use of a single item limits the conclusion of results as the reliability and the estimation may be compromised. However, as the Phase 1 survey was deliberately kept short to improve the response rate, a formal assessment of previous psychopathology was not practicable. It is possible that the relationship between past emotion problems and post-MVA psychopathology (which was also assessed using a simple rating scale) would have been stronger had a formal assessment using standardised measures been conducted. In the following chapter the use of standardised outcome measures at three-month post-MVA will partly address these limitations and should provide a more robust assessment of the path analysis model.

Overall, these findings highlight the complex interactions between pre and peri-trauma influences and post-MVA reactions. The path analysis model developed in this study shows the important role of previous emotional vulnerability factors on initial post-MVA morbidity and the important mediating role of peri-dissociative experiences in the presence of extreme fear due to increasing levels of accident severity. Assessment of pre-accident morbidity and peritraumatic responses are important considerations when identifying individuals who are ‘at risk’ post-accident so that adequate monitoring and increased support can be provided to reduce the likelihood of developing chronic psychological morbidity.
Chapter 6

6.1 Phase 2 Study

6.1.1 Aims of the Study

The overall aim of the Phase 2 study was to describe the incidence of psychopathology (PTSD, depression and alcohol abuse) 3-months post-MVA as well as explore comorbid relationships. A further aim was to examine the relationships between variables assessed at 1-month (Phase 1) and those assessed at 3-months post-MVA (Phase 2) including the potential influence of gender and age. Expanding on the results from Phase 1, additional pre-trauma factors comprising personality characteristics, coping styles, and trait dissociation were examined in relation to their influence on acute peritraumatic reactions and post-MVA psychopathology.

This component of the study also aimed to provide additional support for the core path analysis model developed in Phase 1. Incorporating standardised PTSD and depression measures assessed at 3-months post-MVA will potentially validate the model and with time precedence established, causal pathways may be inferred.

Following a substantial amount of research on the role of peritraumatic dissociation in relation to the development of PTSD, recent questions regarding the predictive validity of peritraumatic dissociation as a global construct have arisen. An exploratory component of this study will be to examine the factor structure of the PDEQ and its relation to PTSD.
6.1.2 Hypothesis

The Phase 2 hypotheses are similar to Phase 1 in that they are based upon theoretical accounts of PTSD as well as empirical findings, and in part will expand on previous Phase 1 findings. The first hypotheses are in relation to pre-trauma factors and their relationship to peritraumatic dissociation and PTSD. Firstly, it was expected that a neurotic predisposition would predict peritraumatic dissociation and to a lesser extent PTSD. It was further hypothesised (2) that participants who have a general tendency to dissociate (trait dissociation) would be more likely to experience greater acute dissociative symptoms in response to trauma exposure. It was expected (3) that pre-trauma avoidant and emotion-focused coping style would influence immediate trauma reactions (peritraumatic dissociation) and would also be associated with PTSD. Furthermore, it was hypothesised (4) that an increased use of alcohol, particularly at hazardous consumption levels, would be associated with poorer post-MVA adjustment (PTSD and depression).

Though several previous studies have found strong associations between injury severity and PTSD, accident characteristics including injury severity showed no (direct) effect on initial post-MVA distress at Phase 1. However, physical disability following MVA injury has been shown to influence and maintain PTSD symptoms. It was hypothesised (5) that the relationship between injury severity and PTSD would be mediated by physical disability.
6.2 Results

6.2.1 Demographics

Of the 337 MVA survivors from Phase 1, 223 (66.2%) volunteered to participate in the follow-up survey. From the 223 surveys sent a total of 133 were returned, equating to a response rate of 59.6%. However, 5 surveys were returned blank or incomplete and therefore were not included in statistical analyses. Not all participants answered every question, therefore, the sample size in some analyses varies slightly. The sample ($N = 128$) comprised 99 participants (77%) from JHH and 29 participants (23%) from the Maitland Hospital. Collectively, there were 48 males and 80 females ranging in age from 18 years to 87 years of age ($M = 42.10, SD = 18.01$).

In order to ascertain if the participants from Phase 2 were representative of Phase 1 participants, direct comparisons were conducted. As shown in Table 6.1, significant differences were found; participants in Phase 2 tended to be older and included a greater proportion of females. They were also more anxious or fearful. These demographic differences are similar to those found between Phase 1 and the hospital MVA population (as noted in chapter 5).
Table 6.1

**Phase 1 and Phase 2 Pre, Peri, and Post-MVA Variable Comparisons**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 209)</td>
<td>(n = 128)</td>
<td>(\chi^2)</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td>6.85**</td>
</tr>
<tr>
<td>Male</td>
<td>109 (52.2%)</td>
<td>48 (37.5%)</td>
<td>-</td>
</tr>
<tr>
<td>Female</td>
<td>100 (48.8%)</td>
<td>80 (62.5%)</td>
<td>-</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>Mean (SD)</td>
<td>37.63 (17.42)</td>
<td>42.10 (18.01)</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td>-</td>
<td>-</td>
<td>3.70 ns</td>
</tr>
<tr>
<td><strong>Income</strong></td>
<td>-</td>
<td>-</td>
<td>3.46 ns</td>
</tr>
<tr>
<td><strong>Previous MVA`s (Yes %)</strong></td>
<td>112 (56.3%)</td>
<td>74 (57.8%)</td>
<td>.573 ns</td>
</tr>
<tr>
<td><strong>Number of Previous MVA`s</strong></td>
<td>Mean (SD)</td>
<td>1.64 (1.41)</td>
<td>1.07 (1.15)</td>
</tr>
<tr>
<td><strong>Previous MVA Severity Rating</strong></td>
<td>Mean (SD)</td>
<td>1.22 (1.30)</td>
<td>1.24 (1.37)</td>
</tr>
<tr>
<td><strong>Past Emotional Problems</strong></td>
<td>Mean (SD)</td>
<td>1.63 (.997)</td>
<td>1.60 (.983)</td>
</tr>
<tr>
<td><strong>MVA Category</strong></td>
<td></td>
<td></td>
<td>8.52 ns</td>
</tr>
<tr>
<td>Driver</td>
<td>106 (50.7%)</td>
<td>84 (65.6%)</td>
<td>-</td>
</tr>
<tr>
<td>Passenger</td>
<td>47 (22.5%)</td>
<td>23 (18.0%)</td>
<td>-</td>
</tr>
<tr>
<td>Motorcyclist/pillion</td>
<td>33 (15.8%)</td>
<td>15 (11.7%)</td>
<td>-</td>
</tr>
<tr>
<td>Pedal Cyclist</td>
<td>16 (7.7%)</td>
<td>4 (3.1%)</td>
<td>-</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>7 (3.3%)</td>
<td>2 (1.6%)</td>
<td>-</td>
</tr>
<tr>
<td><strong>Injury (Yes %)</strong></td>
<td>196 (93.8%)</td>
<td>121 (94.5%)</td>
<td>.080 ns</td>
</tr>
<tr>
<td><strong>Injury Severity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self Report</td>
<td>Mean (SD)</td>
<td>2.85 (.905)</td>
<td>2.90 (.831)</td>
</tr>
<tr>
<td>ISS</td>
<td>Mean (SD)</td>
<td>9.75 (8.67)</td>
<td>8.70 (6.73)</td>
</tr>
<tr>
<td>Accident</td>
<td>Mean (SD)</td>
<td>3.08 (1.04)</td>
<td>3.04 (.900)</td>
</tr>
<tr>
<td><strong>Severity Alertness</strong></td>
<td>Mean (SD)</td>
<td>1.93 (1.43)</td>
<td>1.70 (1.16)</td>
</tr>
<tr>
<td>Fear of Dying</td>
<td>Mean (SD)</td>
<td>2.70 (1.36)</td>
<td>2.75 (1.32)</td>
</tr>
<tr>
<td>Peritraumatic Dissociation</td>
<td>Mean (SD)</td>
<td>21.21 (9.58)</td>
<td>21.79 (9.41)</td>
</tr>
<tr>
<td>Anxious/Fearful</td>
<td>Mean (SD)</td>
<td>2.18 (1.18)</td>
<td>2.47 (1.26)</td>
</tr>
<tr>
<td>Sad/Depressed</td>
<td>Mean (SD)</td>
<td>2.24 (1.20)</td>
<td>2.41 (1.33)</td>
</tr>
</tbody>
</table>

**Note.** ISS = Injury Severity Score.

\(^a^Welsch-Satterthwaite corrected. \(^b^MVA Severity Rating compares current MVA severity to previous MVAs 1 = not as bad, 2 = same, 3 = worse.

\(^*p<.05. \,**p<.01.\)}
6.2.2 Treatment

A total of 111 (87.4%) participants reported that they had received treatment due to their MVA, with 86% \( (n = 86) \) having received treatment for physical injuries, 2% \( (n = 2) \) having received psychological treatment and 20% \( (n = 22) \) of participants having received both physical and psychological treatment. Duration of treatment varied with 19% \( (n = 21) \) of participants having received treatment for 1 day, 15% \( (n = 16) \) for up to 1 week, 7% \( (n = 8) \) up to 1 month, 25% \( (n = 27) \) for between 1 and 3 months and 34% \( (n = 37) \) of participants reported having received treatment for a period longer than 3 months. There were no significant differences between treatment duration and treatment type or between injury severity and treatment type. However, there was a significant main effect of injury severity on treatment duration, \( F(2, 106) = 9.94, p = .001 \). Post-hoc comparisons using Tukey’s test, with a familywise Type I error rate of .05, showed that participants who reported that their injuries were moderate or severe received treatment for a greater duration than those who sustained mild injuries. Demographic comparisons showed that neither gender nor age had an effect on receiving treatment, treatment type, or treatment duration.

6.2.3 Physical Disability

Physical disability scores ranged from 0 to 22 \( (M = 6.97, SD = 6.41) \). Using the predefined disability categories, 34% \( (n = 43) \) of participants reported no disability, 10% \( (n = 13) \) mild, 21% \( (n = 27) \) moderate, and 35% \( (n = 34) \) were categorised as severe. There was a significant main effect of disability on treatment duration, \( F(4, 111) = 9.88, p = .001 \). Post-hoc comparisons (using Tukey’s test, with a familywise Type I error rate of .05), showed that participants who had received treatment for 3-months or longer had significantly greater
disability scores than participants who had received treatment for a lesser period (see Figure 6.1).

![Bar chart showing mean disability scores across different treatment duration categories.]

*Figure 6.1.* Treatment duration categories and disability score means.

Similarly, (though not surprising) injury severity also showed a significant main effect, $F (3, 124) = 11.87, p = .001$. Post-hoc comparisons using Tukey’s test, with a familywise Type I error rate of .05, revealed that participants categorised as having no disability had significantly lower injury mean scores than participants with mild, moderate and severe disability. Examination of post-MVA distress and disability (illustrated in Figure 6.2) showed significant main effects: $F (3, 124) = 7.60, p = .001$ (anxious or fearful); $F (3, 124) = 11.57, p = .001$ (sad or depressed).
Post-hoc analysis (using Tukey’s test, with a familywise Type I error rate of .05) revealed that participants with severe disability had significantly greater anxious or fearful scores than participants with moderate, mild and no disability. Similarly, MVA survivors with severe disability also had significantly greater sad or depressed scores than those categorised with mild or no disability. There were no significant differences between gender, age, or MVA category type and disability scores.

Figure 6.2. Physical disability categories and post-MVA distress levels.
6.3 Psychopathology

6.3.1 Posttraumatic Stress Disorder and Pre-MVA Factors

At 3-months post-MVA 37.6% of MVA survivors \( n = 47 \) could be diagnosed with PTSD based on the PDS (DSM-IV) criteria. No gender differences were found in the proportion of participants with or without PTSD nor was there any significant difference between gender and symptom severity scores. Using a median split (43 years) to define young and old participants, younger MVA survivors experienced greater PTSD symptom severity \( M = 14.45 \) compared to older participants \( M = 10.17 \), \( t (122) = 2.18, p = .031 \), but there was no significant age difference between participants with and without PTSD. Similarly, education and income level, previous MVAs (including number and severity), past emotional problems, extraversion, as well as task and avoidance coping score comparisons were not significantly different between participants with and without PTSD. However, as shown in Table 6.2 participants with PTSD scored significantly higher on the pre-MVA measures of neuroticism, emotion-focused coping, and trait dissociation compared to participants without PTSD.

6.3.2 Posttraumatic Stress Disorder and MVA Characteristics

As shown in Table 6.3 comparisons on MVA category type, injury status, subjective and objective injury severity, accident severity, and level of alertness showed no significant differences between participants with and without PTSD. However, the mean scores on fear of dying and peritraumatic dissociation were significantly greater for participants with PTSD.
Table 6.2

Pre-MVA Variable Comparisons Between Participants With and Without PTSD

<table>
<thead>
<tr>
<th>Pre MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>47 (37.6)</td>
<td>78 (62.4)</td>
<td>-</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td>$\chi^2 = 3.17 \ ns$</td>
</tr>
<tr>
<td>Male n (%)</td>
<td>13 (27.7%)</td>
<td>34 (43.6%)</td>
<td>-</td>
</tr>
<tr>
<td>Female n (%)</td>
<td>34 (72.3%)</td>
<td>44 (56.4%)</td>
<td>-</td>
</tr>
<tr>
<td>Age</td>
<td>Mean (SD)</td>
<td>38.87 (13.83)</td>
<td>43.69 (19.46)</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td>$\chi^2 = 2.69 \ ns$</td>
</tr>
<tr>
<td>Income</td>
<td></td>
<td></td>
<td>$\chi = 3.23 \ ns$</td>
</tr>
<tr>
<td>Previous MVA`s (Yes %)</td>
<td>28 (59.6%)</td>
<td>45 (57.7%)</td>
<td>$\chi^2 = .043 \ ns$</td>
</tr>
<tr>
<td>Number of previous MVA`s</td>
<td>Mean (SD)</td>
<td>1.40 (.496)</td>
<td>1.42 (.497)</td>
</tr>
<tr>
<td>Previous MVA Severity Rating(^a)</td>
<td></td>
<td></td>
<td>$p &gt; .05 \ b$</td>
</tr>
<tr>
<td>Not as bad</td>
<td>10 (35.7%)</td>
<td>7 (15.6%)</td>
<td>-</td>
</tr>
<tr>
<td>Same</td>
<td>2 (7.1%)</td>
<td>4 (8.9%)</td>
<td>-</td>
</tr>
<tr>
<td>Worse</td>
<td>16 (57.2%)</td>
<td>34 (68.5%)</td>
<td>-</td>
</tr>
<tr>
<td>Past Emotional Problems</td>
<td>Mean (SD)</td>
<td>1.79 (.931)</td>
<td>1.50 (1.01)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>Mean (SD)</td>
<td>4.34(2.09)</td>
<td>4.91 (2.00)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Mean (SD)</td>
<td>5.20 (2.11)</td>
<td>3.90 (2.26)</td>
</tr>
<tr>
<td>Task coping</td>
<td>Mean (SD)</td>
<td>50.85 (12.24)</td>
<td>54.85 (12.02)</td>
</tr>
<tr>
<td>Emotion coping</td>
<td>Mean (SD)</td>
<td>47.64 (11.87)</td>
<td>38.81 (11.71)</td>
</tr>
<tr>
<td>Avoidance coping</td>
<td>Mean (SD)</td>
<td>45.38 (9.95)</td>
<td>43.24 (11.34)</td>
</tr>
<tr>
<td>Trait Dissociation</td>
<td>Mean (SD)</td>
<td>47.38 (26.37)</td>
<td>27.53 (19.29)</td>
</tr>
</tbody>
</table>

*Note.* \(^a\) MVA Severity Rating compares current MVA severity to previous MVAs 1 = not as bad, 2 = same, 3 = worse. \(^b\) Fisher’s Exact Test.

**\(p < .01\). ***\(p < .001\).
Table 6.3

Comparisons of MVA Characteristics Between Participants With and Without PTSD

<table>
<thead>
<tr>
<th>MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>47 (37.6)</td>
<td>78 (62.4)</td>
<td>-</td>
</tr>
<tr>
<td>MVA Category</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Driver</td>
<td>30 (63.8%)</td>
<td>52 (66.7%)</td>
<td>$p &gt; .05$</td>
</tr>
<tr>
<td>Passenger</td>
<td>8 (17%)</td>
<td>14 (17.9%)</td>
<td>-</td>
</tr>
<tr>
<td>Motorcyclist/pillion</td>
<td>7 (15%)</td>
<td>8 (10.3%)</td>
<td>-</td>
</tr>
<tr>
<td>Pedal Cyclist</td>
<td>1 (2.1%)</td>
<td>3 (3.8%)</td>
<td>-</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>1 (2.1%)</td>
<td>1 (1.3%)</td>
<td>-</td>
</tr>
<tr>
<td>Injury (Yes %)</td>
<td>46 (39%)</td>
<td>73 (61%)</td>
<td>$p &gt; .05$</td>
</tr>
<tr>
<td>Injury Severity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report</td>
<td>Mean (SD)</td>
<td>3.02 (.707)</td>
<td>2.85 (.869)</td>
</tr>
<tr>
<td>ISS</td>
<td>Mean (SD)</td>
<td>7.83 (6.61)</td>
<td>9.07 (7.00)</td>
</tr>
<tr>
<td>Accident Severity</td>
<td>Mean (SD)</td>
<td>3.19 (.992)</td>
<td>2.95 (.836)</td>
</tr>
<tr>
<td>Alertness</td>
<td>Mean (SD)</td>
<td>1.74 (1.37)</td>
<td>1.65 (1.00)</td>
</tr>
<tr>
<td>Fear of Dying</td>
<td>Mean (SD)</td>
<td>3.09 (1.15)</td>
<td>2.57 (1.37)</td>
</tr>
<tr>
<td>Peritraumatic Dissociation</td>
<td>Mean (SD)</td>
<td>25.94 (9.01)</td>
<td>19.33 (8.86)</td>
</tr>
</tbody>
</table>

Note. ISS = Injury Severity Score.
$^a$Fisher’s Exact Test. $^b$Welsch-Satterthwaite corrected.
* $p < 0.05$. ** $p < 0.001$.

6.3.3 Posttraumatic Stress Disorder and Post-MVA Factors

The results (see Table 6.4) of comparisons of post-accident variables showed that the mean scores for psychological distress (assessed at 30-days post-MVA) were significantly greater for participants who developed PTSD compared to those who did not. Though there was no difference in the proportion of participants (with or without PTSD) who received treatment,
those with PTSD received treatment for a significantly longer period. Comparisons of treatment type showed significant differences with a greater proportion of participants without PTSD (93.8%) receiving treatment for physical injuries and a greater proportion of participants with PTSD (42.2%) receiving treatment for both emotional and physical injury (see Table 6.4).

Further comparisons showed that participants with PTSD scored significantly higher on depression symptom severity (assessed with the BDI-II) and physical disability (assessed with the Brief Disability Questionnaire) compared to participants without PTSD. Analysis of disability categories revealed significant differences with 46.2% of participants without PTSD categorised with no disability and 55.3% of participants with PTSD with severe disability. Comparisons of alcohol use mean scores (assessed using the AUDIT) showed that MVA-survivors with PTSD scored significantly higher than those without PTSD. Analysis of the category proportions of alcohol use since the accident (Decreased, Same or Increased) showed significant differences. Examination of the adjusted residuals¹ showed the largest difference (+/- 3.1) was within the Same category comprising significantly more participants without PTSD. There was also a significant difference (+/- 2.5) within the Increased category comprising more participants with PTSD, as displayed in Table 6.4.

¹ Adjusted (standardised) residuals provide a test for the significance of individual cells. Adjusted residuals that are greater than the absolute value of 2.00 are deemed to make a significant contribution to the overall chi-square value.
Table 6.4

*Comparisons of Post-MVA Characteristics Between Participants With and Without PTSD*

<table>
<thead>
<tr>
<th>Post-MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>47 (37.6%)</td>
<td>78 (62.4%)</td>
<td></td>
</tr>
<tr>
<td>Sad/Depressed</td>
<td>Mean (SD)</td>
<td></td>
<td>t = 5.72***</td>
</tr>
<tr>
<td></td>
<td>3.30 (1.15)</td>
<td>1.91 (1.16)</td>
<td></td>
</tr>
<tr>
<td>Anxious/Fearful</td>
<td>Mean (SD)</td>
<td></td>
<td>t = 6.46***</td>
</tr>
<tr>
<td></td>
<td>3.23 (1.25)</td>
<td>2.04 (1.05)</td>
<td></td>
</tr>
<tr>
<td>Treatment (Yes %)</td>
<td>44 (93.6%)</td>
<td>65 (83.3%)</td>
<td>χ² = 2.77 ns</td>
</tr>
<tr>
<td>Treatment Type</td>
<td>p &lt; .001 a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional</td>
<td>1 (2.2%)</td>
<td>1 (1.5%)</td>
<td></td>
</tr>
<tr>
<td>Physical</td>
<td>25 (55.6%)</td>
<td>60 (93.8%)</td>
<td></td>
</tr>
<tr>
<td>Emotional and Physical</td>
<td>19 (42.2%)</td>
<td>3 (4.7%)</td>
<td></td>
</tr>
<tr>
<td>Treatment Duration</td>
<td>Mean (SD)</td>
<td></td>
<td>t = 2.16*</td>
</tr>
<tr>
<td></td>
<td>3.77 (1.42)</td>
<td>3.13 (1.58)</td>
<td></td>
</tr>
<tr>
<td>Physical Disability</td>
<td>Mean (SD)</td>
<td></td>
<td>t = 5.35***</td>
</tr>
<tr>
<td></td>
<td>10.45 (6.08)</td>
<td>4.85 (5.39)</td>
<td></td>
</tr>
<tr>
<td>Physical Disability Rating</td>
<td>p &lt; .001 a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>6 (12.8%)</td>
<td>36 (46.2%)</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>2 (4.2%)</td>
<td>10 (12.8%)</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>13 (27.7%)</td>
<td>15 (19.2%)</td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>26 (55.3%)</td>
<td>17 (21.8%)</td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td>Mean (SD)</td>
<td></td>
<td>t = 7.42*** b</td>
</tr>
<tr>
<td></td>
<td>19.43 (10.59)</td>
<td>6.50 (7.07)</td>
<td></td>
</tr>
<tr>
<td>AUDIT</td>
<td>Mean (SD)</td>
<td></td>
<td>t = 2.85* b</td>
</tr>
<tr>
<td></td>
<td>7.79 (8.39)</td>
<td>4.35 (4.62)</td>
<td></td>
</tr>
<tr>
<td>Alcohol use post-MVA</td>
<td>p &lt; .01 a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased</td>
<td>9 (19.1%)</td>
<td>7 (9.1%)</td>
<td></td>
</tr>
<tr>
<td>Same</td>
<td>29 (61.8%)</td>
<td>66 (85.7%)</td>
<td></td>
</tr>
<tr>
<td>Increased</td>
<td>9 (19.1%)</td>
<td>4 (5.2%)</td>
<td></td>
</tr>
</tbody>
</table>

*Note. BDI II= Beck Depression Inventory Second Edition. AUDIT = Alcohol Use Disorders Identification Test.  
a Fisher’s Exact Test. b Welsch-Satterthwaite corrected.  
*p < .05. **p < .01. ***p < .001.*
6.3.4 Alcohol, Depression, and Comorbidity

Hazardous levels of alcohol consumption (AUDIT score equal to or greater than 8) was reported by 27.3% ($n = 35$) of participants. Analysis of alcohol consumption patterns, which are displayed in Table 6.5, showed that 12.6% ($n = 16$) of participants had decreased their alcohol consumption since their MVA, 72.2% ($n = 98$) remained the same, and 10.2% ($n = 13$) had increased their alcohol intake. Examination of participants’ consumption patterns whose alcohol intake was categorised as hazardous revealed no significant differences between participants with and without PTSD, nor with and without depression. There was no significant difference in PTSD symptom severity scores between participants who consumed and those who did not consume a hazardous level of alcohol. However, participants whose alcohol intake was hazardous ($M = 15.37$) reported significantly higher depression scores in comparison to participants whose alcohol intake was not hazardous ($M = 9.69$), ($t (123) = 2.75$, $p = .007$).

Table 6.5

*Summary of Alcohol Usage Patterns in MVA Survivors With and Without PTSD and Depression*

<table>
<thead>
<tr>
<th>Usage Pattern</th>
<th>$N$</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Depression</th>
<th>No Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased</td>
<td>16 (6)</td>
<td>9 (3)</td>
<td>7 (3)</td>
<td>5 (3)</td>
<td>11 (3)</td>
</tr>
<tr>
<td>Same</td>
<td>95 (19)</td>
<td>29 (7)</td>
<td>66 (12)</td>
<td>16 (5)</td>
<td>79 (14)</td>
</tr>
<tr>
<td>Increased</td>
<td>13 (10)</td>
<td>9 (6)</td>
<td>4 (4)</td>
<td>5 (4)</td>
<td>7 (6)</td>
</tr>
<tr>
<td>Total</td>
<td>124 (35)</td>
<td>47 (16)</td>
<td>77 (19)</td>
<td>26 (12)</td>
<td>98 (23)</td>
</tr>
</tbody>
</table>

*Note.* Numbers in parenthesis represent hazardous alcohol consumption levels.
There was no significant difference in the proportion of participants who did or did not report hazardous alcohol intake and who did or did not report having experienced past emotional problems. However, examination of past emotional problem categories showed that participants who were extremely bothered by past emotion problems reported significantly higher AUDIT scores in comparison to participants who were not at all, a little, somewhat and bothered a lot by past emotional problems, \((F(4, 122) = 6.93, p = .001)\). Further analysis of pre-MVA variable (mean) scores revealed that participants who were consuming alcohol at a hazardous level scored significantly higher on: neuroticism (5.3 vs. 4.90), \(t(125) = 2.91, p = .004\); emotion-focused coping (47.04 vs. 40.59), \(t(125) = 2.70, p = .008\); and trait dissociation (45.88 vs. 30.76), \(t(125) = 3.28, p = .001\).

Gender comparisons revealed no significant differences on consumption patterns (decreased, same, and increased) or on hazardous level. However, participants categorised as consuming a hazardous level of alcohol were significantly younger than participants who were not consuming at a hazardous level (32.11 years vs. 45.86 years respectively), \(t(126) = -4.07, p = .001\).

Depression (BDI-II score equal to or greater than 20) was reported by 20.5% \((n = 26)\) of participants. No gender or age differences were found between participants categorised with or without depression. A comparison between the proportion of participants with depression and those who had also experienced past emotional problems was significant \((\chi^2 (1, N = 125) = 4.10, p = .043)\), with 53.8% of participants \((n = 14)\) with depression also having experienced past emotional problems.
As illustrated in Figure 6.3, 42.6\% of participants \((n = 20)\) with PTSD experienced comorbid depression, and 34\% \((n = 16)\) reported concurrent hazardous alcohol use. Forty six-point two percent \((n = 12)\) of participants with depression also reported concurrent hazardous alcohol use and nine participants \(7\%\) reported comorbid PTSD, depression, and hazardous alcohol use.

![Venn diagram: PTSD, Depression, Alcohol Abuse comorbid inter-relationships.](image)

**Figure 6.3.** Venn diagram: PTSD, Depression, Alcohol Abuse comorbid inter-relationships.

### 6.3.5 Predictors of Peritraumatic Dissociation

Expanding on the results from Phase 1, additional background factors were included in a path analysis model to examine the interaction and strength of proposed relationships with peritraumatic dissociation. The path model was developed in relation to specific hypothesis according to empirical findings using the same path analytic procedures employed in Phase 1 (see section 5.3.1). Correlation coefficients were examined to determine if relationships between variables of interest were significant. As shown in the correlation matrix (see
Table 6.6) level of alertness and past emotional problems, which were found to be significant in Phase 1, were not significantly associated with peritraumatic dissociation at Phase 2 and therefore were not included in the path analysis model. Age and gender were included in the Phase 1 model as separate group comparisons, however these were not possible at Phase 2 due to the reduced sample size. Additionally, the direction of relationships (path coefficients) between accident characteristics (fear of dying, accident, and injury severity) established at Phase 1 (see Figure 5.8) were integrated into the initial Phase 2 model.

Skew and kurtosis levels of the model were within acceptable limits (< 2.0 & 4.0 respectively), however the initial model did not meet the goodness-of-fit criteria, $\chi^2 = 213.56, p < .001 (df = 36; N = 124), \chi^2/df = 5.93, CFI = .250; RMSEA = .200, AIC = 271.56$. Examination of the standardised path coefficients showed that age ($\beta = -.16, p = .100$), gender ($\beta = .16, p = .068$), emotion-focused ($\beta = .21, p = .058$), avoidance coping ($\beta = .06, p = .422$), trait dissociation ($\beta = .04, p = .836$), and neuroticism ($\beta = .05, p = .501$) did not significantly load onto peritraumatic dissociation. The path coefficient between injury severity and fear of dying was also not significant ($\beta = .12, p = .302$). These paths were removed with the exception of emotion-focused coping which showed a strong trend. To further improve model fit (based on the recommendations of Modification Indices) trait dissociation and neuroticism were allowed to covary and paths from neuroticism and trait dissociation to emotion-focused coping were added. The respecified model (shown in Figure 6.4) provided an excellent fit of the data, $\chi^2 = 7.61, p = .573 (df = 9; N = 124), \chi^2/df = 0.846, CFI = 1.000; RMSEA = .000, AIC = 43.61$. 
Table 6.6

*CORRELATION COEFFICIENTS OF PRE, PERI, AND POST-MVA CHARACTERISTICS ASSOCIATED WITH 3-MONTH PTSD AND DEPRESSION SYMPTOM SEVERITY*

<table>
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<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
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<td>1. Age</td>
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<td>-.06</td>
<td>-.12</td>
<td>-.23*</td>
<td>-.47**</td>
<td>-.07</td>
<td>-.06</td>
<td>-.11</td>
<td>-.24**</td>
</tr>
<tr>
<td>2. Gender</td>
<td>-</td>
<td>.18*</td>
<td>.06</td>
<td>.17</td>
<td>.19*</td>
<td>.05</td>
<td>-.30**</td>
<td>-.11</td>
<td>-.05</td>
<td></td>
</tr>
<tr>
<td>3. Past Emotional Problems</td>
<td>-</td>
<td>.27**</td>
<td>.38**</td>
<td>-.11</td>
<td>.37**</td>
<td>-.01</td>
<td>.02</td>
<td>.18*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Neuroticism a</td>
<td>-</td>
<td>.56**</td>
<td>.04</td>
<td>.59**</td>
<td>-.03</td>
<td>-.05</td>
<td>.14</td>
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<tr>
<td>5. Emotion-Focused Coping a</td>
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<td>.64**</td>
<td>-.13</td>
<td>.13</td>
<td>.20*</td>
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<td>-.14</td>
<td>.09</td>
<td>.15</td>
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<td>7. Trait Dissociation a</td>
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<td>-.05</td>
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<tr>
<td>8. Injury Severity</td>
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<td>.28**</td>
<td>-.01</td>
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<tr>
<td>9. Accident Severity</td>
<td>-</td>
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<tr>
<td>10. Level of Alertness</td>
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<tr>
<td>11. Fear of Dying</td>
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<td>12. Peritraumatic Dissociation</td>
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<tr>
<td>13. Sad or Depressed</td>
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<tr>
<td>14. Anxious or Fearful</td>
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<tr>
<td>15. Physical Disability a</td>
<td></td>
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<tr>
<td>17. PTSD Symptom Severity a</td>
<td></td>
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<tr>
<td>18. Depression Symptom Severity a</td>
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</tbody>
</table>

*Note. N = 128. AUDIT = Alcohol Use Disorders Identification Test. PTSD = Posttraumatic Stress Disorder. a Assessed at 3-months post-MVA. *p < .05. **p < .01.*
Correlation Coefficients of Pre, Peri, and Post-MVA Characteristics Associated with 3-Month PTSD and Depression Symptom Severity (continued)

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<tr>
<th>Variable</th>
<th>11</th>
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<th>13</th>
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<th>15</th>
<th>16</th>
<th>17</th>
<th>18</th>
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<td>.17</td>
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<td>-.10</td>
<td>.20*</td>
<td>.07</td>
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<tr>
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<td>.13</td>
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<td>.25**</td>
<td>.13</td>
<td>.07</td>
<td>.31*</td>
<td>.20*</td>
<td>.36**</td>
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<td>.35**</td>
<td>.36**</td>
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<td>.13</td>
<td>.37**</td>
<td>.38**</td>
<td>.48**</td>
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<td>.20*</td>
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<td>.04</td>
<td>-.14</td>
<td>.03</td>
<td>.07</td>
<td>-.04</td>
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<td>.25**</td>
<td>.21*</td>
<td>.35**</td>
<td>.41**</td>
<td>.58**</td>
</tr>
<tr>
<td>8. Injury Severity</td>
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<td>.06</td>
<td>.14 c</td>
<td>.11</td>
<td>.48**</td>
<td>.04</td>
<td>.15</td>
<td>.05</td>
</tr>
<tr>
<td>9. Accident Severity</td>
<td>.41**</td>
<td>.15 c</td>
<td>.16 c</td>
<td>.18*</td>
<td>.27**</td>
<td>.21*</td>
<td>.29**</td>
<td>.23**</td>
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<td>.02</td>
<td>-.06</td>
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<td>.33**</td>
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<td>.13</td>
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<td>11. Fear of Dying</td>
<td>-</td>
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<td>.28**</td>
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<td>.34**</td>
<td>.14</td>
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<td>.36**</td>
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<td>.51**</td>
<td>.32**</td>
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</tr>
<tr>
<td>13. Sad or Depressed</td>
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<td>.81**</td>
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<td>.65**</td>
<td>.64**</td>
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<tr>
<td>14. Anxious or Fearful</td>
<td>-</td>
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<td>.21*</td>
<td>.61**</td>
<td>.53**</td>
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<td>15. Physical Disability a</td>
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<td>.58**</td>
<td>.54**</td>
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<td>.42**</td>
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<tr>
<td>17. PTSD Symptom Severity a</td>
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<td>.75**</td>
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<tr>
<td>18. Depression Symptom Severity a</td>
<td>-</td>
<td>-</td>
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</tbody>
</table>

Note. $N = 128$. AUDIT = Alcohol Use Disorders Identification Test. PTSD = Posttraumatic Stress Disorder. 
*Assessed at 3-months post-MVA. c Significant correlation in $n = 333$ sample.

*p < .05. **p < .01.
Figure 6.4. Path analysis model with standardized path coefficients for background variables and accident characteristics influencing peritraumatic dissociation. All paths are significant ($p < .05$). The dashed line represents a correlation.
The final model (illustrated in Figure 6.4) shows two distinct pathways influencing peritraumatic dissociation. Similar to the Phase 1 model, fear of dying directly influenced peritraumatic dissociation ($\beta = .34, p = .012$), and fully mediated the relationship between accident severity ($\beta = .42, p = .011$) and peritraumatic dissociation. Emotion-focused coping also directly influenced peritraumatic dissociation ($\beta = .33, p = .010$), and mediated the relationships between trait dissociation ($\beta = .52, p = .005$), neuroticism ($\beta = .22, p = .013$) and peritraumatic dissociation.

6.4 Factor Structure of the PDEQ

6.4.1 Exploratory Factor Analysis

Exploratory factor analysis (EFA) was conducted on the 10 items of the PDEQ (Peritraumatic Dissociative Experiences Questionnaire). Given that the word length of some of the items was quite long an abbreviated item description is used in Table 6.8. Similarly, reference to item numbers as opposed to complete item wording is used in Table 6.7 as well as in the text. For the complete item wording see Appendix B.

Prior to analysis the factorability of the PDEQ items was examined. As displayed in Table 6.7, all 10 items were significantly correlated ($p < .01$) with the majority of correlations above 0.3. The Kaiser-Meyer-Olkin measure of sampling adequacy was above the recommended value (of .70) at .862. Bartlett’s test of sphericity was significant ($\chi^2 (45) = 1451.70, p = .001$) supporting the factorability of the correlation matrix (Leech, Barrett, & Morgan, 2005).
Table 6.7

*Intercorrelations Between Peritraumatic Dissociative Experiences Questionnaire Items*

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
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</thead>
<tbody>
<tr>
<td>Item 1. Losing track of what was going on</td>
<td>-</td>
<td>.50**</td>
<td>.44**</td>
<td>.48**</td>
<td>.31**</td>
<td>.48**</td>
<td>.27**</td>
<td>.61**</td>
<td>.58**</td>
<td>.62**</td>
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<tr>
<td>Item 2. I was on ‘automatic pilot’</td>
<td>-</td>
<td>.49**</td>
<td>.39**</td>
<td>.25**</td>
<td>.23**</td>
<td>.27**</td>
<td>.42**</td>
<td>.48**</td>
<td>.46**</td>
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<td>Item 3. Sense of time changed</td>
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<td>.40**</td>
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<td>.41**</td>
<td>.38**</td>
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<tr>
<td>Item 4. Seem unreal…dream or movie</td>
<td>-</td>
<td>.56**</td>
<td>.39**</td>
<td>.33**</td>
<td>.43**</td>
<td>.46**</td>
<td>.44**</td>
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<tr>
<td>Item 5. Spectator watching what was happening</td>
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<td>.41**</td>
<td>.33**</td>
<td>.28**</td>
<td>.25**</td>
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<td></td>
</tr>
<tr>
<td>Item 6. Body seemed distorted or changed</td>
<td>-</td>
<td>.59**</td>
<td>.34**</td>
<td>.43**</td>
<td>.35**</td>
<td></td>
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</tr>
<tr>
<td>Item 7. Felt things happening to others were happening to me</td>
<td>-</td>
<td>.31**</td>
<td>.38**</td>
<td>.34**</td>
<td></td>
<td></td>
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<tr>
<td>Item 8. Surprised to find out afterwards lots of things</td>
<td>-</td>
<td>.57**</td>
<td>.56**</td>
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<td>Item 9. I felt confused…difficulty making sense</td>
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<td>Item 10. I felt disoriented…uncertain where I was</td>
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*Note.* $N = 325$. 

180
Prior to analysis the 10 items were checked for violations of normality with all items found to have skew levels less than 2 and kurtosis levels less than 3, with the exception of item 7 which showed problematic levels of skew (3.00), and kurtosis (8.56). Further examination of the distribution of item 7 scores showed that 83.6% \((n = 275)\) of participants reported *not at all true* for this item, \((M = 1.33, SD = .870)\).

Given the varied guidelines on what constitutes acceptable limits of skew and kurtosis, an approach was adapted incorporating the recommendations by Curran, West, and Finch, (1996), Fabrigar, Wegener, MacCallum, and Strahan, (1999), and Kline, (2005), using a skew of less than 2 and kurtosis less than 5. A log transformation was conducted on item 7 which reduced the skew to 2.36 and kurtosis to 4.43. However, as the item still showed a departure from normality, principal axis factoring extraction method was used as it is not reliant on distribution assumptions (Costello & Osborne, 2005; Fabrigar et al.). Promax, an oblique rotation method was employed as factors derived from a presumed single construct were expected to be correlated.

Analysis comprised an initial examination of a 1 factor structure. As shown in Table 6.8, the initial single factor had a Cronbach’s alpha of .88, an eigenvalue of 4.88, and explained 48.82% of the variance with all items loading above .5. Analysis was then conducted using eigenvalues of greater than 1 to determine factors. The results revealed a 2 factor solution. Factor 1 explained the same amount of variance and eigenvalue as the single factor solution and factor 2 had an eigenvalue of 1.22 and explained (cumulative percentage) 61.11% of the variance.
Table 6.8

*Exploratory Factor Analysis With Promax Rotated Factor Loadings of the PDEQ Items using a 1 Factor and 2 Factor Structure*

<table>
<thead>
<tr>
<th>Item</th>
<th>1 Factor</th>
<th>2 Factors</th>
<th>2 Factor Loadings</th>
<th>Item 3 removed</th>
<th>Items 3 and 4 removed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Single Factor</td>
<td></td>
<td>Factor 1</td>
<td>Factor 2</td>
<td>Factor 1</td>
</tr>
<tr>
<td>1. Losing track of what was going on</td>
<td>.76</td>
<td>.75</td>
<td>.06</td>
<td>.76</td>
<td>.05</td>
</tr>
<tr>
<td>2. Found I was on ‘automatic pilot’</td>
<td>.62</td>
<td>.57</td>
<td>.09</td>
<td>.57</td>
<td>.05</td>
</tr>
<tr>
<td>3. Sense of time changed…slow motion</td>
<td>.54</td>
<td>.31</td>
<td>.38</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4. Seem unreal…dream or movie</td>
<td>.62</td>
<td>.28</td>
<td>.48</td>
<td>.32</td>
<td>.41</td>
</tr>
<tr>
<td>5. Spectator watching what was happening</td>
<td>.53</td>
<td>-.14</td>
<td>.82</td>
<td>-.08</td>
<td>.75</td>
</tr>
<tr>
<td>6. Body seemed distorted or changed</td>
<td>.69</td>
<td>.05</td>
<td>.69</td>
<td>.05</td>
<td>.72</td>
</tr>
<tr>
<td>7. Felt things happening to others were happening to me</td>
<td>.76</td>
<td>.01</td>
<td>.62</td>
<td>-.02</td>
<td>.67</td>
</tr>
<tr>
<td>8. Surprised to find out afterwards lots of things</td>
<td>.73</td>
<td>.69</td>
<td>.03</td>
<td>.70</td>
<td>.03</td>
</tr>
<tr>
<td>9. I felt confused…difficulty making sense</td>
<td>.63</td>
<td>.83</td>
<td>-.02</td>
<td>.82</td>
<td>-.07</td>
</tr>
<tr>
<td>10. I felt disoriented…uncertain where I was</td>
<td>.67</td>
<td>.88</td>
<td>-.11</td>
<td>.87</td>
<td>-.01</td>
</tr>
<tr>
<td>Eigenvalue</td>
<td>4.88</td>
<td>4.88</td>
<td>1.22</td>
<td>4.48</td>
<td>1.22</td>
</tr>
<tr>
<td>Cumulative % of Variance</td>
<td>48.82</td>
<td>48.82</td>
<td>61.11</td>
<td>49.81</td>
<td>63.42</td>
</tr>
</tbody>
</table>

*Note. N = 128. PDEQ = Peritraumatic Dissociative Experiences Questionnaire. Factor loadings greater than 5 are bolded.*
Examination of item loadings on factor 1 showed that items 1, 2, 8, 9, and 10 displayed moderate to high loadings (from .57 to .88) and items 5, 6, and 7 showed moderate to high loadings (from .62 to .82) on factor 2. Item 4 was found to load on factor 2 (.48) but also displayed a weak cross loading on factor 1 (.28). Item 3 cross loaded on both factors (above .3) and was eliminated from further analysis as it failed to contribute to a simple factor structure.

Examination of the 2 factor solution with item 3 removed (see Table 6.8) revealed the same item clusters on each of the 2 factors with the exception of item 4 which cross loaded (loadings above .3) on both factors and thus was removed. The remaining 8 items (items 3 and 4 removed) were re-analysed showing 2 clear factors with items 1, 2, 8, 9, and 10 loading on factor 1 (Cronbach’s alpha .86, eigenvalue of 4.04, explaining 50.6% of the variance) and items 5, 6, and 7 loading on factor 2 (Cronbach’s alpha .74, eigenvalue of 1.20, explaining 65.7% of the cumulative variance).

### 6.4.2 Confirmatory Factor Analysis

Confirmatory factor analysis (CFA) was conducted using Amos 7. Fit indexes (previously described in section 5.4) were used to determine model fit and a chi-square difference test was used to compare nested models. Log transformation scores for item 7 were used.

Results of the 1 factor structure model indicated that the data provided an excellent fit, $\chi^2 = 15.06, \ p = .859 \ (df = 22; \ N = 333), \ \chi^2/df = 0.685$, CFI = 1.00; RMSEA = .000, AIC = 101.069, supporting the EFA 1 factor structure. An 8 item 2 factor model with items 3 and 4 removed also provided an excellent fit, $\chi^2 = 17.32, \ p = .240 \ (df = 14; \ N = 333), \ \chi^2/df =$
However, nested model comparison with items 3 and 4 constrained within a 10 item 2 factor model in which items 3 and 4 loaded on factor 2 revealed a significant difference between the models (see Table 6.9). These results show that an 8 item 2 factor model does fit that data, however, inclusion of items 3 and 4 significantly improved model fit. The results of the 10 item two factor solution are quite similar to the findings by Bryant et al. (2009). They reported 2 factors comprising ‘altered awareness’ (items 1, 8, 9, and 10) and ‘derealisation’ (items 2, 3, 4, and 6). Given similarity of the item clusters the factor labels proposed by Bryant et al. were retained.

Binary logistic and multiple regression analysis were used to determine the contribution of each of the 2 factors on psychopathology. Both factors were entered simultaneously to predict PTSD as well as PTSD symptom severity. Binary logistic regression results showed that the omnibus test of the model coefficients was significant, $\chi^2 (2, N = 120) = 16.80, p < .001$, and the Hosmer-Lemeshow test was not significant, $\chi^2 (8, N = 120) = 9.17, p = .328$, indicating that model adequately fits the data. Factor 1 was a significant predictor of PTSD (Wald = 12.31, $p = <.001$, OR = 2.86), and factor 2 was not (Wald = 1.21, $p = .270$, OR = 1.27). Similarly, factor 1 was a significant predictor of PTSD symptom severity ($\beta = .537, p = .001$), with factor 2 failing to reach significance ($\beta = .027, p = .079$). The regression model was significant, $F (2, 119) = 9.26, p < .001$, and explained 27.1% of PTSD symptom severity variance.
Table 6.9

Two Factor 10 item and 8 item Nested Model Comparisons and Fit Indices

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$p$</th>
<th>$\chi^2$/df</th>
<th>CFI</th>
<th>RMSEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconstrained</td>
<td>23.41</td>
<td>24</td>
<td>0.495</td>
<td>0.976</td>
<td>1.00</td>
<td>0.000</td>
</tr>
<tr>
<td>Constrained</td>
<td>324.03</td>
<td>26</td>
<td>0.001</td>
<td>12.46</td>
<td>0.790</td>
<td>0.190</td>
</tr>
<tr>
<td>$\Delta \chi^2$</td>
<td>300.61</td>
<td>2</td>
<td>0.001</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. N = 128.

6.5 Predictors of Post-MVA Psychopathology

Analysis of factors that influenced post-MVA psychopathology comprised an initial examination of relationships between pre, peri, and post-accident variables associated with PTSD and depression. As shown in the correlation matrix (see Table 6.6) multiple significant associations were found. However, given the sample size and the number of potential predictor variables as well as interactions, a path analytic approach would first require a reduction in the number of variables to be included in the model(s). In order to accomplish this without discarding important variables that may be strongly aligned with outcome variables, multiple linear regressions were conducted with pre, peri, and post-trauma variables on both PTSD and depression symptom severity to identify variables for inclusion in path analyses.

The first step of the regression analysed the role of pre-MVA variables that had previously been shown to predict PTSD and which were significantly correlated with PTSD (age, gender, emotion-focused coping, neuroticism and trait dissociation), followed by peri-traumatic variables (accident severity, fear of dying and peritraumatic dissociation) and
post-MVA variables (feeling sad or depressed anxious or fearful, physical disability and total AUDIT scores). Based on the regression results, emotion-focused coping ($\beta = .19, p = .050$), accident severity ($\beta = .15, p = .033$), peritraumatic dissociation ($\beta = .29, p = .001$), feeling sad or depressed ($\beta = .40, p = .001$), and physical disability ($\beta = .42, p = .001$), were included in the path model. Additionally, to assess the proposed meditational relationship between injury severity, physical disability and PTSD, injury severity was also included in the initial model.

A similar procedure was also conducted in order to develop a path model examining variable relationships associated with depression symptom severity. Pre (emotion-focused coping, neuroticism, trait dissociation, past emotional problems), peri (accident severity and peritraumatic dissociation) and post-MVA variables (feeling sad or depressed, anxious or fearful, physical disability and total AUDIT score) that were significantly associated with depression were regressed on depression symptom severity. Results showed that trait dissociation, feeling sad or depressed, total AUDIT score, and physical disability significantly predicted depression symptom severity.

6.5.1 Path Analysis: PTSD Symptom Severity

The procedural development of the path model and fit indexes used are previously outlined in section 5.4. Skew and kurtosis levels were found to be within acceptable limits (< 2.0 & 4.0 respectively). However, the initial path model with all variables loading directly onto PTSD symptom severity (except injury severity which was loaded on to physical disability), did not meet the goodness-of-fit criteria, $\chi^2 = 92.87, p = .001$ ($df = 14; N =$
124), $\chi^2/df = 6.63$, CFI = .660; RMSEA = .214, AIC = 134.87. 69. ($\beta = .175$, $p = .022$). The path coefficients from emotion-focused coping ($\beta = .09$, $p = .305$), and accident severity ($\beta = .14$, $p = .089$) to PTSD symptom severity were not significant and were removed. Based on the recommendations of Modification Indices, the path coefficients from feeling sad or depressed to physical disability, and from emotion-focused coping to both peritraumatic dissociation and feeling sad or depressed were added. Accident and injury severity were allowed to covary. The covariance between accident severity and PTSD symptom severity was added ($cov = .22$) though for ease of viewing was not included in the model figure.

The respecified path model provided a good fit of the data, $\chi^2 = 15.99$, $p = .142$ ($df = 11$; $N = 124$), $\chi^2/df = 1.45$, CFI = .978; RMSEA = .061, AIC = 63.99, with the predictor variables explaining 58.5% of PTSD symptom severity variance. As illustrated in Figure 6.5 feeling sad or depressed which was directly influenced by emotion-focused coping and peritraumatic dissociation was the strongest predictor of PTSD symptom severity followed by physical disability and peritraumatic dissociation. Emotion-focused coping had an indirect effect on symptom severity mediated by peritraumatic dissociation as well as sad or depressed. Feeling sad or depressed also mediated the relationship between peritraumatic dissociation and PTSD symptom severity. Physical disability also mediated the relationships between sad or depressed and symptom severity and also between injury severity and PTSD symptom severity.
Figure 6.5. Path analysis model with standardized path coefficients for pre, accident, and post-accident factors influencing PTSD symptom severity. All paths are significant ($p < .05$). Curved dashed line represents correlation.
6.5.2 Path Analysis: Depression Symptom Severity

A depression symptom severity path model was developed using the same procedure as outlined previously (refer to section 5.4). Skew and kurtosis levels were within acceptable limits (< 2.0 & 4.0 respectively). All predictor variable path coefficients were initially directed to depression symptom severity. The path from injury severity to physical disability was retained from the previous model. Results showed that all path coefficients were significant. However, the initial path model did not meet the goodness-of-fit criteria, $\chi^2 = 80.66$, $p = .001$ ($df = 10; N = 124$), $\chi^2/df = 8.06$, CFI = .680, RMSEA = .240, AIC = 114.66.

Using modification indices to guide re-specification the covariance between trait dissociation and sad or depressed was added. Trait dissociation and total AUDIT score were also allowed to covary ($cov = .28$) but not included in the path diagram for ease of viewing. Additionally, path coefficients from sad or depressed to total AUDIT score and physical disability were also added.

The respecified model provided a good fit of the data, $\chi^2 = 11.32$, $p = .079$ ($df = 6; N = 124$), $\chi^2/df = 1.88$, CFI = .976, RMSEA = .085, AIC = 53.32, with the predictor variables explaining 63.4% of depression symptom severity variance. As illustrated in Figure 6.6 trait dissociation had the strongest direct effect on depression followed by feeling sad or depressed, physical disability, and alcohol use. Feeling sad or depressed had the strongest total effect (.52, $p = .011$) as its influence on depression symptom severity was also partially mediated by alcohol use, and physical disability. Physical disability also fully mediated the relation between injury severity and depression symptom severity.
Figure 6.6. Path analysis model with standardized path coefficients for pre, accident, and post-accident factors influencing depression symptom severity. All paths are significant ($p < .05$). Curved dashed line represents correlation.
6.5.3 Building on the Phase 1 Path Model

Expanding on the Phase 1 path model included the addition of the outcome measures PTSD and depression symptom severity. Furthermore, given the direct and meditational influence of physical disability on both PTSD and depression, disability was also included in the initial model. Level of alertness and past emotional problems were not significantly correlated with peritraumatic dissociation and was not included in the model. Adopting the same path analytic procedures as previously used (see 5.4), coefficient paths from anxious or fearful and from sad or depressed to PTSD and depression symptom severity were added. The path from injury severity to physical disability was also added.

Results showed that skew and kurtosis levels were within acceptable limits (< 2.0 & 4.0 respectively). The initial model did not meet the goodness-of-fit criteria, \( \chi^2 = 36.14, p = .053 \) (\( df = 24; N = 128 \)), \( \chi^2/df = 1.50 \), CFI = .977, RMSEA = .063, AIC = 118.14. Examination of the standardised path coefficients showed that five paths were not significant. Three were weak path coefficients from the Phase 1 model: injury severity to fear of dying (\( \beta = .10, p = .388 \)); fear of dying to anxious or fearful (\( \beta = .17, p = .059 \)), and fear of dying to sad or depressed (\( \beta = .13, p = .100 \)). The path coefficients from anxious or fearful to PTSD (\( \beta = .18, p = .175 \)) and to depression symptom severity (\( \beta = .02, p = .864 \)) were also not significant. These paths were excluded to provide a more parsimonious model.

Based on the recommendations of Modification Indices the covariance between anxious or fearful and PTSD symptom severity and between peritraumatic dissociation and depression
symptom severity \((cov = .21)\), were added, however to improve the readability the covariance between peritraumatic dissociation and depression symptom severity was not included in the model diagram. Model respecification (illustrated in Figure 6.7) indicated a good fit of the data, \(\chi^2 = 35.26, p = .132 \ (df = 27; N = 128), \chi^2/df = 1.30, \text{CFI} = .984, \text{RMSEA} = .049, \text{AIC} = 117.26\), with the model accounting for 58.8% and 45.4% of PTSD and depression symptom severity variance respectively.

Results showed that the influence of past emotional problems was reduced over time but still had a direct effect on initial distress (feeling anxious or fearful and/or sad or depressed). The mediating role of fear of dying on dissociation remained and influenced initial distress and ongoing PTSD but not depression symptoms. Initial post-MVA sadness but not fear influenced ongoing PTSD and depression symptom severity. Sadness and injury severity both influenced physical disability which had a direct effect on PTSD and depression symptoms.
Figure 6.7. Path analysis model with standardized path coefficients for Phase 1 variables and Phase 2 variables (PTSD and depression symptom severity as well as physical disability). All paths are significant ($p < .05$). Dashed lines represent correlations.
6.6 Discussion

The Phase 2 component of the study showed that pre-morbid characteristics were vulnerability factors for experiencing peritraumatic dissociation. A neurotic predisposition in the presence of emotion-focused coping contributed to acute dissociation. This finding partially supports the first hypothesis. However, there was no evidence that personality, after controlling for other pre and peritraumatic variables, was associated with PTSD which does not support the second component of the first hypothesis. These results are consistent with previous studies which have found that neuroticism was predictive of peritraumatic dissociation (Groth-Marnat & Jeffs, 2002; Jaycox et al., 2003), but not with others who found that neuroticism was predictive of PTSD (Dorfel et al., 2008; Holeva & Tarrier, 2001). However, these findings are consistent with the results of other MVA studies (Conlon et al., 1999; Mayou et al., 1993) reporting that neuroticism did not predict PTSD and support the suggestion (Bramsen, Dirkzwager, & van der Ploeg, 2000; Schnurr, Friedman, & Rosenberg, 1993) that although personality is influential, exposure characteristics play a more prominent role in the development of PTSD. It is difficult to provide a definitive explanation as to why the influence of neuroticism has been found to be significant in some studies, but not in others. Differences in assessment (prospective vs. retrospective) and statistical analysis (accounting for pre and peritraumatic variables which interact with neuroticism) may account for such discrepancies.

Other researchers (Goldberg, 1999; Spiegel & Greenleaf, 1992) have suggested that the effect of dissociation is influenced by personality. However, this perspective is in direct contrast with the current findings which show two distinct pathways influencing
peritraumatic dissociation. That is, premorbid psychological state (neuroticism interacted with other pre-trauma factors) and exposure characteristics (accident severity and fear of dying) which were not associated with pre-trauma factors.

There was no evidence that trait dissociation had a direct effect on peritraumatic dissociation, therefore the second hypothesis that pre-accident dissociative tendencies would influence acute dissociation was not supported. These results are inconsistent with previous studies finding trait dissociation independently predicted peritraumatic dissociation (McCaslin et al., 2008; Engelhard et al., 2003). However, similar to other studies, trait dissociation did show a weak positive association with peritraumatic dissociation (cf. McCaslin et al., 2008; Tichenor et al., 1996). However, trait dissociation was more strongly aligned with other pre-trauma factors particularly neuroticism and emotion-focused coping. These findings suggest that independent of exposure severity, pre-trauma psychological state (general tendency to dissociate and a neurotic predisposition) influences the use of maladaptive coping strategies, (reducing the stress threshold) and increases the likelihood of a dissociative response upon trauma exposure.

Results of this study showed that participants who generally utilise emotion-focused coping strategies were more likely to dissociate upon trauma exposure which provides partial support for the first component of the third hypothesis, that is, that pre-trauma coping would influence immediate trauma response. Results also provided partial support for the second part of the hypothesis in that emotion-focused coping was indirectly associated with PTSD. These results are not directly comparable to previous MVA studies.
as retrospective assessment of pre-morbid coping has not been previously examined. However, they are consistent with previous studies that have found emotion-focused coping influenced acute dissociation (Collins & French, 1998; Collins & Jones, 2003). They are also in accord with past results showing associations between trait emotion-focused coping and PTSD (Dorfel et al., 2008; Gil, 2005), and between neuroticism and emotion-focused coping (Morgan et al., 1995).

The current study highlights the tripartite relationship between premorbid psychological state, coping and peritraumatic dissociation and is in accord with previous studies linking emotion-focused coping with dissociative tendencies. Coping is influenced by a number of factors such as personality and the nature of the stressor, including the perception of controllability, that is, when a stressful event is perceived as uncontrollable, emotion-focused strategies are more likely to be used. It is reasonable to suggest that premorbid psychological state, particularly given the negative valance associated with neuroticism, affects the perception of personal control when experiencing an MVA. Though not assessed, it is proposed that individuals with a neurotic predisposition may be more inclined to perceive their MVA as something that was out of their control and as such engage in emotion-focused coping strategies.

The finding that participants’ general use of avoidance coping strategies was not related to peritraumatic dissociation may be due to the predominate endorsement of behavioural items. In the immediacy of MVA trauma, the possibility of using behavioural strategies may be limited and therefore not readily utilised compared to the use of emotion-focused
strategies comprising cognitive components (e.g., anxious, tense, freeze, worry, anger) which have been associated with both immediate trauma reactions and ongoing psychopathology. Overall, these findings support the perspective of contemporary cognitive PTSD theories which acknowledge the influence of pre-trauma factors on initial trauma responses and appraisals on post-trauma psychopathology. An interesting component of this study was the finding that two independent pathways predicted peritraumatic dissociation; an exposure/fear pathway and an interrelated premorbid psychological state pathway. These results suggest that some participants dissociate because of high levels of fear and others because of previous vulnerability factors. In both cases it appears that the cognitive and emotional demands of the situation overwhelm coping resources and, as a result, reduce the ability to fully process trauma information.

There was no evidence that increased alcohol use or hazardous consumption was associated with PTSD. Results did indicate that 33% of participants with PTSD reported comorbid alcohol problems, however, 63% were drinking excessive amounts before their MVA. Almost half of the participants with depression (46%) also reported comorbid alcohol problems but 66% were consuming similar amounts of alcohol pre-MVA. These findings do not support the hypothesis proposing that increased use of alcohol, particularly at a hazardous level, would be associated with PTSD and depression post-MVA, and as such are inconsistent with some MVA studies (Blanchard, Hickling, Barton et al., 1996; Bryant & Harvey, 1995a; McFarlane et al., 2009) which found significant associations between alcohol use/abuse and PTSD, but they are consistent with other studies (Blanchard et al., 2004; Blanchard et al., 1994; Mayou et al., 1997) that did not find this association.
The current findings highlight the importance of considering pre-trauma alcohol consumption when assessing the influence of alcohol on post-trauma psychopathology. Controlling for pre-trauma alcohol use prevents an over-inflation of post-trauma (alcohol) assessment results, and may partly explain why some MVA studies found a significant association between alcohol use and PTSD and why others have not.

There was evidence that participants who drank hazardous levels of alcohol had experienced severe past emotional problems, utilised maladaptive coping strategies, had a neurotic predisposition and a general tendency to dissociate in addition to experiencing greater post-MVA (current) depressive symptoms. These results suggest that experiencing an MVA did not increase the likelihood of alcohol usage as participants were consuming hazardous amounts of alcohol before their MVA. Though drinking alcohol was associated with psychological effects both pre and post-MVA, causality cannot be determined. As such, it is unknown if premorbid psychological state influenced alcohol consumption or if pre-MVA drinking influenced pre and/or post-MVA psychological state (past emotional problems and/or depression). Furthermore, it is not known if alcohol was a contributing factor in the MVA as participant’s blood alcohol content was not assessed. Although recent evidence suggests that alcohol use in moderation may be an effective form of coping post-accident (McFarlane et al., 2009), initial screening and identifying hazardous levels of alcohol use pre and post-MVA may help circumvent ongoing dependence particularly in those who are more vulnerable.
Injury severity was not directly associated with PTSD, but it did have a direct effect on physical disability which directly influenced PTSD. This result supports the hypothesis that the relationship between injury severity and PTSD would be mediated by disability.

Subjective reporting of disability may not necessarily be an accurate determinate of actual physical impairment. That does not mean to say that participants who have sustained injury are not physically disabled. Rather, it is not so much the degree of physical impairment but more so the personal meaning, significance, and appraisal of disability that determines post-trauma adjustment in the longer term. Acceptance and learning to adapt to current and potential future physical restriction for many MVA survivors presents a significant challenge, which for some will exceed coping resources. Perceived permanent change, scarring and/or disfigurement, pain and ongoing reduced mobility can act as constant reminders of the accident which may impede recovery and prevent the accident being seen as something that happened ‘in the past’.

The finding that disability was predictive of PTSD is consistent with previous MVA studies (Hamanaka et al., 2006; Jenewein et al., 2009; Mayou & Bryant, 2001; Mayou et al., 1997), and supports the suggestion by Ehlers and Clark (2000) that negative appraisals of trauma sequelae including physical disability serve to maintain symptoms since emotions associated with negative appraisals promote dysfunctional coping strategies which in turn prevent cognitive change.

An exploratory component of this study was to examine the factor structure of the Peritraumatic Dissociative Experiences Questionnaire (PDEQ). Results indicated that the
PDEQ formed a single factor structure and also a correlated (10 item) 2 factor structure which comprised altered awareness (items 1, 2, 8, 9, 10) and derealisation (items 3, 4, 5, 6, 7), with both factor solutions having acceptable psychometric properties. Of these 2 factors, only altered awareness was predictive of PTSD and symptom severity. These findings, though in direct contrast to the results of Bryant et al. (2009), did display quite similar factor items. For example, Bryant et al., after they disconfirmed a single factor, reported a 2 factor structure solution comprising altered awareness (items 1, 8, 9, 10) and derealisation (items 3, 4, 5, 6). They also reported that derealisation, but not altered awareness, was significantly associated with posttraumatic stress.

Whilst acknowledging there are some perceptual similarities between derealisation and altered awareness, only the later construct was found to be predictive of PTSD. The items which comprised altered awareness characterise aspects of detachment, confusion, and disorientation which mask the reality of a situation by limiting awareness. Reduced awareness affects difficulties in encoding trauma information, which results in disjointed and fragmented trauma memories (Horowitz, 1986; van der Kolk, 1987; van der Kolk & van der Hart, 1989).

The inconsistencies between the current results and those of Bryant et al. (2009) may be partly explained by examining sample characteristics. Bryant`s initial sample comprised 78% male participants, who were considerably less distressed than that of their second sample which they used for cross-validation and which comprised a more balanced gender ratio with 53% male participants. Their initial 8 item 2 factor solution was not supported
with the more traumatised second sample; further analysis (again with items 2 and 7 removed) revealed a number of cross-loadings (not dissimilar to the current study with cross-loadings on items 3 and 4). Furthermore, the use of the Hospital Anxiety and Depression Scale (HADS: Zigmond & Snaith, 1983) limits the conclusion of results regarding relationships between PDEQ factors and PTSD. However, more recently, in what appears to be a 3-month follow-up study which included 208 participants from the original sample \((n = 247)\), it was reported that 9.6% of participants had chronic PTSD. Bryant et al. (2011) used path analysis and found that derealisation was predictive of PTSD. The authors suggested that altered awareness, which was not predictive of PTSD, is a common dissociative response that does not necessarily lead to psychopathology. However, it is interesting to note that time distortion and derealisation have also been identified as common non-pathological dissociative responses in non-trauma samples (Collins, 2004; Sterlini & Bryant, 2002).

It has been reported that the degree of dissociation is associated with the severity of the trauma experience (Zatzick, Marmar, Weiss, & Metzler, 1994). The initial sample used by Bryant et al. (2009) to examine the factor structure of the PDEQ was markedly less traumatised than the sample used for cross-validation as well as the sample used in the current study. Variations in the severity of traumatisation are likely to be reflected in the analysis of dissociative responses and may account for the differences of results.

The results of this study suggest that the PDEQ is a unidimensional construct. However, evidence of a correlated 2 factor structure was also apparent. The removal of cross loading
items (3 and 4) using EFA were not supported using CFA and, given the strong relationship between the 2 factors, it is not surprising to find some items cross-loaded. Other researchers have commented that a lack of model fit using CFA based on factor solutions supported by EFA may be due to the more stringent restrictions used in CFA (van Prooijen & van der Kloot, 2001). To date this is the only other study to examine specific PDEQ dissociative factors and their association with PTSD. Though the current results did not replicate previous findings they do provide additional information into acute dissociative phenomena and the development of PTSD. It is acknowledged that these results are limited to an MVA population and require further validation across a range of trauma groups to ascertain which specific dissociative responses are associated with post-trauma psychopathology.

One of the aims of this study was to describe the incidence of psychopathology and comorbidity. The finding that 37% of MVA survivors had PTSD at 3-months post-MVA shows a considerable degree of psychopathology within the sample. The current PTSD rate is somewhat higher than the 24% average reported by Blanchard and Veazey (2001), however it is consistent with a number of previous MVA studies (Blanchard et al., 1996; Blanchard et al., 1997; Coronas et al., 2008), and is within the range of MVA 3-month incident rates (8 to 50%) reviewed in section 3.2.

Comorbid depression was evident since 43% of participants with PTSD also reported concurrent depressive symptoms, which is consistent with the comorbid rate in previous MVA studies (Blanchard, Buckley et al., 1998; Blanchard et al., 1994, 1995, 2004;
Fromberger et al., 1998; Kuch et al., 1995). This study showed that although PTSD and depression were highly correlated they are in part independent trauma sequelae. That is, they each had separate predictor variables as well as sharing predictor variables. Individuals with a general tendency to dissociate, those consuming alcohol, experiencing initial feelings of sadness and sustaining physical disability were more likely to experience greater depressive symptoms. Similarly, initial feelings of sadness and physical disability were also predictive of chronic PTSD, but having experienced peritraumatic dissociation was predictive of PTSD but not depression. These findings are in accord with previous MVA studies which have found a reciprocal relationship between PTSD and depression, resulting in greater symptomatology (Buckley et al., 1998; Shalev et al., 1998; Wolf et al., 2001). Similarly, they are also consistent with the results published by O’Donnell et al. (2004) who found that the same variables predicted both PTSD and depression, indicating a shared vulnerability. It could be assumed that depression developed post-accident. However, as prior depression was not assessed causality cannot be determined, that is, whether depression was a vulnerability factor for PTSD, or a consequence, or whether current depressive symptoms are an exacerbation of previous symptomology.

The finding that participants who had a general tendency to dissociate were more likely to experience initial sadness as well as depressive symptoms at 3-months post-MVA was somewhat surprising and may reflect an overlap between constructs, for example, emotional numbing. These findings suggest that pre-trauma psychological state comprising interrelated factors (trait dissociation and poor coping, neuroticism, as well as past
emotional problems) are vulnerabilities for immediate trauma response, initial emotional distress and ongoing depression.

A further aim of the study was to incorporate standardised outcome measures (PTSD and depression) and further develop the Phase 1 path analysis model. Results showed that the influence of past emotional problems was reduced over time, though they had a direct effect on initial distress (feeling anxious or fearful and/or sad or depressed) at 1-month. However, at 3-months, past emotional problems had no direct influence on the severity of PTSD or depression symptoms, though it did have an indirect influence on depression. The assessment of previous experience of psychological problems is important to identify individuals who are likely to experience initial post-accident distress which may lead to ongoing psychological morbidity.

Peritraumatic variables (fear and dissociation) had direct and indirect effects at both time points. Individuals who feared they were going to die and experienced dissociative symptoms during the MVA experienced initial emotional distress. However, at 3-months these variables were only predictive of PTSD symptom severity. This finding is consistent with past research (Blanchard, Hickling, Mitnick et al., 1995; Ehlers et al., 1998; Jeavons et al., 2000; Murray et al., 2002) which has shown these psychological reactions are vulnerability factors for PTSD.

The mediating role of fear of dying on dissociation remained and influenced initial distress and ongoing PTSD but not depression symptoms. Initial post-MVA sadness but not fear
influenced ongoing PTSD and depression symptom severity. Sadness and injury severity independently influenced physical disability, which had a direct effect on PTSD and depression symptoms. These findings are consistent with previous studies which found that physical disability was associated with PTSD and the maintenance of symptoms (Hamanaka et al., 2006; Mayou & Bryant, 2001; Mayou et al., 1997), as well as depression (Jenewein et al., 2009; Mayou & Bryant, 2002; O’Donnell et al., 2004). This study provides evidence that disability is not only influenced by physical injury as would be expected, but also by psychological state, which indicates that (initial) post-MVA sadness affects the perception of disability. These results are in accord with previous studies that have shown that a negative appraisal of injury as well as negative appraisal of the future impact of injury (physical impairment) are predictive of PTSD (Ehlers et al., 1998; O’Donnell et al., 2004), and that distressed trauma survivors are more likely to appraise their injury from a negative perspective, thus exaggerating the impact of injury and subsequent disability (Ehlers et al., 1998; Mayou & Bryant, 2001).

Higher levels of exposure (accident severity) contributed to initial emotional distress and ongoing morbidity predominantly in the presence of high levels of fear and subsequent dissociative experiences. These findings demonstrate the complexity of the meditational role of dissociation. These findings are consistent with previous research showing that exposure (Bryant & Panasetis, 2005; Marmar et al., 1996) and fear of dying (Bryant & Panasetis, 2005; Fikretoglu et al., 2007; Nixon et al., 2002) are associated with dissociation.
One of the interesting findings from the ongoing morbidity assessment was the predominance of the effect (direct and indirect via physical disability) of initial sadness on both depression and PTSD symptom severity. There was an ongoing indirect effect of initial fear on subsequent PTSD symptoms with dissociation contributing to maintenance of PTSD symptoms. This suggests that reported fear responses one month post-MVA is to be expected and does not indicate a direct vulnerability to ongoing morbidity. A better indicator may be reported sadness in the initial aftermath of an MVA and ongoing disability.

Although malingering was not assessed (nor is it being inferred), it may be worthwhile to incorporate a measure in future studies, particularly given the prominent role of physical disability on psychopathology, to rule out over-reporting associated with secondary gain. The compensation process has been shown to hinder recovery and increase anxiety which exacerbates symptoms. It may be worthwhile for future studies to consider the influence of making a compensation claim post-MVA to rule out increased (secondary) symptomatology associated with seeking compensation. The absence of such an assessment presents as a potential limitation of this study.

There was no effect of age or gender on post-trauma psychopathology with the exception of alcohol in which younger age was associated with hazardous consumption. However, given that hazardous consumption levels were evident pre-MVA, this result is more indicative of premorbidity than a post-trauma outcome and highlights the importance of assessing pre-trauma alcohol consumption. From a clinical perspective it is concerning that
a considerable number of younger participants were consuming alcohol at dangerous levels before their MVA. Given that chronic alcohol abuse and hazardous consumption can contribute to behavioural problems, addiction, and subsequent poor adjustment, it is important from a treatment perspective to assess and monitor the consumption of alcohol post-trauma. It is not completely surprising that age and gender did not influence post-trauma outcomes as previous studies have produced mixed results. It may be that in the presence of other (more influential) variables, the effects of age and gender on posttraumatic stress are reduced. Results from Phase 1 indicated that females experienced greater acute dissociative symptoms. However, factors that influenced dissociation (accident severity and fear of dying) were not influenced by gender. Similarly, in the current study phase, neither premorbid psychological state nor exposure characteristics (which independently predicted peritraumatic dissociation) were influenced by gender. These findings clearly suggest that other factors which were not assessed in the current study are associated with females increased risk of peritraumatic dissociation. Further research considering other factors (e.g., neurobiological responses) is required and may help explain why female gender poses an increased risk for acute dissociative responses as a result of trauma exposure.

6.7 Limitations and Summary

Structural equation modelling is a powerful statistical method that allows a clearer differentiation of the unique contributions of each variable and, although the models provided an excellent fit to the data, it is possible that different models may also fit the data equally well. The models were derived from theory and previous findings and not
developed to examine potential model variations. One of the limitations of this study was the high attrition rate which resulted in a greater proportion of females and older participants in the later phases of the study. Participants in this phase of the study also reported more initial anxiety compared to participants in the Phase 1 sample. However, there was no evidence of injury severity bias based on ISS comparisons. The partial retrospective design limits conclusions regarding causal relationships. The use of unstandardised self-report pre and initial post-accident distress measures is a limitation, however, the use of standardised pre-morbid (personality, coping, and trait dissociation) and follow up (PTSD and depression) measures provide more support for the conclusions. Furthermore, the correlations between the single item questions regarding fear and sadness in Phase 1 and the PDS and BDI-II in the later phase was .61 and .64 respectively (see table 6.6), which indicates that the simple rating scales share a moderate degree of variance with equivalent standardised measures and validates their use as screening tools.

Building on the Phase 1 path analysis model by including standardised outcome measures including physical disability has provided a more thorough picture of the complex interactions between pre, peri, and post-MVA reactions at 3-months. The finding that sadness (partly influenced by prior emotional problems) in the first month following MVA and physical disability at 3-months (influenced by injury severity and initial sadness) influences ongoing depression as well as PTSD symptom severity suggests a partial shared vulnerability. Assessment of pre-accident morbidity, initial feelings of sadness and subjective reporting of more severe physical disability post-MVA may help identify MVA
survivors who could benefit from psychological intervention to reduce ongoing psychological morbidity.

This study has identified a number of important considerations in identifying MVA survivors who are at greater risk for post-trauma psychopathology and has also demonstrated the complexity of relationships. Participant’s pre-morbid psychological state was a vulnerability factor for peritraumatic dissociation, initial post-trauma distress, and PTSD symptom severity. Severity of the MVA directly influenced an immediate fear response which also increased participants’ likelihood of experiencing peritraumatic dissociation. These results are in accord with Ehlers and Clark’s (2000) cognitive model of PTSD which suggests that background factors as well as trauma characteristics and state factors are likely to influence initial emotional reactions, processing (reduced ability to process the trauma conceptually), and trauma appraisals. These results also provide support for the perspective that intense emotional reactions elicit peritraumatic dissociation which in turn influences posttraumatic symptoms through incomplete processing.

Furthermore, the results of this study suggest that MVA survivors with initial post-MVA feelings of sadness were more likely to report physical disability, depression and PTSD symptoms at 3-months post-accident. These findings provide support for the proposal by Ehlers and Clark (2000) that negative appraisal of trauma sequelae, including physical consequences and emotion responses, contribute to persistent PTSD, and that appraisals associated with perceived loss lead to sadness, and appraisals of perceived certainty of loss are associated with depression. Given the prominent role of emotional response it may be
beneficial for future MVA studies to assess specific appraisals associated with initial post-accident sadness and their associations with physical disability as well as chronic PTSD and depression.

The first 2 studies of this research (Phases 1 and 2) examined factors associated with the development and chronicity of PTSD. The following chapters comprise Phase 3 and 4 which will examine factors associated with the maintenance of the disorder including the influence of personality, coping, and ongoing dissociation. The final study (Phase 4) includes results of a structured clinical interview to confirm diagnoses and the assessment of cognitive appraisals associated with PTSD.
Chapter 7

7.1 Phase 3 Study

7.1.1 Aims of the Study

The overall aim of this phase of the study was to describe the incidence of psychopathology as well as examine comorbid relationships (PTSD, depression and alcohol abuse) at 6-months post-MVA. A further aim was to examine the associations between variables assessed at 1 and 3-months in relation to ongoing psychopathology. This study will also examine the associations between pre and post-MVA measures of personality, coping, and dissociation and their influence on PTSD. An additional aim of this study is to determine factors that contribute to the maintenance of PTSD.

7.1.2 Hypotheses

On the basis of previous results, and also taking into account theoretical accounts regarding the maintenance of PTSD, it is hypothesized that subjective report of injury severity, initial psychological distress (feeling sad or depressed), as well as physical disability at 3-months will predict on-going disability 6-months post-MVA. It is further hypothesized (2) that initial psychological distress and physical disability will be associated with the maintenance depression and PTSD symptom severity at 6-months. Additionally, of the measures of dissociation, it is expected (3) that ongoing dissociation (influenced by pre and peritraumatic dissociation) will have the strongest influence on PTSD at 6-months post-accident. It is further hypothesized (4) that participants with PTSD will have higher pre and post-MVA neuroticism scores compared to participants without PTSD. Given the results of the previous study which showed that hazardous alcohol consumption was not associated
with PTSD, but was associated with depression as participants who had drunk hazardous levels reported greater depressive symptoms, it is hypothesized (5) that drinking alcohol over the longer term (6-months) at a hazardous level will be associated with the maintenance of depressive symptoms. The results from the previous study also showed that trait emotion-focused coping was a vulnerability factor for immediate trauma response (peritraumatic dissociation), initial psychological distress (feeling sad or depressed) and indirectly influenced PTSD symptom severity. Therefore, it is hypothesized (6) that trait emotion-focused coping will be associated with the maintenance of PTSD.

7.2 Results
7.2.1 Demographics
Each of the 128 MVA survivors who completed Phase 2 was posted an invitation to participate in a second follow-up survey. A total of 65 participants (50.78%) agreed to participate and were posted the second follow-up survey. From the 65 surveys which were sent a total of 63 were returned, which equates to a response rate of 96.9%. However, 5 surveys were returned blank or incomplete and therefore were not included in statistical analyses. Not all participants answered every question therefore the sample size in some analyses varies. The sample \(N = 58\) comprised 42 participants (72.4%) from JHH and 16 participants (27.6%) from The Maitland Hospital. Collectively, there were 20 males (34.5%) and 38 females (65.5%) who ranged from 18 to 81 years of age \(M = 45.19, SD = 18.12\).

In order to ascertain if the participants from Phase 3 were representative from Phase 2 direct comparisons were conducted (see Table 7.1).
Table 7.1

*Phase 2 and Phase 3 Pre, Peri, and Post-MVA 3-Month Variable Comparisons*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Phase 2 (n = 70)</th>
<th>Phase 3 (n = 58)</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>-</td>
<td>-</td>
<td>$\chi^2 = .412$ ns</td>
</tr>
<tr>
<td>Male</td>
<td>28 (40%)</td>
<td>20 (34.5%)</td>
<td>-</td>
</tr>
<tr>
<td>Female</td>
<td>42 (60%)</td>
<td>38 (65.5%)</td>
<td>-</td>
</tr>
<tr>
<td>Age Mean (SD)</td>
<td>39.54 (17.63)</td>
<td>45.19 (18.12)</td>
<td>$t = - 1.78$ ns</td>
</tr>
<tr>
<td>Education</td>
<td>-</td>
<td>-</td>
<td>$\chi^2 = 7.50$ ns</td>
</tr>
<tr>
<td>Income</td>
<td>-</td>
<td>-</td>
<td>$\chi^2 = 5.30$ ns</td>
</tr>
<tr>
<td>Previous MVA`s (Yes %)</td>
<td>38 (54.3%)</td>
<td>36 (62.1%)</td>
<td>$\chi^2 = .788$ ns</td>
</tr>
<tr>
<td>Number of Previous MVA`s Mean (SD)</td>
<td>.94 (1.08)</td>
<td>1.22 (1.42)</td>
<td>$t = -1.26$ ns</td>
</tr>
<tr>
<td>Previous MVA Rating a</td>
<td></td>
<td>$p &gt; .05$ b</td>
<td></td>
</tr>
<tr>
<td>Not as bad</td>
<td>10 (26.3%)</td>
<td>7 (19.4%)</td>
<td>-</td>
</tr>
<tr>
<td>Same</td>
<td>5 (13.2%)</td>
<td>1 (2.8%)</td>
<td>-</td>
</tr>
<tr>
<td>Worse</td>
<td>23 (60.5%)</td>
<td>28 (77.8%)</td>
<td>-</td>
</tr>
<tr>
<td>Past Emotional Problems Mean (SD)</td>
<td>1.63 (.997)</td>
<td>1.60 (.983)</td>
<td>$t = -2.70$ ns</td>
</tr>
<tr>
<td>MVA Category</td>
<td></td>
<td>$p &gt; .05$ b</td>
<td></td>
</tr>
<tr>
<td>Driver</td>
<td>42 (60%)</td>
<td>42 (72.4%)</td>
<td>-</td>
</tr>
<tr>
<td>Passenger</td>
<td>16 (22.9%)</td>
<td>7 (12.1%)</td>
<td>-</td>
</tr>
<tr>
<td>Motorcyclist/pillion</td>
<td>9 (12.9%)</td>
<td>6 (10.3%)</td>
<td>-</td>
</tr>
<tr>
<td>Pedal Cyclist</td>
<td>2 (2.9%)</td>
<td>2 (3.4%)</td>
<td>-</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>1 (1.4%)</td>
<td>1 (1.6%)</td>
<td>-</td>
</tr>
<tr>
<td>Injury (Yes %)</td>
<td>64 (91.4%)</td>
<td>57 (98.3%)</td>
<td>$p &gt; .05$ b</td>
</tr>
<tr>
<td>Injury Severity Self Report Mean (SD)</td>
<td>2.76 (.892)</td>
<td>3.07 (.722)</td>
<td>$t = 2.18^* c$</td>
</tr>
<tr>
<td>ISS Mean (SD)</td>
<td>9.89 (5.44)</td>
<td>7.73 (7.76)</td>
<td>$t = .704$ ns</td>
</tr>
<tr>
<td>Accident Severity Mean (SD)</td>
<td>2.93 (.890)</td>
<td>3.17 (.901)</td>
<td>$t = -1.53$ ns</td>
</tr>
<tr>
<td>Alertness</td>
<td>1.80 (1.30)</td>
<td>1.57 (.957)</td>
<td>$t = 1.15$ ns c</td>
</tr>
<tr>
<td>Fear of Dying</td>
<td>2.70 (1.30)</td>
<td>2.81 (1.34)</td>
<td>$t = -.471$ ns</td>
</tr>
<tr>
<td>Peritraumatic Dissociation Mean (SD)</td>
<td>21.69 (9.54)</td>
<td>21.90 (9.16)</td>
<td>$t = -.127$ ns</td>
</tr>
</tbody>
</table>

*Note: ISS = Injury Severity Score.*

MVA Severity Rating compares current MVA severity to previous MVAs 1 = not as bad, 2 = same, 3 = worse.

$^a$ Fisher’s Exact Test. $^b$ Welsch-Satterthwaite corrected.

$^*p < .05.$
Phase 2 and Phase 3 Pre, Peri, and Post-MVA 3-Month Variable Comparisons (continued)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 70)</td>
<td>(n = 58)</td>
<td></td>
</tr>
<tr>
<td>Anxious/Fearful</td>
<td>Mean (SD)</td>
<td>2.40 (1.26)</td>
<td>2.55 (1.27)</td>
</tr>
<tr>
<td>Sad/Depressed</td>
<td>Mean (SD)</td>
<td>2.36 (1.34)</td>
<td>2.48 (1.34)</td>
</tr>
<tr>
<td>Treatment (Yes %)</td>
<td></td>
<td>58 (82.9%)</td>
<td>53 (91.4%)</td>
</tr>
<tr>
<td>Treatment Type</td>
<td></td>
<td></td>
<td>( p &gt; .05 ) b</td>
</tr>
<tr>
<td>Emotional</td>
<td>1 (1.7%)</td>
<td>1 (1.9%)</td>
<td></td>
</tr>
<tr>
<td>Physical</td>
<td>48 (82.8%)</td>
<td>38 (71.7%)</td>
<td></td>
</tr>
<tr>
<td>Both</td>
<td>9 (15.5)</td>
<td>14 (26.4)</td>
<td></td>
</tr>
<tr>
<td>Treatment Duration</td>
<td></td>
<td></td>
<td>( p &gt; .05 ) b</td>
</tr>
<tr>
<td>1 day</td>
<td>13 (22.4%)</td>
<td>8 (15.1%)</td>
<td></td>
</tr>
<tr>
<td>Up to 1 week</td>
<td>11 (19%)</td>
<td>5 (9.4%)</td>
<td></td>
</tr>
<tr>
<td>Up to 1 month</td>
<td>5 (8.6%)</td>
<td>4 (7.5%)</td>
<td></td>
</tr>
<tr>
<td>One to 3 months</td>
<td>13 (22.4%)</td>
<td>14 (26.5%)</td>
<td></td>
</tr>
<tr>
<td>More than 3 months</td>
<td>16 (27.6%)</td>
<td>22 (41.5%)</td>
<td></td>
</tr>
<tr>
<td>Physical Disability</td>
<td>Mean (SD)</td>
<td>6.04 (6.72)</td>
<td>8.07 (5.83)</td>
</tr>
<tr>
<td>PTSD Symptom Severity</td>
<td>Mean (SD)</td>
<td>11.77 (10.57)</td>
<td>11.78 (10.85)</td>
</tr>
<tr>
<td>BDI-II</td>
<td>Mean (SD)</td>
<td>10.86 (10.27)</td>
<td>11.78 (10.85)</td>
</tr>
<tr>
<td>AUDIT</td>
<td>Mean (SD)</td>
<td>5.97 (6.00)</td>
<td>5.09 (6.94)</td>
</tr>
<tr>
<td>Drinking Patterns</td>
<td></td>
<td></td>
<td>( \chi^2 = 1.99 ) ns</td>
</tr>
<tr>
<td>Less</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Same</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>More</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extraversion d</td>
<td>Mean (SD)</td>
<td>4.84 (2.11)</td>
<td>4.53 (1.97)</td>
</tr>
<tr>
<td>Neuroticism d</td>
<td>Mean (SD)</td>
<td>4.84 (2.36)d</td>
<td>4.00 (2.14)</td>
</tr>
<tr>
<td>Coping d</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Task</td>
<td>Mean (SD)</td>
<td>52.91 (12.76)d</td>
<td>54.35 (11.72)</td>
</tr>
<tr>
<td>Emotion</td>
<td>Mean (SD)</td>
<td>43.19 (11.75)d</td>
<td>41.39 (13.26)</td>
</tr>
<tr>
<td>Avoidance</td>
<td>Mean (SD)</td>
<td>5.90 (9.82)d</td>
<td>41.89 (11.46)</td>
</tr>
<tr>
<td>Trait Dissociation d</td>
<td>Mean (SD)</td>
<td>35.57 (25.81)d</td>
<td>34.07 (21.76)</td>
</tr>
</tbody>
</table>

Note. BDI-II = Beck Depression Inventory-Second Edition. AUDIT = Alcohol Use Disorders Identification Test.

b Fisher’s Exact Test. d Retrospective pre-MVA assessment.

* \( p < .05 \).
As shown in Table 7.1, a small number of significant differences were found; participants in Phase 3 reported being more severely injured and scored lower on the measures of neuroticism and avoidance-coping compared to participants in Phase 2. However, unlike the previous studies (Phase 1 and 2) there were no significant age or gender differences.

7.2.2 Treatment

A total 54 (93.14%) participants reported that they had received treatment due to their MVA with 68.5% \((n = 37)\) having received treatment for physical injuries, 9.3% \((n = 5)\) having received psychological treatment, and 22.2% \((n = 12)\) of participants having received both physical and psychological treatment. Duration of treatment varied with 11.1% \((n = 6)\) of participants having received treatment for 1 day, 9.3% \((n = 5)\) for up to 1-week, 9.3% \((n = 5)\) up to 1-month, 29.6% \((n = 16)\) for between 1 and 3-months and 40.7% \((n = 22)\) of participants reported having received treatment for more than 3-months. There were no significant differences between treatment duration and treatment type nor between injury severity and treatment type. However, there was a significant main effect of injury severity on treatment duration, \(F(2, 53) = 3.78, p = .029\). Post-hoc comparisons using Tukey’s test, with a familywise Type I error rate of .05, showed that participants who reported their injuries were severe received treatment for a greater duration than those who sustained mild injury. Demographic comparisons showed that neither gender nor age had an effect on receiving treatment, treatment type, or treatment duration.

7.2.3 Physical Disability

Physical disability scores ranged from 0 to 21 \((M = 7.28, SD = 6.51)\). Using the predefined disability categories, 32.8% \((n = 19)\) of participants reported no disability, 12.1% \((n = 7)\)
mild, 20.7% \( (n = 12) \) moderate, and 34.5% \( (n = 20) \) were categorised as severe. There was a significant main effect of disability on treatment duration, \( F(4, 53) = 4.97, p = .002 \). Post-hoc comparisons (using Tukey’s test, with a familywise Type I error rate of .05), showed that participants who had received treatment for 3-months or longer had significantly greater disability scores than participants who received treatment for a period of between 1 and 3 months and those who received treatment for 1 day (see Figure 7.1).

![Figure 7.1. Treatment duration categories and disability score means.](image)

Similarly, (though not surprising) injury severity also showed a significant main effect, \( F(3, 57) = 3.87, p = .014 \). Post-hoc comparisons using Tukey’s test, with a familywise Type I error rate of .05, revealed that participants who had been categorised as having severe disability had significantly greater injury mean scores than participants with mild disability. Examination of post-MVA distress and disability (illustrated in Figure 7.2) showed a significant main effect: \( F(3, 53) = 7.24, p = .001 \) (anxious or fearful);
\( F (3, 54) = 5.53, p = .002 \) (sad or depressed). Post-hoc analysis (using Tukey’s test, with a familywise Type I error rate of .05) revealed that participants with severe disability had significantly greater anxious or fearful scores than participants with no disability. Similarly, MVA survivors with severe and moderate disability had significantly greater sad or depressed scores than those categorised as having no disability. There were no significant differences between gender, age, or MVA category type and disability scores.

![Figure 7.2](image.png)

**Figure 7.2.** Physical disability categories and post-MVA distress levels.

A path analysis model was developed to test the hypothesis that injury severity, initial psychological distress (feeling sad or depressed) as well as physical disability at 3-months would predict on-going disability 6-months post-MVA. In order to examine the unique contribution of injury severity and psychological distress on physical disability at 6-months post-MVA, a nested path analysis model was developed in which the path coefficient from 3-month to 6-month physical disability was constrained. Furthermore, to determine the independent effect of subjective injury severity on physical disability, the relationship
between the objective measure of injury severity (ISS) and disability was examined. The model was developed using the same path analytic procedures employed in Phase 1 (see section 5.3.1). Correlation coefficients between variables of interest were all significant with the exception of objectively assessed injury severity (see Table 7.2) which was not included in the path model. Skew and kurtosis levels were within acceptable limits (< 2.0 and 5.0 respectively).

Table 7.2

*Correlation Coefficients of Variables Associated with Physical Disability*

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 ISS</td>
<td>-</td>
<td>-.03</td>
<td>.18</td>
<td>-.05</td>
<td>-.17</td>
</tr>
<tr>
<td>2 Injury Severity</td>
<td>-</td>
<td>.16</td>
<td>.40**</td>
<td>.34**</td>
<td></td>
</tr>
<tr>
<td>3 Sad or Depressed</td>
<td>-</td>
<td>.52**</td>
<td>.48**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 3-Month Physical Disability</td>
<td>-</td>
<td>.81**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 6-Month Physical Disability</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. N = 58. ISS = Injury Severity Score.*

**p < .01.

The finding that objective injury severity was not significantly associated with subjective injury severity, feeling sad or depressed nor physical disability at 6-months post-MVA may have been influenced by the small sample size. Only MVA survivors admitted to JHH had their ISS recorded (n = 60), 11 of whom participated in Phase 3. To further explore these relationships with a larger sample, data analysis from Phase 2, which comprised 20 participants’ ISS ratings, was conducted. Results, quite similar to those at 6-months, indicated that at 3-months post-MVA, objective injury severity was not significantly
associated with the subjective injury severity rating \( (r = -.08, p = .731) \), feeling sad or depressed \( (r = .22, p = .345) \), nor with physical disability \( (r = -.04, p = .873) \).

The (unconstrained) path model provided a good fit to the data, \( \chi^2 = 1.56, p < .212 \) \( (df = 1; N = 58) \), \( \chi^2/df = 1.56 \), CFI = .993; RMSEA = .099, AIC =19.56, and explained 64.4% of 6-month physical disability variance. As illustrated in Figure 7.3 the path coefficient from 3-month to 6-month physical disability was significant, as were the direct paths from injury severity and sad or depressed to 3-month physical disability. The direct paths from injury severity and sad or depressed to 6-month disability were not significant, though indirectly via 3-month disability both injury severity \( (\beta = .25, p = .003) \) and sad or depressed \( (\beta = .36, p = .003) \) had a significant effect on 6-month disability. When the path from 3-month to 6-month disability was constrained the direct paths from injury severity and sad or depressed to 6-month disability were significant.

The results of nested model comparisons (see Table 7.3) showed that the unconstrained model provided a significantly better fit to the data. Overall, these findings indicate that the strongest influence on 6-month physical disability was 3-month disability which was directly influenced by injury severity and feeling sad or depressed. Injury severity and feeling sad or depressed also had a significant influence on disability over time, but the effects were indirect and fully mediated via 3-month physical disability.
7.3 Psychopathology

7.3.1 Posttraumatic Stress Disorder and Pre-MVA Factors

At 6-months post-MVA 34.5% of MVA survivors \((n = 20)\) had PTSD. No significant gender differences were found in the proportion of participants with or without PTSD nor between gender and PTSD symptom severity scores (see Table 7.4).
Table 7.4

*Pre-MVA Variable Comparisons Between Participants With and Without PTSD*

<table>
<thead>
<tr>
<th>Pre MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>20 (34.5%)</td>
<td>38 (75.5%)</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male n (%)</td>
<td>5 (25%)</td>
<td>15 (39.5%)</td>
<td>$\chi^2 = 1.21 \ ns$</td>
</tr>
<tr>
<td>Female n (%)</td>
<td>15 (75%)</td>
<td>23 (60.5%)</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>Mean (SD)</td>
<td></td>
<td>$t = -1.34 \ ns$</td>
</tr>
<tr>
<td>Education</td>
<td>-</td>
<td>-</td>
<td>$p &gt; .05 \ a$</td>
</tr>
<tr>
<td>Income</td>
<td>-</td>
<td>-</td>
<td>$p &gt; .05 \ a$</td>
</tr>
<tr>
<td>Previous MVA`s (Yes %)</td>
<td>14 (70%)</td>
<td>22 (57.9%)</td>
<td>$\chi^2 = .816 \ ns$</td>
</tr>
<tr>
<td>Number of previous MVA`s</td>
<td>Mean (SD)</td>
<td>1.05 (1.05)</td>
<td>1.32 (1.59)</td>
</tr>
<tr>
<td>MVA Severity Rating c</td>
<td></td>
<td></td>
<td>$p &lt; .001 \ a$</td>
</tr>
<tr>
<td>Not as bad</td>
<td>6 (42.9%)</td>
<td>1 (4.5%)</td>
<td></td>
</tr>
<tr>
<td>Same</td>
<td>0 (0%)</td>
<td>1 (4.5%)</td>
<td></td>
</tr>
<tr>
<td>Worse</td>
<td>8 (57.1%)</td>
<td>20 (91%)</td>
<td></td>
</tr>
<tr>
<td>Past Emotional Problems</td>
<td>Mean (SD)</td>
<td>1.95 (1.19)</td>
<td>1.39 (.946)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>Mean (SD)</td>
<td>4.45(2.28)</td>
<td>4.58 (1.82)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Mean (SD)</td>
<td>4.58 (2.19)</td>
<td>3.69 (2.08)</td>
</tr>
<tr>
<td>Task coping</td>
<td>Mean (SD)</td>
<td>52.00 (12.40)</td>
<td>55.62 (11.30)</td>
</tr>
<tr>
<td>Emotion coping</td>
<td>Mean (SD)</td>
<td>47.60 (12.97)</td>
<td>38.03 (12.31)</td>
</tr>
<tr>
<td>Avoidance coping</td>
<td>Mean (SD)</td>
<td>42.10 (9.59)</td>
<td>41.78 (12.47)</td>
</tr>
</tbody>
</table>

Note. $^a$ Fisher’s Exact Test. $^b$ Welsch-Satterthwaite corrected. $^c$ MVA Severity Rating compares current MVA severity to previous MVAs 1 = not as bad, 2 = same, 3 = worse.

*p < .05. **p < .01. ***p < .001.
Differentiating young from old participants using a median split (45 years) showed that younger MVA survivors experienced greater PTSD symptom severity \( (M = 17.59) \) compared to older participants \( (M = 9.97) \), \( t (56) = 2.40, p = .020 \), but there was no significant age difference between participants with and without PTSD. Education and income level, previous MVAs (including number and severity), past emotional problems, extraversion, as well as task and avoidance coping score comparisons were not significantly different between participants with and without PTSD. However, participants with PTSD scored significantly higher on the pre-MVA measures of emotion-focused coping and trait dissociation compared to participants without PTSD.

### 7.3.2 Posttraumatic Stress Disorder and MVA Characteristics

As shown in Table 7.5, comparisons on MVA category type, injury status, neither subjective and objective injury severity, accident severity, level of alertness, fear of dying nor peritraumatic dissociation showed any significant differences between participants with and without PTSD.
### Table 7.5

**Comparisons of MVA Characteristics Between Participants With and Without PTSD**

<table>
<thead>
<tr>
<th>MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>20 (34.5%)</td>
<td>38 (75.5%)</td>
<td></td>
</tr>
<tr>
<td><strong>MVA Category</strong></td>
<td></td>
<td></td>
<td><strong>p &gt; .05</strong> a</td>
</tr>
<tr>
<td>Driver</td>
<td>12 (60%)</td>
<td>30 (78.9%)</td>
<td></td>
</tr>
<tr>
<td>Passenger</td>
<td>4 (20%)</td>
<td>3 (7.9%)</td>
<td></td>
</tr>
<tr>
<td>Motorcyclist/pillion</td>
<td>1 (5%)</td>
<td>5 (13.2%)</td>
<td></td>
</tr>
<tr>
<td>Pedal Cyclist</td>
<td>2 (10%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Pedestrian</td>
<td>1 (5%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Injury Yes (%)</td>
<td>20 (100%)</td>
<td>37 (97.4%)</td>
<td><strong>p &gt; .05</strong> a</td>
</tr>
<tr>
<td><strong>Injury Severity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report</td>
<td>Mean (SD)</td>
<td>3.30 (.865)</td>
<td>2.95 (.769)</td>
</tr>
<tr>
<td>Accident Severity</td>
<td>Mean (SD)</td>
<td>3.30 (.865)</td>
<td>3.11 (.924)</td>
</tr>
<tr>
<td>Alertness</td>
<td>Mean (SD)</td>
<td>1.75 (1.33)</td>
<td>1.47 (.687)</td>
</tr>
<tr>
<td>Fear of Dying</td>
<td>Mean (SD)</td>
<td>3.25 (1.11)</td>
<td>2.58 (1.40)</td>
</tr>
<tr>
<td>Peritraumatic Dissociation</td>
<td>Mean (SD)</td>
<td>24.25 (9.04)</td>
<td>20.66 (9.09)</td>
</tr>
</tbody>
</table>

*Note.* aFisher’s Exact Test. bWelsch-Satterthwaite corrected.

#### 7.3.3 Posttraumatic Stress Disorder and Post-MVA Factors

As shown in Table 7.6 the results of post-accident variable comparisons showed that the mean scores of psychological distress (assessed at 30-days) were significantly greater for participants with PTSD compared to those without PTSD. There was no difference in the proportion of participants (with or without PTSD) who received treatment.
Table 7.6

Comparisons of Post-MVA Characteristics Between Participants With and Without PTSD Assessed at 6-Months

<table>
<thead>
<tr>
<th>Post-MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>20 (34.5%)</td>
<td>38 (75.5%)</td>
<td></td>
</tr>
<tr>
<td>Sad/Depressed</td>
<td>Mean (SD) 3.70 (1.08)</td>
<td>1.84 (.973)</td>
<td>$t = 6.65^{***}$</td>
</tr>
<tr>
<td>Anxious/Fearful</td>
<td>Mean (SD) 3.75 (1.07)</td>
<td>1.92 (.850)</td>
<td>$t = 7.11^{***}$</td>
</tr>
<tr>
<td>Treatment</td>
<td>(Yes %) 19 (95%)</td>
<td>34 (89.5%)</td>
<td>$p &gt; .05$^a</td>
</tr>
<tr>
<td>Treatment Type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional</td>
<td>3 (15.8%)</td>
<td>2 (5.7%)</td>
<td></td>
</tr>
<tr>
<td>Physical</td>
<td>8 (42.1%)</td>
<td>29 (82.9%)</td>
<td></td>
</tr>
<tr>
<td>Emotional and Physical</td>
<td>8 (42.1%)</td>
<td>4 (11.4%)</td>
<td></td>
</tr>
<tr>
<td>Treatment Duration</td>
<td>Mean (SD) 4.35 (1.30)</td>
<td>3.08 (1.61)</td>
<td>$t = 3.23^{**}$^b</td>
</tr>
<tr>
<td>Physical Disability</td>
<td>Mean (SD) 11.70 (6.03)</td>
<td>4.92 (5.54)</td>
<td>$t = 4.29^{***}$</td>
</tr>
<tr>
<td>Physical Disability Rating</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>0 (0%)</td>
<td>19 (50.0%)</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>3 (15%)</td>
<td>5 (13.2%)</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>5 (25%)</td>
<td>7 (18.4%)</td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>12 (60%)</td>
<td>7 (21.4%)</td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td>Mean (SD) 20.35 (10.59)</td>
<td>6.50 (7.07)</td>
<td>$t = 4.75^{***}$^b</td>
</tr>
<tr>
<td>AUDIT</td>
<td>Mean (SD) 8.50 (10.25)</td>
<td>4.16 (4.76)</td>
<td>$t = 1.79 ns$^b</td>
</tr>
<tr>
<td>Alcohol use post-MVA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased</td>
<td>3 (15%)</td>
<td>8 (21.1%)</td>
<td></td>
</tr>
<tr>
<td>Same</td>
<td>10 (50%)</td>
<td>26 (68.4%)</td>
<td></td>
</tr>
<tr>
<td>Increased</td>
<td>7 (35%)</td>
<td>4 (10.5%)</td>
<td></td>
</tr>
<tr>
<td>Extraversion</td>
<td>Mean (SD) 3.05 (2.21)</td>
<td>4.45 (2.31)</td>
<td>$t = -2.22^*$</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Mean (SD) 6.85 (1.66)</td>
<td>3.89 (2.53)</td>
<td>$t = 5.33^{***}$^b</td>
</tr>
<tr>
<td>Task coping</td>
<td>Mean (SD) 50.00 (9.49)</td>
<td>57.08 (9.05)</td>
<td>$t = -2.78^{**}$</td>
</tr>
<tr>
<td>Emotion coping</td>
<td>Mean (SD) 49.55 (13.98)</td>
<td>36.68 (11.34)</td>
<td>$t = 3.78^{***}$</td>
</tr>
<tr>
<td>Avoidance coping</td>
<td>Mean (SD) 41.65 (9.22)</td>
<td>41.34 (12.20)</td>
<td>$t = 0.09 ns$</td>
</tr>
<tr>
<td>Trait Dissociation</td>
<td>Mean (SD) 53.30 (29.84)</td>
<td>29.42 (18.56)</td>
<td>$t = 3.26^{**}$^b</td>
</tr>
</tbody>
</table>

Note. BDI-II = Beck Depression Inventory-Second Edition. AUDIT = Alcohol Use Disorders Identification Test.

^a Fisher’s Exact Test. ^b Welsch-Satterthwaite corrected.

*p < .05. **p < .01. ***p < .001.
However, comparisons of the type of treatment received showed significant differences. A greater proportion of participants with PTSD received emotional and physical treatment (41.2% vs. 11.4%) and a greater proportion of participants without PTSD received treatment for physical injuries only (82.9% vs. 41.25%). Additionally, results also showed that participants with PTSD received treatment for a significantly longer duration compared to those without PTSD.

Further comparisons showed that participants with PTSD scored significantly higher on depression symptom severity and physical disability compared to participants without PTSD. Analysis of disability categories revealed significant differences with 50% of participants without PTSD categorised with no disability (vs. 0% with PTSD), and 60% of participants with PTSD with severe disability (vs. 21.4% without PTSD). Comparisons of AUDIT mean scores showed that MVA-survivors with PTSD scored significantly more highly but the variances of the groups were not equal, and as such the Welsch-Satterthwaite corrected degrees of freedom and probability values were used. This increased the probability from 0.031 to 0.086. Analysis of the category proportions of alcohol use since the accident (Decreased, Same or Increased) showed no significant differences.

In terms of post-MVA personality, participants without PTSD reported significantly greater levels of extraversion whereas participants with PTSD reported significantly greater levels of neuroticism. Analysis of post-accident coping revealed that there was no significant difference between participants with and without PTSD on the use of avoidance coping strategies, however, participants without PTSD endorsed using significantly more task coping strategies and participants with PTSD endorsed significantly more emotion-focused
coping strategies. Comparisons of post-accident dissociation mean scores showed that participants with PTSD experienced a significantly greater level of dissociation than participants without PTSD, as shown in Table 7.6.

### 7.3.4 Alcohol, Depression, and Comorbidity

Hazardous levels of alcohol consumption (Audit score equal or greater than 8) was reported by 24.1% ($n = 14$) participants. Analysis of alcohol consumption patterns displayed in Table 7.7 show that 19% ($n = 11$) of participants had decreased their alcohol consumption since their MVA, 62% ($n = 36$) remained the same, and 19% ($n = 11$) had increased their alcohol intake. Examination of participants’ consumption patterns (whose alcohol intake was categorised as hazardous) revealed no significant differences between participants with and without PTSD and also with and without depression.

Table 7.7

*Summary of Alcohol Usage Patterns in MVA Survivors With and Without PTSD and Depression*

<table>
<thead>
<tr>
<th>Usage Pattern</th>
<th>$N$</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Depression</th>
<th>No Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased</td>
<td>11 (2)</td>
<td>3 (0)</td>
<td>8 (2)</td>
<td>4 (1)</td>
<td>7 (1)</td>
</tr>
<tr>
<td>Same</td>
<td>36 (4)</td>
<td>10 (2)</td>
<td>26 (2)</td>
<td>4 (2)</td>
<td>32 (2)</td>
</tr>
<tr>
<td>Increased</td>
<td>11 (8)</td>
<td>7 (5)</td>
<td>4 (3)</td>
<td>4 (3)</td>
<td>7 (5)</td>
</tr>
<tr>
<td>Total</td>
<td>58 (14)</td>
<td>20 (7)</td>
<td>38 (7)</td>
<td>12 (6)</td>
<td>46 (8)</td>
</tr>
</tbody>
</table>

*Note.* Numbers in parenthesis represent hazardous alcohol consumption levels

There was a significant difference of depression symptom severity scores between participants who consumed ($M = 19.29$) and those who did not consume hazardous alcohol
levels ($M = 9.89$), ($t (56) = 3.21, p = .002$). A significant difference was also found on PTSD symptom severity scores between participants who consumed hazardous alcohol levels ($M = 19.86$) and those who did not ($M = 11.84$), ($t (56) = 2.14, p = .036$). Analysis of the PTSD sub-scales (avoidance, intrusion and arousal) revealed that intrusion mean scores were significantly higher for participants who drank alcohol at a hazardous level ($M = 5.64$) compared to participants who did not ($M = 3.18$), ($t (56) = 1.84, p = .034$).

There was no significant difference in the proportion of participants who did or did not report hazardous alcohol intake and whether or not they had experienced past emotional problems. Comparisons of post-MVA personality and coping variables revealed that participants who had been consuming alcohol at a hazardous level had significantly higher neuroticism scores ($M = 6.43$) than participants whose alcohol consumption was not hazardous ($M = 4.43$), ($t (56) = 2.55, p = .013$), and also on avoidance coping ($M = 46.57$ vs. $M = 39.57$ respectively), ($t (56) = 2.02, p = .048$). There was no significant difference on physical disability mean scores between participants who consumed and those who did not consume hazardous levels of alcohol, nor between disability categories. No significant differences were found on task or emotion focused coping nor on post-MVA dissociation. Gender comparisons revealed no significant differences on consumption patterns (decreased, same, and increased) or on hazardous level. Age comparisons on consumption patterns were significant, ($\chi^2 (2, N = 58) = 12.18, p = .001$); only 17.2% of older participants changed their drinking pattern compared to 58.6% of younger participants who reported that alcohol consumption had changed since the accident.
Further analysis of age showed that participants categorised as consuming a hazardous level of alcohol were significantly younger than participants who were not consuming a hazardous level (36.79 years vs. 47.86 years respectively), $t(56) = -2.04$, $p = .045$.

Depression (BDI-II total score equal or greater than 20) was reported by 20.7% ($n = 12$) of participants. No gender or age differences were found between participants categorised with or without depression. Comparisons of the proportion of participants with depression and those who had also experienced past emotional problems were not significant. As illustrated in Figure 7.4, 45% of participants ($n = 9$) with PTSD experienced comorbid depression, and 35% ($n = 7$) also reported concurrent hazardous alcohol use. Fifty percent ($n = 6$) of participants with depression also reported concurrent hazardous alcohol use and four (6.89%) participants reported comorbid PTSD, depression, and hazardous alcohol use.

*Figure 7.4. PTSD, Depression, Alcohol Abuse comorbid inter-relationships.*
7.4 Predictors of Post-MVA Psychopathology

Analyses of factors that predicted post-MVA psychopathology comprised an initial examination of the correlation coefficients between pre, peri, and 3-month post-accident variables associated with PTSD and depression symptom severity at 6-months post-MVA, the results of which are summarised in Table 7.8. Separate Multiple Linear Regression (MLR) analyses were then conducted separately for pre, peri, and post-MVA variables that were significantly correlated with PTSD as well as depression symptom severity. Hierarchical multiple regression was then conducted using the pre, peri, and post-MVA variables from the MLRs that significantly predicted PTSD and depression severity.

7.4.1 PTSD Symptom Severity

As shown in Table 7.8 pre-accident variables that were significantly associated with PTSD symptom severity comprised age, emotion focused coping, and trait dissociation. Regression analysis revealed that the model was significant, $F(3, 57) = 5.61, p = .002$, and accounted for 23.8% of the variance, with emotion focused coping significantly predicting PTSD symptom severity ($\beta = .36, p = .038$). Accident related variables that were significantly correlated with PTSD symptom severity comprised fear of dying, peritraumatic dissociation, and accident severity. The regression model was significant, $F(3, 57) = 3.67, p = .018$, and explained 17% of the variance with peritraumatic dissociation ($\beta = .27, p = .043$) the only significant predictor of PTSD symptom severity. Examination of post-MVA variable correlations revealed that feeling sad or depressed and anxious or fearful (assessed at 1-month), and treatment duration, physical disability, and total audit score (assessed at 3-months post-MVA) were significantly correlated with PTSD symptom severity (see Table 7.8).
Table 7.8

Summary of Significant Pre, Peri, and Post-MVA Correlation Coefficients for 6-Month PTSD and Depression Symptom Severity

<table>
<thead>
<tr>
<th>Variable</th>
<th>PTSD Symptom Severity</th>
<th>Depression Symptom Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre and MVA Characteristics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-.28*</td>
<td>-.15</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>.18</td>
<td>.30*</td>
</tr>
<tr>
<td>Trait Dissociation</td>
<td>.36**</td>
<td>.46**</td>
</tr>
<tr>
<td>Emotion Focused Coping</td>
<td>.47**</td>
<td>.53**</td>
</tr>
<tr>
<td>Past Emotional Problems</td>
<td>.18</td>
<td>.41**</td>
</tr>
<tr>
<td>Level of Alertness</td>
<td>.24</td>
<td>.31*</td>
</tr>
<tr>
<td>Fear of Dying</td>
<td>.27*</td>
<td>.20</td>
</tr>
<tr>
<td>Peritraumatic Dissociation</td>
<td>.31*</td>
<td>.23</td>
</tr>
<tr>
<td>Accident Severity</td>
<td>.28*</td>
<td>.23</td>
</tr>
<tr>
<td>3-Months Post-MVA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sad or Depressed a</td>
<td>.69**</td>
<td>.67**</td>
</tr>
<tr>
<td>Anxious or Fearful a</td>
<td>.71**</td>
<td>.62**</td>
</tr>
<tr>
<td>Treatment Duration</td>
<td>.34**</td>
<td>.09</td>
</tr>
<tr>
<td>Physical Disability</td>
<td>.58**</td>
<td>.49**</td>
</tr>
<tr>
<td>AUDIT</td>
<td>.37**</td>
<td>.49**</td>
</tr>
</tbody>
</table>

Note. N = 58. AUDIT = Alcohol Use Disorders Identification Test.

\(^a\) Assessed at 1-Month Post-MVA.

\(\ast p < .05\). \(\ast\ast p < .01\).

Regression analysis of 1-month variables showed that the model was significant, \((F (2, 57) = 31.42, p < .001)\) and explained 53.3% of the variance. Anxious or fearful had a stronger influence \((\beta = .43, p = .014)\) than sad or depressed \((\beta = .33, p = .055)\) which displayed a clear trend towards significance. Regression analysis of 3-month variables showed that the model was significant \((F (3, 57) = 12.04, p < .001)\) and explained 40% of the variance. Physical disability was the strongest predictor \((\beta = .41, p = .003)\) followed by total audit
score ($\beta = .28 \ p = .019$). Results of multicollinearity tests from the regressions revealed that tolerance levels were all well above zero (range = .297 to .924) and VIF were not large (range = 1.08 to 3.36). It was noted that the 1-month variables of feeling sad or depressed and anxious or fearful were strongly correlated ($r = .84, \ p < .001$) indicating a high degree of shared variance.

As shown in Table 7.9 results from the hierarchical multiple regression indicated that the best fitting model is Model 4 ($F (5, 57) = 15.05, \ p < .001, \ R^2 = .59$) in which anxious or fearful ($\beta = .42, \ p = .002$) and physical disability ($\beta = .28, \ p = .015$) significantly predicted PTSD symptom severity. Tests for multicollinearity indicated a low level, with tolerance levels ranging from .484 to .871 and VIF between 1.14 and 2.06.

Table 7.9

*Hierarchical Multiple Regression Analysis Predicting PTSD Symptom Severity*

*Note.* Audit = Alcohol Use Disorders Identification Test.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\beta$</td>
<td>$p$</td>
<td>$\beta$</td>
<td>$p$</td>
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<tr>
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<td>.001</td>
<td>.41</td>
<td>.002</td>
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<td>Peritraumatic Dissociation</td>
<td>.16</td>
<td>.195</td>
<td>.14</td>
<td>.169</td>
</tr>
<tr>
<td>Anxious or Fearful</td>
<td>.63</td>
<td>.001</td>
<td>.42</td>
<td>.002</td>
</tr>
<tr>
<td>Physical Disability</td>
<td></td>
<td></td>
<td>.28</td>
<td>.015</td>
</tr>
<tr>
<td>Total AUDIT Score</td>
<td></td>
<td></td>
<td>.06</td>
<td>.533</td>
</tr>
<tr>
<td>Total $R^2$</td>
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<td>.24</td>
<td>.54</td>
<td>.59</td>
</tr>
<tr>
<td>$\Delta R^2 (p)$</td>
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<td>.195</td>
<td>.29</td>
<td>.001</td>
</tr>
</tbody>
</table>

*Note.* $N = 58$.  


7.4.2 Depression Symptom Severity

The same procedure was used to determine which variables were predictive of depression symptom severity. Examination of correlation coefficients (summarized in Table 7.8) showed that the pre-MVA variables of neuroticism, emotion focused coping, trait dissociation, past emotional problems, and level of alertness were significantly correlated with depression. Multiple linear regression was conducted with results showing the model was significant, \( F(5, 57) = 5.77, p < .001 \), and explained 35.7% of the variance. Of the pre-accident variables, emotion-focused coping was the only predictor to reach significance (\( \beta = .36, p = .044 \)). Examination of post-MVA variable correlations revealed that feeling sad or depressed and anxious or fearful (assessed at 1-month) and physical disability as well as total AUDIT score (assessed at 3-months post-MVA) was significantly correlated with depression symptom severity. Regression analysis of 1-month variables was conducted first; results showed that the model was significant, \( F(2, 57) = 23.92, p < .001 \) and explained 46.5% of the variance. Sad or depressed was found to be a significant predictor (\( \beta = .51, p = .006 \)).

Regression analysis of 3-month variables showed that the model was significant \( F(2, 57) = 16.21, p < .001 \) and explained 31.7% of the variance, with physical disability (\( \beta = .38, p = .001 \)) and total AUDIT score (\( \beta = .38, p = .001 \)) significantly predicting depression symptom severity. Multicollinearity tests from the 3 regression equations showed that all tolerance levels were above zero, ranging from .297 to .951, and VIFs were not large, ranging from 1.05 to 3.36.
Results from the hierarchical multiple regression (see Table 7.10) indicated that the best fitting model was Model 3 ($F(5, 57) = 16.79, p < .001, R^2 = .56$) in which sad or depressed ($\beta = .35, p = .010$) and (trait) emotion-focused coping ($\beta = .24, p = .042$) significantly predicted depression symptom severity. Physical disability ($\beta = .22, p = .052$) and total Audit score ($\beta = .19, p = .068$), did not quite reach significance, but did display a clear trend. Tests for multicollinearity indicated a low level with tolerance levels ranging from .482 to .786 and VIF between 1.27 and 2.07.

Table 7.10

*Hierarchical Multiple Regression Analysis Predicting Depression Symptom Severity*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th></th>
<th>Model 2</th>
<th></th>
<th>Model 3</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>$\beta$</td>
<td>$p$</td>
<td>$\beta$</td>
<td>$p$</td>
<td>$\beta$</td>
<td>$p$</td>
</tr>
<tr>
<td>Trait Emotion Focused Coping</td>
<td>.53</td>
<td>.001</td>
<td>.23</td>
<td>.053</td>
<td>.24</td>
<td>.042</td>
</tr>
<tr>
<td>Sad or Depressed</td>
<td>.55</td>
<td>.001</td>
<td>.35</td>
<td>.010</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical Disability</td>
<td></td>
<td></td>
<td>.22</td>
<td>.052</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total AUDIT Score</td>
<td></td>
<td></td>
<td>.19</td>
<td>.068</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total $R^2$</td>
<td>.28</td>
<td>.49</td>
<td>.56</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\Delta R^2$ ($p$)</td>
<td>.21</td>
<td>(.001)</td>
<td>.07</td>
<td>(.022)</td>
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<td></td>
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</tbody>
</table>

*Note. N = 58. AUDIT = Alcohol Use Disorders Identification Test.*

7.5 Variable Comparisons Over-Time

In order to assess the influence of variables over time in relation to PTSD diagnosis, repeated measures ANOVAs were conducted using Greenhouse-Geisser correction to account for any violations of sphericity. Using PTSD status as a grouping variable, analysis of (retrospective) pre-MVA and 6-month post-MVA personality scores showed significant
effects of neuroticism, \( F(1, 56) = 26.37, p < .001 \), and PTSD status \( F(1, 56) = 12.76, p = .001 \), as well as the Neuroticism x PTSD status interaction, \( F(1, 56) = 13.38, p = .001 \) (see Figure 7.5). Examination of pre-MVA neuroticism scores showed no difference between groups, however at 6-months post-MVA there was a significant difference. For participants with PTSD their scores had increased over time, \( t(19) = -5.45, p < .001 \), and were significantly greater than participants who did not have PTSD \( t(53) = 5.31, p < .001 \) (Welsch-Satterthwaite corrected).

Figure 7.5. Comparisons of pre and post-MVA neuroticism mean scores for participants with and without PTSD.

Results also showed a significant effect of extraversion, \( F(1, 56) = 8.91, p = .004 \), as well as the Extraversion x PTSD status interaction, \( F(1, 56) = 6.11, p = .016 \), but there was no
significant between groups effect, $F (1, 56) = 2.05, p = .157$. As illustrated in Figure 7.6 pre-MVA extraversion scores were similar for both groups, but for the PTSD group extraversion scores decreased significantly over-time, $t (19) = 3.29, p = .004$, with the PTSD group reporting significantly lower scores at 6-months post-MVA than the no-PTSD group, $t (56) = -2.22, p = .030$.

**Figure 7.6.** Comparisons of pre and post-MVA extraversion mean scores for participants with and without PTSD.

Comparisons of pre and post-MVA coping styles, and of 3 and 6-month post-MVA AUDIT scores, treatment duration, and physical disability scores showed significant group effects. However, these group differences have been previously described in sections 7.3.1 and
7.3.4 (see also Tables 7.4 and 7.6) when variable differences between participants with and without PTSD were examined.

Analysis on the effect of dissociation revealed a clear trend towards significance, $F(1, 56) = 3.94, p = .052$, and similarly for the interaction between Dissociation x PTSD status, $F(1, 56) = 3.90, p = .053$. There was a significant group effect, $F(1, 56) = 11.62, p = .001$, with the PTSD group scoring significantly higher than the group without PTSD at both time points (see Figure 7.7).

![Figure 7.7. Comparisons of pre and post-MVA dissociation mean scores for participants with and without PTSD.](image)

Analysis of the inter-relationships between pre, peri, and post-MVA dissociation as well as PTSD symptom severity and PTSD diagnosis (coded: 1 = PTSD and 2 = No PTSD)
showed that pre-MVA dissociation was strongly associated with post-MVA dissociation as well as being significantly correlated with PTSD symptom severity and diagnosis, but not with peritraumatic dissociation (see Table 7.11). Peritraumatic dissociation displayed relatively weak, though significant correlations with 6-month dissociation and PTSD symptom severity, but not with PTSD diagnosis. Of the dissociation measures, persistent dissociation had the strongest association with both PTSD symptom severity and diagnosis.

Table 7.11

*Pre, Peri, and Post-MVA Dissociation Correlation Coefficients for PTSD Symptom Severity and Diagnosis*

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
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<tr>
<td>1 Pre-MVA Dissociation</td>
<td>-</td>
<td>.17</td>
<td>.67**</td>
<td>.36**</td>
<td>.30*</td>
</tr>
<tr>
<td>2 Peritraumatic Dissociation</td>
<td>-</td>
<td>.27*</td>
<td>.31*</td>
<td>.19</td>
<td></td>
</tr>
<tr>
<td>3 6-Month Post-MVA Dissociation</td>
<td>-</td>
<td></td>
<td>.69**</td>
<td>.45**</td>
<td></td>
</tr>
<tr>
<td>4 PTSD Symptom Severity</td>
<td>-</td>
<td></td>
<td></td>
<td>.71**</td>
<td></td>
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<tr>
<td>5 PTSD Diagnosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-</td>
</tr>
</tbody>
</table>

*Note. N = 58. PTSD = Posttraumatic Stress Disorder
*p < .05. **p < .01.*

In order to further assess the influence of dissociation on PTSD symptom severity and PTSD diagnosis hierarchical multiple regression and binary logistic regression (respectively) was conducted with pre-MVA dissociation entered first, followed by peritraumatic dissociation, and then 6-month dissociation. As shown in Table 7.12 pre-MVA dissociation had a stronger influence than peritraumatic dissociation in the
prediction of PTSD symptom severity (Model 2), however when 6-month post-MVA dissociation was included (Model 3), the influence of these variables was cancelled out. Hence, the best fitting model for predicting PTSD symptom severity was Model 3 ($F (3, 57) = 19.59, p < .001, R^2 = .52$) with ongoing dissociation having a strong influence on PTSD symptom severity. Tests for multicollinearity indicated a low level with tolerance levels ranging from .524 to .930 and VIF between 1.07 and 1.91.

The influence of pre and post- MVA dissociation on PTSD diagnostic status (peritraumatic dissociation was not included in the analysis as it was not significantly correlated with PTSD) displayed a similar pattern. In isolation, pre-MVA dissociation was a significant predictor of PTSD. However, as shown in Table 7.13, when persistent dissociation was included in the regression equation the influence of pre-MVA dissociation was canceled out. The omnibus test of the model coefficients was significant, $\chi^2 (2, N = 58) = 12.32, p = .002$, and the Hosmer-Lemeshow test was not significant, $\chi^2 (8, N = 58) = 6.78, p = .560$, indicating that model adequately fits the data. Nested model comparisons using the $-2 \log$ likelihood values indicated that the addition of dissociation at 6-months significantly improved model fit, $\chi^2 (2, N = 58) = 7.161 p = .023$, with post-MVA dissociation having the strongest influence on PTSD. In the PTSD group 10/20 was correctly classified and in the non-PTSD group 33/38 were correctly classified which provided an overall accuracy of 74.1%, a sensitivity of 50% and specificity of 86.8%.
Table 7.12

*Hierarchical Multiple Regression of Pre, Peri, and Post-MVA Dissociation on PTSD Symptom Severity*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>$\beta$</td>
<td>$p$</td>
<td>$\beta$</td>
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<tr>
<td>Pre-MVA Dissociation</td>
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<td>.31</td>
</tr>
<tr>
<td>Peritraumatic Dissociation</td>
<td>.25</td>
<td>.044</td>
<td>.14</td>
</tr>
<tr>
<td>6-Month Post-MVA Dissociation</td>
<td></td>
<td></td>
<td>.79</td>
</tr>
<tr>
<td>Total $R^2$</td>
<td>.13</td>
<td>-</td>
<td>.19</td>
</tr>
<tr>
<td>$\Delta R^2$ ($p$)</td>
<td>-</td>
<td>-</td>
<td>.06</td>
</tr>
</tbody>
</table>

*Note.* $N = 58.$

Table 7.13

*Binary Logistic Regression of Pre and Post-MVA Dissociation on PTSD Diagnosis*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wald</td>
<td>OR</td>
</tr>
<tr>
<td>Pre-MVA Dissociation</td>
<td>4.75</td>
<td>1.03</td>
</tr>
<tr>
<td>6-Month Post-MVA Dissociation</td>
<td>5.39</td>
<td>1.047</td>
</tr>
</tbody>
</table>

*Note.* $N = 58.$ OR = odds ratio.
7.6 Discussion

The Phase 3 component of the study showed that the strongest influence on physical disability at 6-months post-accident was physical disability assessed 3-months earlier. Results from Phase 2 showed that subjective reports of injury severity and initial psychological distress (feeling sad or depressed) were predictive of physical disability at 3-months post-MVA. However, in the presence of 3-month physical disability the effect of injury severity and sadness on physical disability at 6-months was reduced and was mediated via 3-month physical disability.

There was no evidence that objectively assessed injury severity was associated with physical disability. This finding was somewhat surprising as it was assumed that an initial medical rating of injury severity would be associated with physical disability. The use of different types of assessment (subjective vs. objective) may account for this finding, and perhaps an objective measure of injury severity would be more strongly aligned with an objective assessment of physical disability, which is something future studies may need to consider. Furthermore, the assessment of disability used in the current study was predominantly based on physical activity restrictions (reduced mobility), and as such may not readily capture the full extent of injuries sustained in the MVA that do not restrict mobility, such as cosmetic impairments and facial deformities.

These findings provide support for the first hypothesis that subjective reports of injury severity, initial psychological distress (feeling sad or depressed) and physical disability at 3-months would predict on-going disability 6-months post-MVA. These results highlight the influential role of personal appraisal of injury severity and emotional response in
maintaining physical disability over time. Furthermore, results also showed that initial psychological distress and physical disability contributed to the maintenance of PTSD and depression symptom severity. These results support the second hypothesis that initial psychological distress and physical disability would be associated with the maintenance depression and PTSD symptom severity at 6-months, and are consistent with the results of previous studies (Blanchard et al., 1997; Ehlers et al., 1998; Green et al., 1993; Hamanaka et al., 2006; Jenewein et al., 2009; Mayou & Bryant, 2001, 2002; Mayou et al., 1997; O’Donnell et al, 2004). Participants who at 30-days post-accident experienced feelings of fear and anxiety, and who reported physical disability at 3-months after their MVA, were more likely to experience greater PTSD symptom severity. Participants who initially felt sad or depressed at 30-days post-accident were also more likely to experience greater depressive symptoms at 6-months post-accident. The influence of disability on the maintenance of depressive symptoms when controlling for initial psychological distress was significant. However, the strength of this relationship was reduced when initial psychological distress (sad or depressed) was taken into account. Overall, these findings suggest that MVA survivors’ own perception of injury severity, and particularly the degree of initial psychological distress they experience (feeling sad or depressed), independently contribute to ongoing physical disability. Further to this, initial psychological distress at 1-month, and physical disability at 3-months, significantly contribute to the maintenance of both PTSD and depression symptom severity 6-months post-MVA.

These results are in accord with the cognitive model of PTSD developed by Ehlers and Clark which suggests that negative idiosyncratic appraisals of trauma sequelae, including injury and disability, produce a sense of current threat which maintains symptoms by
directly producing negative emotions (such as fear, sadness, and anger) that promote dysfunctional coping strategies that in turn prevent cognitive change from occurring. For some MVA survivors physical disability, including scarring and disfigurement, ongoing medical treatment, as well as pain, present as significant secondary stressors. These secondary stressors for some trauma survivors can be independent causes of anxiety and depression which may make it difficult to distinguish between psychogenic and organic causes of anxiety and depression symptoms (see O’Donnell, Creamer, Bryant, Schnyder, & Shalev, 2003). For example, initial psychological distress (feeling sad or depressed) may have both a psychogenic and an organic derivation. Similarly, physical disability was influenced by injury severity (organic) and psychological distress (psychogenic and/or organic) which in turn contributed to the maintenance of psychopathology. Given the likelihood of sustaining injury as a result of experiencing an MVA, it is important to acknowledge the potential additive effect of injury and physical disability on psychopathology irrespective of whether the cause is physical, psychological or an interaction of both. From a treatment perspective it is important for clinicians to consider the impact of ongoing physical disability which, unfortunately for some MVA survivors, is an integral part of their post-MVA trauma experience.

The results indicated that of the measures of dissociation, comprising pre, peri, and post-MVA, post-MVA (persistent) dissociation was the strongest predictor of PTSD and symptom severity. This supports the third hypothesis, that ongoing dissociation would have the strongest influence on PTSD at 6-months post-accident, and is consistent with the findings of previous studies (Briere et al., 2005; Halligan et al., 2003; Murray et al., 2002). When ongoing dissociation was controlled (6-months post-accident), pre-MVA dissociation
(trait) was predictive of PTSD and symptom severity and, similarly, peritraumatic dissociation was also predictive of symptom severity. However, in the presence of ongoing dissociation the effects of pre and peritraumatic dissociation were no longer significant. These results suggest that at six months post-accident MVA survivors who continue to experience dissociative symptoms are at a higher risk of PTSD severity above and beyond the influence of pre and peritraumatic dissociation. Results also showed that participants who developed PTSD (compared to those who did not) were more inclined to experience dissociative symptoms both prior to and post-MVA. The results of the previous study (Phase 2) showed that pre-accident dissociation was associated with pre-morbid psychological state and was partly independent of peritraumatic dissociation (mediated via emotion focused coping) which was predictive of PTSD symptom severity at 3-months post-accident. Overall, these findings show that pre-MVA dissociation is a vulnerability factor for PTSD and, to a lesser extent, peritraumatic dissociation. There was also a clear trend \( p = .052 \) for participants with PTSD to experience an increase in dissociative symptoms over time. The effect of persistent post-trauma dissociation is thought to contribute to the maintenance of PTSD by impeding the access, integration, and resolution of trauma memories (see Ehlers & Clark, 2000; Foa & Hearst-Ikeda, 1996).

This study showed that participants with PTSD did not differ from those without PTSD in pre-MVA neuroticism scores. However, at 6-months post-MVA neuroticism scores of participants with PTSD had increased significantly and were significantly higher on this measure than participants without PTSD. These findings partly support the fourth hypothesis that higher pre and post-MVA neuroticism scores would be associated with PTSD. These results are consistent with some MVA studies (Conlon et al., 1999; Mayou et
al., 1993) which have reported that neuroticism was not associated with PTSD, but are inconsistent with others (Dorfel et al., 2008; Holeva & Tarrier, 2001) which have reported that neuroticism predicted PTSD. However, the findings of the later studies need to be interpreted with a degree of caution. Time since MVA in the cross-sectional study of Dorfel et al. was (on average) 52.9 months with a sample size of 44, and in the study of Holeva and Tarrier the response rate at time 1 was only 14%, and this decreased further at time 2. These methodological limitations reduce the strength of the conclusions that may be drawn since sample representativeness and recall bias pose as potential confounds.

Results from the Phase 2 study showed that a neurotic predisposition was not predictive of PTSD, though it was indirectly associated with peritraumatic dissociation. Overall, these findings suggest that a neurotic predisposition is a vulnerability factor (interacting with other pre-morbid variables) for acute dissociation, but not for PTSD at either 3 or 6 months post-accident. The increase in the level of neuroticism over time for participants with PTSD suggests that neuroticism may be a consequence rather than a cause of PTSD, which is in accord with the findings of Clark, Watson, and Mineka (1994) who, in their review of personality and psychopathology, concluded that the experience of psychopathology (anxiety or mood disorder) can lead to temporary or permanent changes in the level of neuroticism. Though extraversion was not a focus of this study, the significant decrease (from pre to post) in the level of extraversion for participants with PTSD also illustrates personality change associated psychopathology. Extraversion levels for participants who did not have PTSD remained stable, but for participants with PTSD their level of extraversion decreased significantly at 6-months and was significantly lower compared to
participants without PTSD. These results are consistent with the findings of previous MVA studies (Dorfel et al., 2008; Holeva & Tarrier, 2001; Nightingale & Williams, 2000).

This study showed that participants who consumed alcohol at a hazardous level were more likely to experience depressive symptoms 6-months after their MVA. It was also shown in the previous phase that drinking alcohol at a hazardous level was associated with depression symptom severity, but not with PTSD (3-months post-MVA). However, 6-months after the MVA, participants who consumed alcohol at a hazardous level experienced greater PTSD symptom severity in addition to that of depression, compared to participants whose alcohol intake was not hazardous. These results support the fifth hypothesis, which proposed that hazardous alcohol levels would be associated with the maintenance of depression symptoms. The finding that drinking at hazardous levels was associated with greater PTSD symptom severity at 6-months but not at 3-months may in part be explained by a change in drinking patterns. Overall, usage patterns increased from 28.5% (participants indicating that they had increased their alcohol consumption since the accident) at three months to 57% at six months. Furthermore, at three months post-MVA 37.5% of participants with PTSD who had been consuming alcohol at a hazardous level had increased their consumption (compared to before the MVA), whereas at 6-months 71% of participants with PTSD who had been drinking at a hazardous level had increased their consumption. This pattern of results indicates that at 3-months hazardous consumption levels were more strongly associated with premorbidity, whereas at 6-months post-MVA increased use of alcohol was associated with the maintenance of both PTSD and depression symptoms. Given that hazardous consumption of alcohol was associated with increased
intrusions and avoidance coping strategies, it is reasonable to suggest that the change in
drinking patterns may, in part, reflect maladaptive coping in response to (ongoing)
distressing intrusions, which is consistent with the self-medication hypothesis. Avoidance
strategies, including the use of alcohol, maintains symptoms by preventing emotional
processing of the event and this interferes with the integration of trauma memories and
restructuring of dysfunctional trauma cognitions (Clohessy & Ehlers, 1999; Dunmore et al.,
2001; Ehlers et al., 1998; Steil & Ehlers, 2000).

The results of this study showed that, independent of peritraumatic dissociation,
participants who generally utilise emotion-focused coping strategies were more likely to
experience greater PTSD symptom severity 6-months post-MVA. These results support the
sixth hypothesis that trait emotion-focused coping would be associated with the
maintenance of PTSD. However, in the presence of 1-month (psychological distress) and 3-
month (physical disability) outcome variables, the influence of coping was reduced, such
that its effect was no longer significant. Overall, for participants who generally use
emotion-focused coping strategies it appears that upon trauma exposure, the cognitive and
emotional demands of the situation may overwhelm coping resources and these increase the
likelihood of acute dissociation, which is, in turn, associated with the development of
PTSD. The results from the present study extend previous research by suggesting that the
effect of emotion-focused coping as a trait is most influential on immediate trauma
response and the development of PTSD but not the maintenance of symptoms.
One of the aims of this study was to describe the incidence of psychopathology and comorbidity. The incidence rate of PTSD at 6-months post-MVA of 34% is similar to that found at Phase 2 (37%). The number of participants who also could be categorised as having depression (using predefined BDI-II scoring criteria) at 3-months (20%) was similar to that at 6-months post-MVA at 21%. These results show, even with a PTSD recovery rate of 42.5%, a considerable degree of persistent psychopathology. Delayed onset PTSD was reported by 5% \((n = 3)\) of participants who indicated that their symptoms commenced 6-months after their MVA. The current PTSD rate of 34% is somewhat higher than the weighted average of 15% from the MVA studies reported by Blanchard and Veazey (2001) but it is within the range of MVA 6-month incidence rates (3 to 45%) which were reviewed in section 3.2.

Comorbid depression was common since 45% of participants with PTSD also reported concurrent depressive symptoms, which is consistent with the comorbid rate of 43% found at 3-months post-MVA in Phase 2, and is within the range reported by previous MVA studies (Blanchard, Buckley et al., 1998; Blanchard et al., 1994, 1995, 2004; Frommberger et al., 1998; Kuch et al., 1995). Participants who generally utilise emotion-focused coping strategies, and those who experienced initial feelings of sadness at 1-month post-MVA, were more likely to experience greater depression symptom severity 6-months after their accident. Participants who experienced initial feelings of anxiety 1-month post-MVA, and participants who reported physical disability at 3-months after their accident, were more likely to experience greater PTSD symptom severity at 6-months but, unlike depression symptom severity, trait coping and alcohol use at 3-months were not associated with the maintenance of PTSD symptoms. In contrast to the results of Phase 2, these findings
suggest only a partial shared vulnerability. Three month physical disability contributed to the maintenance of PTSD and to a lesser extent depression symptom severity at 6-months, whereas both physical disability and initial feelings of sadness were comparatively strong predictors PTSD and depression symptom severity at 3-months post-MVA. It is possible that as symptoms become more chronic different predictor variables have a greater influence. It is also acknowledged that the use of different statistical techniques between Phase 2 (path analysis) and Phase 3 (hierarchical multiple regression), and a smaller sample in Phase 3, which resulted in reduced statistical power, may, in part, account for discrepancies between the results.

7.7 Limitations and Summary

One of the limitations of this study was the high attrition rate which resulted in a greater proportion of participants who reported more severe injury compared to participants from Phase 2. However there was no evidence that the severity of injury, as it was assessed objectively with the ISS, differed between Phase 2 and Phase 3. Participants in this phase of the study also scored lower on pre-morbid neuroticism and trait avoidance coping compared to participants from Phase 2, but there were no age or gender biases which were present in the previous study phase. The partial retrospective design (statistical analysis using premorbid factors) limits conclusions regarding causal relationships. Results may have been influenced by recall basis since current psychological state may have affected recall and response to not only current but also premorbid assessments. However, the temporal stability for factors which were assessed at 3-months (retrospectively) and at 6-months was found to be satisfactory: avoidance coping ($r = .80$); emotion focused coping ($r = .75$); neuroticism ($r = .63$); extraversion ($r = .61$); and dissociation ($r = .67$).
The correlations between the single item questions regarding fear/anxiety and sad/depressed in Phase 1 and the PDS and BDI-II at 6-months was .71 and .67 respectively (see table 7.8). Additional support for the use of the simple rating scales was also evident as initial anxiety was found to predict PTSD symptom severity and initial sadness predictive of depression symptom severity.

This study has identified several important factors that have been shown to be associated with ongoing post-MVA psychopathology. The results suggest that MVA survivors with initial post-MVA distress were more likely to report physical disability, PTSD and depression symptoms at 6-months post-accident. In addition to initial fear, participants with physical disability (influenced by injury severity and sadness) and those who experienced persistent dissociation and were at greater risk of PTSD symptom severity 6-months after their accident. The average use of alcohol had increased by 6-months, at which time participants who had been drinking at hazardous levels were at greater risk for depression and PTSD symptom severity. It is proposed that the change in drinking patterns may represent a form of maladaptive coping associated with ongoing distressing intrusions. These results are in accord with the cognitive model of PTSD developed by Ehlers and Clark which suggests that negative idiosyncratic appraisals of trauma sequelae, including disability, the effect of persistent post-trauma dissociation, and avoidance strategies including the use of alcohol, maintain PTSD symptoms by preventing emotional processing of the event which interferes with the integration of trauma memories and the restructuring of dysfunctional trauma cognitions.
The first 3 studies of this research (Phases 1, 2, and 3) examined factors associated with the development, chronicity, and the maintenance of PTSD. The following chapter comprises Phase 4 which includes results of a structured clinical interview to confirm diagnoses and the assessment of cognitive appraisals associated with PTSD at approximately 9-months post-MVA.
Chapter 8

8.1 Phase 4 Study

8.1.1 Aims of the Study

The overall aim of this phase of the study was to describe the incidence of psychopathology and to examine comorbid relationships (PTSD, depression and alcohol abuse). A further aim was to confirm diagnosis (using DSM-IV criteria) of PTSD, and to examine relationships between negative cognitive appraisals and PTSD.

8.1.2 Hypothesis

Based on previous findings and in accordance with the cognitive model of Ehlers and Clark (2000) it is hypothesised that negative appraisals, more specifically negative idiosyncratic appraisals of self and the world, will predict PTSD.
8.2 Results

8.2.1 Demographics

All of the 58 participants from Phase 3 initially agreed to be interviewed, but when they were contacted to arrange a suitable interview time, seven participants declined. Time since MVA to interview ranged from 6.4 to 12.3 months ($M = 8.63$, $SD = 1.48$). The sample ($N = 51$) comprised 38 participants (74.5%) from JHH and 13 participants (25.5%) from The Maitland Hospital. Collectively, there were 19 males and 32 females ranging in age from 18 to 81 years of age ($M = 45.33$, $SD = 17.73$).

In order to ascertain if the participants from Phase 4 were representative from Phase 3 direct comparisons were conducted. As shown in Table 8.1 significant differences were found; participants in Phase 4 had reported feeling less sad or depressed at 1-month and reported having used less avoidant coping and more task coping strategies at 6-months post-accident compared to participants from Phase 3.

8.3 Psychopathology

8.3.1 Posttraumatic Stress Disorder and Pre-MVA Factors

At approximately 9-months post-MVA 11.8% of MVA survivors ($n = 6$) could be diagnosed with PTSD using the Composite International Diagnostic Interview (CIDI), which is based on the DSM-IV criteria. As shown in Table 8.2, comparisons of pre-MVA factors showed that participants with PTSD had significantly higher trait emotion-focused coping scores compared to participants without PTSD.
Table 8.1

Phase 3 and Phase 4 Pre, Peri, and Post-MVA 6-Month Variable Comparisons

<table>
<thead>
<tr>
<th>Variables</th>
<th>Phase 3 (n = 7)</th>
<th>Phase 4 (n = 51)</th>
<th>Patterns of significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>-</td>
<td>-</td>
<td><em>p</em> &gt; .05 (^a)</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>1 (14.3%)</td>
<td>19 (37.3%)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>6 (85.7%)</td>
<td>32 (62.7%)</td>
</tr>
<tr>
<td>Age</td>
<td>Mean (SD)</td>
<td>44.14 (22.28)</td>
<td>45.33 (17.73)</td>
</tr>
<tr>
<td></td>
<td>t = -.162 (ns)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>-</td>
<td>-</td>
<td><em>p</em> &gt; .05 (^a)</td>
</tr>
<tr>
<td>Income</td>
<td>-</td>
<td>-</td>
<td><em>p</em> &gt; .05 (^a)</td>
</tr>
<tr>
<td>Previous MVA`s (Yes %)</td>
<td>3 (42.9%)</td>
<td>33 (64.7%)</td>
<td><em>p</em> &gt; .05 (^a)</td>
</tr>
<tr>
<td>Number of Previous MVA`s</td>
<td>Mean (SD)</td>
<td>1.67 (.577)</td>
<td>2.00 (1.39)</td>
</tr>
<tr>
<td></td>
<td>t = -.407 (ns)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous MVA Rating (^b)</td>
<td>-</td>
<td>-</td>
<td>(\chi^2 = 1.75 (ns)^a)</td>
</tr>
<tr>
<td>Not as bad</td>
<td>1 (33.3%)</td>
<td>6 (18.2%)</td>
<td>-</td>
</tr>
<tr>
<td>Same</td>
<td>0 (0%)</td>
<td>1 (3%)</td>
<td>-</td>
</tr>
<tr>
<td>Worse</td>
<td>2 (66.6%)</td>
<td>26 (78.8%)</td>
<td>-</td>
</tr>
<tr>
<td>Past Emotional Problems</td>
<td>Mean (SD)</td>
<td>1.43 (.787)</td>
<td>1.61 (1.09)</td>
</tr>
<tr>
<td></td>
<td>t = -.416 (ns)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MVA Category</td>
<td>-</td>
<td>-</td>
<td><em>p</em> &gt; .05 (^a)</td>
</tr>
<tr>
<td>Driver</td>
<td>6 (85.7%)</td>
<td>36 (70.6%)</td>
<td>-</td>
</tr>
<tr>
<td>Passenger</td>
<td>1 (14.3%)</td>
<td>6 (11.8%)</td>
<td>-</td>
</tr>
<tr>
<td>Motorcyclist/pillion</td>
<td>0 (0%)</td>
<td>6 (11.8%)</td>
<td>-</td>
</tr>
<tr>
<td>Pedal Cyclist</td>
<td>0 (0%)</td>
<td>2 (3.8%)</td>
<td>-</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
<td>-</td>
</tr>
<tr>
<td>Injury (Yes %)</td>
<td>7 (100%)</td>
<td>50 (98%)</td>
<td><em>p</em> &gt; .05 (^a)</td>
</tr>
<tr>
<td>Injury Severity Self Report</td>
<td>Mean (SD)</td>
<td>3.14 (.378)</td>
<td>3.06 (.759)</td>
</tr>
<tr>
<td></td>
<td>t = .286 (ns)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Accident Severity</td>
<td>Mean (SD)</td>
<td>3.29 (1.11)</td>
<td>3.16 (.880)</td>
</tr>
<tr>
<td></td>
<td>t = .352 (ns)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alertness</td>
<td>Mean (SD)</td>
<td>2.00 (1.82)</td>
<td>1.51 (.784)</td>
</tr>
<tr>
<td></td>
<td>t = .702 (ns)^c</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear of Dying</td>
<td>Mean (SD)</td>
<td>2.71 (1.49)</td>
<td>2.82 (1.33)</td>
</tr>
<tr>
<td></td>
<td>t = -.200 (ns)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peritraumatic Dissociation</td>
<td>Mean (SD)</td>
<td>25.57 (8.14)</td>
<td>21.39 (9.25)</td>
</tr>
<tr>
<td></td>
<td>t = 1.13 (ns)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxious/Fearful</td>
<td>Mean (SD)</td>
<td>3.00 (1.63)</td>
<td>2.49 (1.22)</td>
</tr>
<tr>
<td></td>
<td>t = .994 (ns)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sad/Depressed</td>
<td>Mean (SD)</td>
<td>3.57 (1.39)</td>
<td>2.33 (1.27)</td>
</tr>
<tr>
<td></td>
<td>t = 2.38(^*)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. \(^a\) Fisher’s Exact Test. \(^b\) MVA Severity Rating compares current MVA severity to previous MVAs. \(^c\) Welsch-Satterthwaite corrected.

\(^*\) \(p < .05\).
### Phase 3 and Phase 4 Pre, Peri, and Post-MVA 6-Month Variable Comparisons (continued)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Phase 3 (n = 7)</th>
<th>Phase 4 (n = 51)</th>
<th>Patterns of significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment (Yes %)</td>
<td>7 (100%)</td>
<td>47 (92.2%)</td>
<td><em>p &gt; .05</em></td>
</tr>
<tr>
<td>Treatment Type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional</td>
<td>2 (28.6%)</td>
<td>3 (6.4%)</td>
<td></td>
</tr>
<tr>
<td>Physical</td>
<td>3 (42.8%)</td>
<td>34 (72.3%)</td>
<td></td>
</tr>
<tr>
<td>Both</td>
<td>2 (28.6%)</td>
<td>10 (21.3%)</td>
<td></td>
</tr>
<tr>
<td>Treatment Duration</td>
<td></td>
<td></td>
<td><em>p &gt; .05</em></td>
</tr>
<tr>
<td>1 day</td>
<td>0 (0%)</td>
<td>6 (12.8%)</td>
<td></td>
</tr>
<tr>
<td>Up to 1 week</td>
<td>3 (42.8%)</td>
<td>2 (4.3%)</td>
<td></td>
</tr>
<tr>
<td>Up to 1 month</td>
<td>0 (0%)</td>
<td>5 (10.6%)</td>
<td></td>
</tr>
<tr>
<td>One to 3 months</td>
<td>2 (28.6%)</td>
<td>14 (29.8%)</td>
<td></td>
</tr>
<tr>
<td>More than 3 months</td>
<td>2 (28.6%)</td>
<td>20 (42.5%)</td>
<td></td>
</tr>
<tr>
<td>Physical Disability</td>
<td>Mean (SD) 10.71 (7.47)</td>
<td>6.61 (6.23)</td>
<td><em>t = 1.59 ns</em></td>
</tr>
<tr>
<td>PTSD Symptom Severity</td>
<td>Mean (SD) 16.71 (16.24)</td>
<td>13.37 (12.12)</td>
<td><em>t = .656 ns</em></td>
</tr>
<tr>
<td>BDI-II</td>
<td>Mean (SD) 15.29 (14.72)</td>
<td>11.73 (9.64)</td>
<td><em>t = .857 ns</em></td>
</tr>
<tr>
<td>AUDIT</td>
<td>Mean (SD) 9.57 (11.35)</td>
<td>5.12 (6.61)</td>
<td><em>t = 1.52 ns</em></td>
</tr>
<tr>
<td>Drinking Patterns</td>
<td></td>
<td></td>
<td><em>p &gt; .05</em></td>
</tr>
<tr>
<td>Less</td>
<td>0 (0%)</td>
<td>11 (21.6%)</td>
<td></td>
</tr>
<tr>
<td>Same</td>
<td>4 (57.1%)</td>
<td>32 (62.7%)</td>
<td></td>
</tr>
<tr>
<td>More</td>
<td>3 (42.9%)</td>
<td>8 (15.7%)</td>
<td></td>
</tr>
<tr>
<td>Extraversion</td>
<td>Mean (SD) 4.43 (1.81)</td>
<td>3.90 (2.42)</td>
<td><em>t = .522 ns</em></td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Mean (SD) 5.00 (3.10)</td>
<td>4.90 (2.63)</td>
<td><em>t = .091 ns</em></td>
</tr>
<tr>
<td>Coping</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Task</td>
<td>Mean (SD) 47.43 (8.77)</td>
<td>55.63 (9.51)</td>
<td><em>t = -2.15</em></td>
</tr>
<tr>
<td>Emotion</td>
<td>Mean (SD) 45.00 (9.12)</td>
<td>40.59 (14.16)</td>
<td><em>t = .798 ns</em></td>
</tr>
<tr>
<td>Avoidance</td>
<td>Mean (SD) 52.00 (10.92)</td>
<td>40.00 (10.51)</td>
<td><em>t = 2.81</em>*</td>
</tr>
<tr>
<td>Dissociation</td>
<td>Mean (SD) 36.86 (28.04)</td>
<td>37.76 (25.46)</td>
<td><em>t = -.087 ns</em></td>
</tr>
</tbody>
</table>

**Note.** BDI-II = Beck Depression Inventory II. AUDIT = Alcohol Use Disorders Identification Test.

*a* Fisher’s Exact Test.

* *p < .05. **p < .01
Table 8.2

Pre-MVA Variable Comparisons Between Participants With and Without PTSD

<table>
<thead>
<tr>
<th>Pre MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>6 (11.8%)</td>
<td>45 (88.2%)</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male n (%)</td>
<td>2 (33.3%)</td>
<td>17 (37.8%)</td>
<td></td>
</tr>
<tr>
<td>Female n (%)</td>
<td>4 (66.7%)</td>
<td>28 (62.2%)</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>Mean (SD)</td>
<td>38.67 (6.05)</td>
<td>t = -2.03 ns b</td>
</tr>
<tr>
<td>Education</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Previous MVA`s (Yes %)</td>
<td>5 (83.3%)</td>
<td>28 (62.2%)</td>
<td></td>
</tr>
<tr>
<td>Number of previous MVA`s</td>
<td>Mean (SD)</td>
<td>0.83 (.408)</td>
<td>t = -1.83 ns b</td>
</tr>
<tr>
<td>MVA Severity Rating c</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Not as bad</td>
<td>1 (20%)</td>
<td>5 (17.8%)</td>
<td></td>
</tr>
<tr>
<td>Same</td>
<td>0 (0%)</td>
<td>1 (3.6%)</td>
<td></td>
</tr>
<tr>
<td>Worse</td>
<td>4 (80%)</td>
<td>22 (78.6%)</td>
<td></td>
</tr>
<tr>
<td>Past Emotional Problems</td>
<td>Mean (SD)</td>
<td>2.17 (1.60)</td>
<td>t = 1.33 ns</td>
</tr>
<tr>
<td>Extraversion</td>
<td>Mean (SD)</td>
<td>5.17 (1.72)</td>
<td>t = .797 ns</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Mean (SD)</td>
<td>3.17 (1.72)</td>
<td>t = -.753 ns</td>
</tr>
<tr>
<td>Task coping</td>
<td>Mean (SD)</td>
<td>60.00 (5.47)</td>
<td>t = .952 ns</td>
</tr>
<tr>
<td>Emotion coping</td>
<td>Mean (SD)</td>
<td>50.17 (12.15)</td>
<td>t = 2.04*</td>
</tr>
<tr>
<td>Avoidance coping</td>
<td>Mean (SD)</td>
<td>37.83 (8.86)</td>
<td>t = -.709 ns</td>
</tr>
<tr>
<td>Trait Dissociation</td>
<td>Mean (SD)</td>
<td>43.50 (22.08)</td>
<td>t = 1.20 ns</td>
</tr>
</tbody>
</table>

Note: a Fisher’s Exact Test. b Welsch-Satterthwaite corrected. c MVA Severity Rating compares current MVA severity to previous MVAs 1 = not as bad, 2 = same, 3 = worse. *p < .05.

8.3.2 Posttraumatic Stress Disorder and MVA Characteristics

Comparisons of accident characteristics showed that participants with PTSD experienced a significantly greater level of fear compared to participants without PTSD (see Table 8.3).
Table 8.3

*Comparisons of MVA Characteristics Between Participants With and Without PTSD*

<table>
<thead>
<tr>
<th>MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>6 (11.8%)</td>
<td>45 (88.2%)</td>
<td>p &gt; .05 a</td>
</tr>
<tr>
<td>MVA Category</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Driver</td>
<td>4 (66.7%)</td>
<td>32 (71.1%)</td>
<td></td>
</tr>
<tr>
<td>Passenger</td>
<td>2 (33.3%)</td>
<td>4 (8.9%)</td>
<td></td>
</tr>
<tr>
<td>Motorcyclist/pillion</td>
<td>0 (0%)</td>
<td>6 (13.3%)</td>
<td></td>
</tr>
<tr>
<td>Pedal Cyclist</td>
<td>0 (10%)</td>
<td>2 (4.4%)</td>
<td></td>
</tr>
<tr>
<td>Pedestrian</td>
<td>0 (0%)</td>
<td>1 (2.2%)</td>
<td></td>
</tr>
<tr>
<td>Injury Yes (%)</td>
<td>6 (100%)</td>
<td>44 (97.8%)</td>
<td>p &gt; .05 a</td>
</tr>
<tr>
<td>Injury Severity (Self-report)</td>
<td>3.17 (.408)</td>
<td>3.09 (.741)</td>
<td>t = .244 ns</td>
</tr>
<tr>
<td>Accident Severity</td>
<td>3.67 (1.03)</td>
<td>3.09 (.848)</td>
<td>t = 1.53 ns</td>
</tr>
<tr>
<td>Alertness</td>
<td>1.67 (1.03)</td>
<td>1.49 (.757)</td>
<td>t = .518 ns</td>
</tr>
<tr>
<td>Fear of Dying</td>
<td>4.00 (.632)</td>
<td>2.67 (1.33)</td>
<td>t = 4.09 *** b</td>
</tr>
<tr>
<td>Peritraumatic Dissociation</td>
<td>24.83 (9.45)</td>
<td>20.93 (9.23)</td>
<td>t = .969 ns</td>
</tr>
</tbody>
</table>

*Note.* a Fisher’s Exact Test. b Welsch-Satterthwaite corrected. ***p < .001.

8.3.3 Posttraumatic Stress Disorder and Post-MVA Characteristics

Comparisons of post-MVA variables assessed at 6-months (see Table 8.4) showed that participants with PTSD received treatment for a longer period, reported more physical disability, and experienced greater depression symptom severity. They also reported significantly higher levels of neuroticism and emotion focused coping compared to participants without PTSD.
Table 8.4

Comparisons of Post-MVA Characteristics Between Participants With and Without PTSD Assessed at 6-Months

<table>
<thead>
<tr>
<th>Post-MVA Characteristics</th>
<th>PTSD</th>
<th>No PTSD</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>6 (11.8%)</td>
<td>45 (88.2%)</td>
<td>$p &gt; .05^a$</td>
</tr>
<tr>
<td>Treatment (Yes %)</td>
<td>6 (100%)</td>
<td>41 (91.1%)</td>
<td></td>
</tr>
<tr>
<td>Treatment Type</td>
<td></td>
<td></td>
<td>$p &gt; .05^a$</td>
</tr>
<tr>
<td>Emotional</td>
<td>1 (16.7%)</td>
<td>2 (4.9%)</td>
<td></td>
</tr>
<tr>
<td>Physical</td>
<td>2 (33.3%)</td>
<td>32 (78%)</td>
<td></td>
</tr>
<tr>
<td>Emotional and Physical</td>
<td>3 (50%)</td>
<td>7 (17.1%)</td>
<td></td>
</tr>
<tr>
<td>Treatment Duration</td>
<td>Mean (SD)</td>
<td>4.83 (.408)</td>
<td>3.38 (1.70)</td>
</tr>
<tr>
<td>Physical Disability</td>
<td>Mean (SD)</td>
<td>13.33 (5.68)</td>
<td>5.71 (5.79)</td>
</tr>
<tr>
<td>Physical Disability Rating</td>
<td></td>
<td></td>
<td>$p &gt; .05^a$</td>
</tr>
<tr>
<td>None</td>
<td>0 (0%)</td>
<td>18 (40.0%)</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>0 (0%)</td>
<td>7 (15.6%)</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>2 (33.3%)</td>
<td>9 (20%)</td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>4 (66.7%)</td>
<td>11 (24.4%)</td>
<td></td>
</tr>
<tr>
<td>BDI-II</td>
<td>Mean (SD)</td>
<td>30.00 (6.16)</td>
<td>9.29 (7.04)</td>
</tr>
<tr>
<td>AUDIT</td>
<td>Mean (SD)</td>
<td>9.67 (12.73)</td>
<td>4.51 (5.29)</td>
</tr>
<tr>
<td>Alcohol use post-MVA</td>
<td></td>
<td></td>
<td>$p &gt; .05^a$</td>
</tr>
<tr>
<td>Decreased</td>
<td>2 (33.3%)</td>
<td>9 (20%)</td>
<td></td>
</tr>
<tr>
<td>Same</td>
<td>2 (33.3%)</td>
<td>30 (66.7%)</td>
<td></td>
</tr>
<tr>
<td>Increased</td>
<td>2 (33.3%)</td>
<td>6 (13.3%)</td>
<td></td>
</tr>
<tr>
<td>Extraversion</td>
<td>Mean (SD)</td>
<td>2.50 (2.16)</td>
<td>4.09 (2.42)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>Mean (SD)</td>
<td>7.17 (.753)</td>
<td>4.60 (2.64)</td>
</tr>
<tr>
<td>Task coping</td>
<td>Mean (SD)</td>
<td>52.67 (4.84)</td>
<td>56.02 (9.94)</td>
</tr>
<tr>
<td>Emotion coping</td>
<td>Mean (SD)</td>
<td>57.17 (13.79)</td>
<td>38.38 (12.79)</td>
</tr>
<tr>
<td>Avoidance coping</td>
<td>Mean (SD)</td>
<td>33.33 (8.54)</td>
<td>40.89 (10.51)</td>
</tr>
<tr>
<td>Trait Dissociation</td>
<td>Mean (SD)</td>
<td>72.33 (38.19)</td>
<td>33.16 (19.65)</td>
</tr>
</tbody>
</table>

Note: BDI-II = Beck Depression Inventory-Second Edition. AUDIT = Alcohol Use Disorders Identification Test. $^a$ Fisher’s Exact Test. $^b$ Welsch-Satterthwaite corrected.

**$p < .01$. ***$p < .001$. **
Participants with PTSD also reported having experienced more dissociative symptoms at 6-months. However, given that the variances between the groups was unequal, the Welsch-Satterthwaite corrected degrees of freedom and probability values were used, which increased the probability from $p < .001$ to $p = .053$.

Comparisons of the incidence rates of PTSD at 6 and 9-months (34.5% vs. 11.8% respectively) showed that the rate of PTSD at 9-months was significantly less than that at 6-months post-MVA (McNemar Test $p < .001$). It was noted previously (see section 3.2) that the use of questionnaires (self-report) may limit the reliability of diagnostic classification. Biases (e.g., acquiescence and social desirability), which can influence the accuracy of assessment, may be more likely to occur using self-report measures, whereas assessment using a structured interview may help circumvent participant biases as it allows for direct observation, the use of probe questions, and clinical judgement to confirm diagnosis. It is possible that the significant reduction in PTSD from 6 to 9-months post-MVA may have been influenced by the use of different psychometric assessments (standardised self-report questionnaire vs. structured interview). It is also possible that for some MVA survivors PTSD symptoms may have remitted. Comparisons of the symptom severity scores for participants who met diagnostic criteria at 6-months but not at 9-months and those who met criteria at both time points showed that participants who had PTSD at 6 but not at 9-months had significantly lower symptom severity scores ($M = 19.17, SD = 8.41$) than participants who met diagnostic criteria at both time points ($M = 36.17, SD = 11.05$), $t (16) = 3.64, p = .002$. 
8.3.4 PTSD, Alcohol Abuse Disorder, and Major Depression

Based on the results of the CIDI, 11.8% \((n = 6)\) of participants could be diagnosed with Alcohol Abuse Disorder, and 9.8% \((n = 5)\) of participants could be diagnosed with Major Depression based on the DSM-IV criteria. An examination of previous alcohol usage showed that 50% \((n = 3)\) of participants with Alcohol Abuse Disorder were not drinking at a hazardous level 3-months earlier. As illustrated in Figure 8.1, 33% of participants \((n = 2)\) with PTSD experienced comorbid depression, and (2%) participants \((n = 1)\) experienced comorbid PTSD, Alcohol Abuse Disorder, and Major Depression.

![Figure 8.1. Venn diagram: PTSD, Depression, Alcohol Abuse comorbid inter-relationships.](image)

8.3.5 PTSD and Negative Appraisals

Comparisons of the Posttraumatic Cognitions Inventory (PTCI) appraisal mean scores between participants with and without PTSD (see table 8.5) showed participants with PTSD had significantly higher negative self and negative world appraisals. Analysis of the negative appraisals that predicted PTSD comprised an initial examination of the correlation...
coefficients between self, world, and self-blame with PTSD diagnosis. As shown in Table 8.6, both negative self and negative world displayed moderately strong correlations with PTSD. Negative self-blame displayed a weak significant correlation with negative self but did not significantly correlate with negative world nor with PTSD, and therefore was excluded from the subsequent logistic regression analysis.

Table 8.5

*PTSD and Non-PTSD Group Comparisons of Post-Trauma Appraisals*

<table>
<thead>
<tr>
<th>Appraisals</th>
<th>PTSD</th>
<th>Non-PTSD</th>
<th>Mean</th>
<th>SD</th>
<th>Mean</th>
<th>SD</th>
<th>t</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTCI Negative Self</td>
<td>3.67</td>
<td>1.37</td>
<td>1.64</td>
<td>.70</td>
<td>3.54</td>
<td>5.35</td>
<td>.015</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTCI Negative World</td>
<td>5.26</td>
<td>.89</td>
<td>3.18</td>
<td>1.22</td>
<td>4.02</td>
<td>49</td>
<td>&lt;.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTCI Negative Self-Blame</td>
<td>2.43</td>
<td>1.41</td>
<td>2.32</td>
<td>1.44</td>
<td>.18</td>
<td>49</td>
<td>.857</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* N = 51. PTCI = Posttraumatic Cognitions Inventory. PTSD = Posttraumatic Stress Disorder.

Table 8.6

*Correlation coefficients between PTCI appraisals and PTSD*

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 PTCI Negative Self</td>
<td>-</td>
<td>.58**</td>
<td>.28*</td>
<td>.64**</td>
</tr>
<tr>
<td>2 PTCI Negative World</td>
<td>-</td>
<td>.26</td>
<td>.50**</td>
<td></td>
</tr>
<tr>
<td>3 PTCI Negative Self-Blame</td>
<td>-</td>
<td>.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 PTSD Diagnosis</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* N = 51. PTCI = Posttraumatic Cognitions Inventory. PTSD = Posttraumatic Stress Disorder.
Logistic regression analysis was conducted with PTSD as the criterion and negative self and negative world (entered simultaneously) as predictors. Results showed (see Table 8.7) the influence of negative world was not significant whereas the effect negative self, though stronger than negative world (indicated by larger Wald and odds ratio statistics), just failed to reach significance ($p = .054$). The omnibus test of the model coefficients was significant, $\chi^2 (2, N = 51) = 19.57, p < .001$, and the Hosmer-Lemeshow test was not significant, $\chi^2 (8, N = 51) = 5.15, p = .740$, indicating that the model adequately fits the data. In the PTSD group 5/6 were correctly classified and in the non-PTSD group 44/45 were correctly classified, which provided an overall accuracy of 96.1%, a sensitivity of 83.3% and specificity of 97.8%.

Table 8.7

*Results of Logistic Regression Analysis with Negative Self and Negative World Predicting PTSD Diagnosis*

<table>
<thead>
<tr>
<th>Appraisal</th>
<th>Wald</th>
<th>OR</th>
<th>$p$</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTCI Negative World</td>
<td>1.98</td>
<td>2.39</td>
<td>.159</td>
<td>.975 14.76</td>
</tr>
<tr>
<td>PTCI Negative Self</td>
<td>3.70</td>
<td>3.79</td>
<td>.054</td>
<td>.710 8.10</td>
</tr>
</tbody>
</table>

*Note. N = 51. PTCI = Posttraumatic Cognitions Inventory. PTSD = Posttraumatic Stress Disorder. OR = odds ratio.*

In order to ascertain the ability of negative self-appraisals to screen for PTSD, receiver operating characteristic (ROC) plot analysis was conducted (Altman & Bland, 1994). That is, the sensitivity of negative self appraisals were plotted against the false-positive rate.
(1 - specificity). The resulting area under the ROC curve was .91, \( p = .001 \), which indicated that the probability that a random MVA survivor with PTSD had a higher negative appraisal score than a random MVA survivor without PTSD, was 91%. The negative appraisal cut-off score which provided a comparative balance between sensitivity and specificity, was 2.8, which resulted in a sensitivity = .83 and specificity = .91.
8.4 Discussion

The Phase 4 component of the study showed that both negative self and negative world appraisals differentiated participants with and without PTSD. However, negative self appraisal displayed the strongest influence in the prediction of PTSD, which is consistent with the results of previous studies (Beck et al., 2004; Bryant & Guthrie, 2005; Daie-Gabbai, Aderka, Allon-Schindel, Foa, & Gilboa-Schechtman, 2011; Karl et al., 2009; O'Donnell et al., 2004; Startup et al., 2007). This finding provides partial support for the hypothesis proposing that both negative appraisal of self and negative appraisal of the world would predict PTSD. These results provide evidence of the important role post-trauma negative appraisals play in maintaining PTSD, which supports the proposal by Ehlers and Clark (2000) that PTSD is maintained by a sense of current threat, which is described as either external (world) or more commonly as internal (self). One of the processes that lead to the perception of current threat is negative appraisals of the trauma and its sequelae. The ROC analysis showed that negative self appraisal scores performed well in screening for PTSD status among MVA survivors, with a possible cut-off score that provided both high sensitivity and high specificity simultaneously. This finding, though specific to participants who had experienced an MVA, has implications for both researchers and clinicians wishing to identify trauma survivors with probable PTSD without wasting undue resources on false positives or missing many false negatives. The cut-off score of 2.8 may not necessarily be applicable to other trauma populations and hence requires further validation.
The finding that negative self-blame was not associated with the maintenance of PTSD was not completely surprising given the results of previous MVA studies. For example, Karl et al. (2009) found that self-blame did not predict PTSD diagnosis, the study by Beck et al. (2004) showed that negative self-blame did not discriminate between participants with or without PTSD (nor was it associated with litigation), though others have found that self-blame attribution was associated with improved psychological adjustment (Delahanty et al., 1997; Hickling, Blanchard, Buckley, & Taylor, 1999; 1992; O’Donnell et al., 2007).

Though the results of the current study are constrained by the sample size and reduced statistical power\(^1\), it would appear that the effects of self-blame appraisals on the maintenance of PTSD are negligible and may be specific to trauma type (e.g., interpersonal violence). Furthermore, in finding that self-blame was not associated with PTSD, Beck et al. (2004) raised the possibility that participants in their study may not have been responsible for their MVA, and as such, may not have blamed themselves for the accident. Accident causality was not assessed in the current study, so it is unknown what factors may have contributed to the accident and whether participants were at fault. There are several factors that can contribute to the occurrence of an MVA, some of which are controllable, for example, speed, fatigue, and drink-driving, and other factors such as road conditions, weather, and unexpected road obstacles (including animals) which are uncontrollable. It may be beneficial for future MVA studies to consider the role of contributing factors, responsibility, and examine potential differences between drivers/riders and passengers in

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\(^1\) When binary logistic regression is conducted it is recommended that the minimum number of cases per variable is not less than 10 (Hosmer & Stanley, 2000). The minimum number of cases in the current sample is six (number of participants with PTSD) with two predictor variables, which results in a lack of statistical power.
relation to self-blame. Additionally, it may be worthwhile for future studies to consider the
differential role of self-blame sub-categories, characterological and behavioural self-blame,
in the maintenance of PTSD. Startup et al. (2007) suggested that behavioural self-blame
may be associated with a greater sense of controllability which may provide a protective
role against PTSD. The findings by Karl et al (2009) support the suggestions by Startup et
al and highlight the need to consider the effect of self-blame in light of specific sub-
categories.

One of the aims of this study was to describe the incidence of psychopathology and
comorbidity. The current incidence rate of PTSD at approximately 9-months post-accident
of 11.8% is significantly less than that found at Phase 3 (34%), with participants who were
less symptomatic at 6-months being less likely to meet diagnostic criteria at 9-months. The
current rate of PTSD is comparable (though slightly lower) than results reported for other
MVA studies that used a structured clinical interview to assess PTSD. For example,
Blanchard et al. (1997) reported that at approximately 9-months post-MVA, 17.24% of
participants had PTSD, and similarly, Ursano et al. (1999) reported an incidence rate of
17.6%. However, the current rate is considerably lower than that reported by Chan, Air,
and McFarlane (2003) who, using a standardised self-report assessment, found that 29% of
participants had PTSD at 9-months post-MVA.

The incidence rates of Major Depression and Alcohol Abuse Disorder were 9.8% and
11.8% (respectively). However, these results are not directly comparable to the findings
from Phase 3, as the assessment of depression (BDI-II total score ≥ 20, which was
categorised as indicating symptomatic levels depression) and alcohol use (AUDIT total
score \( \geq 8 \) categorised with problematic/hazardous drinking) are based on symptomatology, not diagnosis as such. However, it is interesting to note a consistent reduction, similar to that of PTSD diagnosis, in the rates of depression and alcohol use from 6 to 9-months. It is possible that the reduction in the number of participants with depression and alcohol use problems may be a result of fewer participants (70% recovery rate) who met the criteria for PTSD. Comorbid depression was evident since 33% of participants with PTSD also reported concurrent Major Depression. This rate is somewhat lower than the comorbid rate of 45% found at 6-months post-MVA, as well as being lower than the comorbid rates from the MVA samples reported in section 3.3. It was interesting to note that only 17% of participants \( (n = 1) \) with Alcohol Abuse Disorder (as shown in Figure 8.1) had a coexisting disorder (PTSD and Major Depression). These findings suggest that post-MVA Alcohol Abuse Disorder occurred independently of PTSD. Results indicated that 60% of participants with a single diagnosis of Alcohol Abuse Disorder \( (3/5) \) had increased their alcohol intake (i.e. they were not consuming alcohol at a hazardous rate when assessed 3-months earlier). It is possible that alcohol abuse may mask the symptoms of PTSD, which makes it difficult to determine a direct association. It is also possible that Alcohol Abuse Disorder may have occurred independently of experiencing an MVA.

8.5 Limitations and Summary

One of the limitations of this study was the high attrition rate, which resulted in a greater proportion of participants who utilised task coping strategies. However, there was no evidence of subjective injury severity bias. Participants in this phase of the study also scored lower on initial feelings of sadness and on avoidance coping compared to participants from Phase 3, but there were no age or gender biases which were present in the
earlier study phases. The cross-sectional design limits the conclusions that can be drawn regarding causal relationships between cognitive appraisals and PTSD. Reduced statistical power also confines the conclusion of results and may have limited the detection (and magnitude) of relationships which may have otherwise been found with a larger sample.

In accordance with contemporary cognitive theories of PTSD, this study provided evidence that internally driven negative appraisals of self played a prominent role in maintaining PTSD. The results also showed that for the majority of MVA survivors who had previously met diagnostic criteria for PTSD, symptoms had remitted at 9-months post-accident. From a clinical perspective the reduction in the number of MVA survivors with PTSD is a positive outcome. However, as a result of the small number of participants who still met diagnostic criteria, as well as the reduced overall sample size, additional statistical analysis was not possible. A larger sample would have permitted analysis of the pre, peri, and post-MVA factors associated with PTSD (see Tables 8.2, 8.3, and 8.4 respectively).

It may be useful for future studies to examine the relationships and interactions between dysfunctional cognitive appraisals and other factors associated with the maintenance of PTSD (e.g., physical disability and persistent dissociation). This may provide a more thorough picture into the cognitive, behavioural, and emotional processes that maintain PTSD many months after experiencing an MVA.
Chapter 9

9.1 General Discussion

9.1.1 Summary of Main Findings and Clinical Implications

The overall aim throughout the research presented in this thesis was to examine the influence of, and interaction between, pre, peri, and post-trauma factors in relation to the development and maintenance of PTSD following an MVA.

9.1.2 Phase 1

Evidence from the Phase 1 study showed complex interactions between pre and peri-trauma influences on post-MVA psychological distress at 1-month post-accident. Past emotion problems was a vulnerability factor for acute dissociation and initial psychological distress. Higher levels of exposure (more severe accident and more severe injuries) contributed to psychological distress predominately in the presence of extreme levels of fear responses and subsequent dissociative experiences. Overall, these findings support the theoretical perspective that high levels of fear and dissociation potentiate posttraumatic symptoms through incomplete (data driven) processing and subsequent memory recall and resulting deficits in trauma memories (Ehlers & Clarke, 2000; Foa & Hearst-Ikeda, 1996).

Previous research has shown elements of these relationships. However, this study has extended previous findings by incorporating pre and peritraumatic variables, and accident characteristics in a path analysis model to demonstrate the complex inter-relationships. Furthermore, to the author’s knowledge this study is the first to explore, from a psychological perspective, the role of fatigue in an MVA sample. Results, though
provisional, suggest that participants who are less alert are more likely to experience peritraumatic dissociation. It is possible that a reduced level of alertness may contribute to a state of unreality and possible perceptual distortions (features of dissociation) that may result in deficits in trauma memory. Fatigue has been identified as a contributing factor for MVAs and, if current results can be replicated, may indicate that fatigue is an additional vulnerability marker for peritraumatic dissociative responses.

9.1.3 Phase 2

The Phase 2 study expanded the findings from Phase 1 as it incorporated additional premorbid measures and provided further support for the initial path analysis model with standardised measures of PTSD and depression symptom severity assessed at 3-months post-accident. Results showed two independent pathways predicted peritraumatic dissociation: an exposure/fear pathway (more severe accident as well as extreme fear appraisals) and an interrelated premorbid psychological state pathway (neurotic predisposition, tendency to dissociate and use of [maladaptive] emotion-focused coping strategies). These results suggest that some participants dissociate because of high levels of fear and others because of previous vulnerability factors. In both cases it appears that the cognitive and emotional demands of the situation overwhelm coping resources and, as a result, reduce the ability to fully process trauma information.

This study provided evidence that the Peritraumatic Dissociative Experiences Questionnaire (PDEQ) comprised a single factor structure and a correlated 2 factor structure, both of which had acceptable psychometric properties. The 2 factor structure comprised altered awareness and derealisation, but only altered awareness was predictive of
PTSD and symptom severity. These findings suggest that participants who experience altered awareness (e.g., detachment, confusion, and disorientation) are at greater risk of developing PTSD as the ability to encode trauma information is impeded, which results in disjointed and fragmented trauma memories (Horowitz, 1986; van der Kolk, 1987; van der Kolk & van der Hart, 1989). These findings are inconsistent with the results of the only other study that has examined the facture structure of the PDEQ in relation to PTSD. Bryant et al. (2009) reported a 2 factor structure with similar factor items to that of the current study, however, their initial findings and those at 3-month follow-up (Bryant et al. 2011) showed that derealisation (as opposed to altered awareness) was predictive of PTSD. Differences in trauma type and degree of traumatisation are likely to be reflected in the analysis of dissociative responses and may explain the discrepancy of findings between the two studies. The current results, although they did not replicate those of Bryant et al., provide important additional information which contributes to understanding of the role of acute dissociative responses and subsequent development of PTSD. It is acknowledged that these findings are limited to a specific trauma group and therefore may not generalise to other trauma populations. Future studies that recruit a diverse range of trauma populations (with varying degrees of traumatisation) may provide a more thorough picture in relation to specific acute dissociative responses and their associations with PTSD.

Results from Phase 2 also showed that 33% of participants with PTSD had comorbid alcohol use problems. However, an increase in the use of alcohol since the accident and consumption at hazardous levels was not associated with PTSD, as 66% of participants consumed alcohol at a hazardous level before their MVA. These results suggest that drinking alcohol was not associated with post-MVA adjustment; instead it appears that it is
more indicative of premorbid state. From a clinical perspective it is concerning that a considerable number of participants were consuming alcohol at dangerous levels before their MVA. Given that chronic alcohol abuse and hazardous consumption can contribute to behavioural problems, addiction, and subsequent poor adjustment, it is important from a treatment perspective to assess and monitor the consumption of alcohol. Initial screening and identification of hazardous levels of alcohol use pre and post-trauma may help circumvent ongoing dependence particularly in those who are more vulnerable. Future studies examining the associations between psychotropic substances and post-trauma psychopathology should control for pre-trauma usage and consumption patterns to avoid over-inflated post-trauma incidence rates.

This study provided evidence that physical disability was not only predicted by injury severity as expected, but also independently by psychological state, as initial post-MVA sadness directly affected the perception of disability. In turn, physical disability was found to predict both PTSD and depression symptom severity, which is consistent with the findings of previous MVA studies (Hamanaka et al., 2006; Jenewein et al., 2009; Mayou & Bryant, 2001, 2002; Mayou et al., 1997; O’Donnell et al., 2004). These results illustrate that distressed trauma survivors, independent of injury severity, are more likely to perceive themselves as physically disabled. Physical disabilities can act as constant reminders of the accident and this may impede recovery, and for some MVA survivors may be perceived as a permanent (negative) change, which hinders reappraisal and prevents the accident as being seen as something that happened ‘in the past’.
Path analysis results showed that the influence of past emotional problems was reduced over time, though they did have a direct effect on initial distress at 1-month. However, at 3-months post-MVA, past emotional problems had no direct influence on the severity of PTSD or depression symptoms. Higher levels of exposure (accident severity) contributed to initial emotional distress and ongoing morbidity predominantly in the presence of high levels of fear and subsequent dissociative experiences. These findings demonstrate the complexity of the meditational role of dissociation and, in accord with previous findings, show that exposure (Bryant & Panasetis, 2005; Marmar et al., 1996) and fear of dying (Bryant & Panasetis, 2005; Fikretoglu et al., 2007; Nixon et al., 2002) are associated with acute dissociation. These results provide support for the perspective that intense emotional reactions elicit peritraumatic dissociation.

Peritraumatic variables (fear and dissociation) had direct and indirect effects on post-MVA psychopathology at both 1 and 3-months. Individuals who feared they were going to die and experienced dissociative symptoms during the MVA experienced initial emotional distress. However, at 3-months these variables were only predictive of PTSD symptom severity, findings which are consistent with past research (Blanchard, Hickling, Mitnick et al., 1995; Ehlers et al., 1998; Jeavons et al., 2000; Murray et al., 2002) that has shown these psychological reactions are vulnerability factors for PTSD.

One of the interesting findings was the predominance of the effect (direct and indirect mediated by physical disability) of initial sadness on physical disability as well as depression and PTSD symptom severity. There was an ongoing indirect effect of initial fear on subsequent PTSD symptoms with dissociation contributing to maintenance of
PTSD symptoms. This suggests that reported fear responses one month post-MVA are to be expected and do not indicate a direct vulnerability to ongoing morbidity. A better indicator appears to be reported sadness in the initial aftermath of an MVA and ongoing disability, which reflects a partial shared vulnerability.

Participants’ pre-morbid psychological state was a vulnerability factor for peritraumatic dissociation, initial post-MVA distress, and indirectly for PTSD symptom severity. Severity of the MVA directly influenced an immediate fear response which also increased participants’ likelihood of experiencing peritraumatic dissociation. These results are in accord with Ehlers and Clark’s (2000) cognitive model of PTSD which suggests that background factors as well as trauma characteristics are likely to influence initial emotional reactions, processing (reduced ability to process the trauma conceptually), and trauma appraisals. Furthermore, this study has demonstrated the important mediating role of physical disability appraisal on post-accident psychopathology, which also provides support for Ehlers and Clark’s cognitive model which suggests that negative appraisals, including those associated with physical disability, contribute to a sense of current threat.

9.1.4 Phase 3

Participants’ perception of injury severity, and particularly the degree of initial sadness they experienced continued to influence (direct and indirect mediated by 3-month physical disability) subjective appraisal of physical disability 6-months post-accident. Results showed that an objective medical rating of injury severity was not associated with physical disability. This finding suggests that it is the personal meaning assigned to injury, rather than the actual degree of injury itself, which influences the appraisal of physical disability.
It is possible that an objective rating of injury severity may be more strongly aligned with an objective assessment of physical disability. It may be useful for future studies to incorporate an objective assessment of physical disability as well as a subjective report, to determine if participants’ own appraisal of disability, rather than the actual degree of disability itself, is more influential in the maintenance of PTSD. Additionally, given that the current assessment of physical disability was predominately based on physical activity restrictions, it may be worthwhile for future studies to consider the psychological impact of other types of injury not associated with reduced mobility, such as cosmetic impairments and facial deformities.

Participants with physical disabilities at 3-months were more likely to experience greater PTSD symptom severity (and to a lesser extent depression) 6-months after their accident. These results are consistent with findings of previous MVA studies (Blanchard et al., 1997; Ehlers et al., 1998; Green et al., 1993; Hamanaka et al., 2006; Jenewein et al., 2009; Mayou & Bryant, 2001, 2002; Mayou et al., 1997; O’Donnell et al, 2004), and highlight the important role that appraisal of trauma sequelae has on the maintenance of PTSD. In their cognitive model of PTSD, Ehlers and Clark (2000) explained that negative appraisals of trauma sequelae, including injury and disability, produce a sense of current threat. This, they propose, maintains symptoms by directly producing negative emotions (such as fear, sadness, and anger) that promote dysfunctional coping strategies, which in turn, prevent cognitive change from occurring. Physical disability including scarring and disfigurement as well as perceived permanent change can act as constant reminders of the accident.
Furthermore, ongoing medical treatment, pain, as well as learning to adapt and accept reduced physical functioning, can for some MVA survivors represent significant secondary stressors which may exceed coping recourses. Given the influential role physical disability plays in the maintenance of PTSD, it is important then, from a treatment perspective, that clinicians enquire about and consider the psychological impact of physical disability, which unfortunately for some MVA survivors becomes an integral part of their post-MVA experience. It is also important that clinicians to enquire about claims for compensation given that the legal process of seeking compensation can hinder recovery and exacerbate symptoms. A limitation of this study is that it is not known if participants had sought compensation, and as such, whether or not (and to what extent) this process, particularly for participants with physical disabilities, may have contributed to the maintenance of symptoms.

This phase of the study provided evidence that participants who continued to dissociate after their MVA were at greater risk of chronic PTSD, which is consistent with the results of previous studies (Briere et al., 2005; Halligan et al., 2003; Murray et al., 2002). Persistent dissociation is thought to contribute to the maintenance of PTSD by impeding the access, integration, and resolution of trauma memories (see Ehlers & Clark, 2000; Foa & Hearst-Ikeda, 1996). There was no evidence that trait dissociation or peritraumatic dissociation, in the presence of persistent dissociation, were associated with PTSD or severity of symptoms at 6-months post-accident.

The previous study phases provided evidence that background characteristics; past emotional problems as well as fatigue (directly) in Phase 1, and premorbid neuroticism,
trait emotion-focused coping and trait dissociation (indirectly) in Phase 2, were vulnerability factors for peritraumatic dissociation. Results from Phase 2 also showed that an exposure/fear pathway, independent of background factors, was predictive of peritraumatic dissociation. In turn, peritraumatic dissociation predicted post-accident psychological distress at 1-month as well as PTSD symptom severity at 3-months post-MVA. Collectively, these findings suggest that: participants’ background factors as well as accident characteristics, including emotional response, increase the likelihood of dissociative responses upon trauma exposure; that peritraumatic dissociation plays an important (meditational) role in the development of initial psychological distress as well as acute PTSD; and that participants who continue to dissociate are at greater risk of chronic PTSD. Overall, these findings demonstrate a multifaceted and sequential effect of pre, peri, and post-trauma dissociation associated with initial psychological distress, as well as acute and chronic post-trauma psychopathology.

Participants with PTSD had elevated neuroticism levels at 6-months post-accident. These levels were found to be significantly greater compared to participants without PTSD, but there were no significant group differences on pre-MVA neuroticism levels. Results from Phase 2 showed that a neurotic predisposition was not predictive of PTSD, which is consistent with some previous MVA studies (Conlon et al., 1999; Mayou et al., 1993), but not with others (Dorfel et al., 2008; Holeva & Tarrier, 2001). Neuroticism did however interact with other premorbid factors that predicted peritraumatic dissociation. This result provides partial support for previous findings which found that neuroticism predicted peritraumatic dissociation (Groth-Marnat & Jeffs, 2002; Jaycox et al., 2000). Temporal stability was found to be satisfactory, which indicates that overall, neuroticism remained
relatively stable. The finding that participants with PTSD reported increased neuroticism levels 6-months after their MVA suggests that their neuroticism levels were a consequence rather than a cause of PTSD, which provides evidence of personality change associated with psychopathology (see Clark et al., 1994).

Participants’ consumption of alcohol increased at 6-months, particularly at hazardous levels. Of the participants with PTSD who drank at dangerous levels, 71% had increased their consumption since the MVA, compared to 37.5% at Phase 2 (3-months earlier). Consumption at hazardous levels was associated with intrusions and the use of avoidance coping strategies. This suggests then, that participants’ use of alcohol may have been in response to persistent distressing intrusions, which reflects a form dysfunctional coping (re; self-mediation hypothesis). Avoidance strategies (including the use alcohol), contribute to the maintenance of PTSD by preventing emotional processing of the event and this then interferes with the integration of trauma memories and also the re-appraisal of trauma cognitions (Clohessy & Ehlers, 1999; Dunmore et al., 2001; Ehlers & Clark, 2000; Ehlers et al., 1998; Steil & Ehlers, 2000).

9.1.5 Phase 4

The incidence rate of PTSD at 9-months post-MVA (11.8%) was considerably lower than that at 3 (37.6%) and 6-months (34.5%). At this time, with the exception of participants who experienced greater PTSD symptomatology, most MVA survivors who previously met diagnostic criteria had recovered. Though negative self and negative world appraisals differentiated participants with and without PTSD, participants’ negative self cognitions were most influential in the prediction of PTSD diagnostic status. These results are in
accord with previous findings (Beck et al., 2004; Bryant & Guthrie, 2005; Daie-Gabbai, Aderka, Allon-Schindel, Foa, & Gilboa-Schechtman, 2011; Karl et al., 2009; O’Donnell et al., 2004; Startup et al., 2007), and provide further evidence of the important role post-trauma negative appraisals play in maintaining PTSD. Furthermore, these results support the proposal by Ehlers and Clark (2000) that PTSD is maintained by a sense of current threat, which is described as either external (world) or more commonly as internal (self). One of the processes that lead to the perception of current threat is negative appraisals of the trauma and its sequelae.

Accident causality was not assessed, so it is unknown what factors may have contributed to the accident and whether participants were at fault and blamed themselves for the accident, which perhaps may explain why self-blame negative appraisals were not associated with PTSD. It may be beneficial for future MVA studies to consider the role of contributing factors and examine potential differences in the appraisal of self-blame between drivers/riders and passengers, in relation to post-MVA adjustment. Given the prominent role negative appraisals play in the early phase on symptom onset and in the maintenance of PTSD, it is important for clinicians to identify and address (for example, via cognitive restructuring) erroneous dysfunctional beliefs associated with the trauma and sequelae.

At 3-months post-MVA the consumption of alcohol at hazardous levels was not associated with post-MVA adjustment, instead it was more indicative of participants’ premorbid state. At 6-months drinking patterns had changed, as 71% of participants with PTSD who drank at dangerous levels had increased their consumption since the MVA (compared to 37.5% at 3-months). At 9-months post-accident 11.8% of participants had Alcohol Abuse Disorder,
however, for 83% of these participants this occurred in isolation, independent of PTSD and Major Depression. An increase in alcohol consumption was evident, as 60% of participants with a single diagnosis of Alcohol Abuse had increased their alcohol intake compared to 3-months earlier. It is difficult to provide a clear explanation why an increase in the use of alcohol and associated Alcohol Abuse Disorder was not, for the majority of participants, associated with PTSD (or Major Depression). It is possible that the use of alcohol, particularly at diagnostic levels, may mask the symptoms of PTSD. It is also reasonable to suggest that causal factors associated with participants’ Alcohol Abuse Disorder may not be related to their MVA experience.

9.2 Limitations and Conclusions

The sample was self-selective and though the sample size in the Phase 1 study was sufficient, a low response rate and subsequent age and gender biases (females and slightly older MVA survivors were more likely to volunteer to participate), meant the sample was not representative of the hospital MVA population. However, demographic biases are not completely uncommon within the MVA literature, but perhaps more importantly, in the initial and later phases of this study, there was no evidence of objective injury severity bias (based on ISS comparisons) which is often used an indicator of accident/trauma severity.

The cross-sectional retrospective design in Phase 1 precludes inferences regarding causality, most notably in relation to the conclusions drawn from the path analysis results. The proposed direction of path coefficients was based on previous empirical findings and theoretical considerations, and though the path model provided quite a good fit to the data, it is possible that an alternate model(s) may fit the data equally well. It is acknowledged
that subjective recall of premorbid psychological state, immediate cognitive and emotional
responses, and accident characteristics are retrospective, and as such, may be influenced by
memory bias. The time-frame between MVA and assessment, particularly in relation to
peritraumatic responses, and as well as the effect of post-accident psychopathology, can
also influence the accuracy and reliability of participants’ recollections. Ideally, the time-
frame from accident to initial assessment are best conducted as soon as practicable. It was
anticipated that at 2-weeks after MVA survivors were discharged from hospital they would
be mailed, (by the CNC’s of trauma services) an invitation to participate in the initial study
phase. However, given the varied times between accident and participants’ completion of
the Phase 1 survey, it is possible that not all MVA survivors received their invitation at that
time (some may have been admitted to hospital for an extended period) or that participants
after having received the survey did not complete it straight away.

Additionally, the use of unstandardised pre and post-morbidity measures precludes
conclusions regarding diagnosis. However, the use of standardised premorbid (coping,
personality and dissociation) as well as follow-up measures (PTSD and depression) in the
later study phase provides support for conclusions. Furthermore, the correlations between
the single items used to assess initial psychological distress (feeling anxious or fearful, and
feeling sad or depressed) at 1-month, and the PDS and BDI-II at both 3 and 6-months,
indicated that the simple rating scales share a moderately strong degree of variance with
equivalent standardised measures. Further support was also evident as feeling anxious or
fearful at 1-month was predictive of PTSD symptom severity at 6-months, and similarly,
initially feeling sad or depressed was predictive of depression symptom severity 6-months
post MVA, which further validates the utility of the single item screening measures.
A further limitation was the high attrition rate which was most evident in study Phases 3 and 4. However, in both of these study phases there were no gender or age biases which were present in the earlier study phases. The partial retrospective design in study Phases 2 and 3 (statistical analysis using premorbid measures) limits the conclusions regarding causal relationships. Test re-test results did however indicate a satisfactory level of temporal consistency between variables assessed (retrospectively) at 3-months and again at 6-months post-MVA, which lends support to the stability of the retrospective assessments.

It was anticipated that by having four (potential) hospital recruitment sites a sufficient number of participants in each study phase would be recruited. This would have permitted a thorough investigation of pre, peri, and post-trauma factors associated with PTSD using a path analytic approach for each, and between the four study phases. Unfortunately this was not the case. The small number of participants in the later study phases limited the analysis that could be conducted, confined by a lack of statistical power. It should be noted also, that Bonferroni adjustments were not used, as this procedure to reduce type I error rates inevitably increases the likelihood of type II errors (Perneger, 1998). Ideally

Notwithstanding limitations, the findings from this thesis complement and extend previous research. The results have provided evidence which supports aspects of the cognitive model of PTSD proposed by Ehlers and Clark (2000), and has identified methodological issues that warrant consideration for future research. This thesis incorporated a path analytic approach, which demonstrated the complex and in part, sequential interaction between pre, peri, and post-trauma factors associated with initial post-MVA psychological distress, as well as acute and chronic PTSD. The examination of several pre, peri, and post-
trauma factors, together with the simultaneous analysis with multiple dependent variables has provided a comprehensive and unique examination of the pathways (that included mediation models), associated with post-MVA psychopathology.

Without psychological intervention in the early phase of symptoms onset, unnecessary and preventable chronic psychological suffering will continue. In terms of the psychological needs of MVA survivors much can be done. Raising awareness in the community of the psychological impact of experiencing an MVA and perhaps more importantly within hospital settings, which for many is the first point of contact after experiencing an MVA, would facilitate a greater understanding of the treatment needs of MVA survivors, other than their medical needs. Identifying vulnerabilities that may aid in the procedural screening for those at risk as well as psychological aftercare would reduce much personal suffering, and in the process reduce the long-term psychological costs of experiencing an MVA on an individual and community level.
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appraisals of intrusions, and severity of PTSD symptoms. *Journal of 
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Appendix A: Hunter Area Research Ethics Committee Approval Letter
Hunter Area Research Ethics Committee
Ph: (02) 2421 4950
Ph: (02) 4921 4943
Fax: (02) 4921 4818
Email: nicole.gerrand@hunter.health.nsw.gov.au
       michelle.lane@hunter.health.nsw.gov.au

1 February 2004

Dr R Webster
Department of Psychology
University of Newcastle

Dear Dr Webster,

RE: The Impact of Motor Vehicle Accidents upon Psychological Well Being
(02/12/11/3.31).

The above protocol was approved by the Hunter Area Research Ethics Committee on 17 February 2003. The National Statement on Ethical Conduct in Research Involving Humans requires that an annual report is required for all research protocols that received approval from an Human Research Ethics Committee on the anniversary of that approval. Could you please complete the attached form and return it and any additional documentation (for example a half to one page summary of the progress of the protocol so far) by 28 February 2004.

Yours sincerely,

Ms Michelle Lane
Administrative Officer
Hunter Area Research Ethics Committee
Appendix A1: The University of Newcastle Human Research Ethics Committee Approval Letter
HUMAN RESEARCH ETHICS COMMITTEE

Certificate of Approval
for a research project involving humans

<table>
<thead>
<tr>
<th>Applicant</th>
</tr>
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<tbody>
<tr>
<td>Chief Investigator/Project Supervisor: Dr Rosemary Webster</td>
</tr>
<tr>
<td>Other Investigators: Mr Robert C Hodgson, Professor Mike Startup</td>
</tr>
<tr>
<td>Project Title: The impact of motor vehicle accidents upon psychological well being</td>
</tr>
</tbody>
</table>

In approving this project, the Human Research Ethics Committee (HREC) is of the opinion that the project complies with the provisions contained in the National Statement on Ethical Conduct in Research Involving Humans, 1999, and the requirements within this University relating to human research.

Details of Approval

<table>
<thead>
<tr>
<th>HREC Approval No:</th>
<th>H-317-0203</th>
<th>Date of Approval:</th>
<th>19 February 2003</th>
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<tbody>
<tr>
<td>Approval valid for:</td>
<td>3 years</td>
<td>Progress reports due:</td>
<td>Annually</td>
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</tbody>
</table>

Comments or conditions:

Considered in consultation with the Hunter Area Research Ethics Committee (HAREC). Approval granted 19 February 2003.

Variations to Approved Protocol:

21 July 2004
Variation to:
1. Amend documents to reflect student’s upgrade to a PhD.
2. Include Maitland Hospital as an additional recruitment site.
3. Incorporate a third and fourth phase into the current study:
   (i) The third phase involved a survey at six months post-accident. The survey was identical to the second stage follow-up survey.
   (ii) The fourth phase involved an interview component at six to nine months post accident.
4. Add a follow-up survey reminder.

Approval granted by the Chair on 7 July 2004, subject to amendments to the Information Statements and submission of the interview questions. Ratified.

23 September 2004
Approval confirmed.
Approval covers:
- Invitation Letter – Version 2, dated 20 July 2004
- Participant Consent Form – Version 2, dated 20 July 2004
- Information Sheet (Follow-up study) – Version 2, dated 20 July 2004
- Information Sheet (Interview) – Version 2, dated 20 July 2004
- Contact Details Form (Follow-up) – Version 1, dated 28 June 2004
- Posttraumatic Cognitions Inventory (PTCI)

Signed: Ms Susan O'Connor
Secretary to the Committee
Appendix A2: Central Coast Area Health Service Ethics Committee Approval Letter
Ref: DD: vlr

12 September 2003

Dr Rosemary Webster
The University of Newcastle
School of Behavioural Sciences
University Drive
CALLAGHAN NSW 2308

Dear Dr Webster

Re: 03/54 The Impact of Motor Vehicle Accidents upon Psychological Well Being

At the Ethics Meeting held on 10 September 2003 the above study was reconsidered and approved.

I was nominated as supervisor for the study. The Committee wishes you well with your project.

Yours sincerely

Dr Darrell Duncan
Executive Secretary
Ethics Committee
Appendix A3: The University of Newcastle Human Resource Approval to Conduct Clinical Interviews at Participants Homes
TO: Ms Liz Pilgrim
FROM: Joan Brien, Safety Standards Officer
DATE: August 6, 2004
SUBJECT: Education Application E2/2004 - Webster

This project involves measuring the psychological impact of motor vehicle accidents on victim's well-being. The Study will be carried out by a registered Psychologist and will involve visiting victim's homes and interviewing procedures.

The participants have been described as "representative of a normal community sample" and if, after speaking to the people on the phone, it is deemed inappropriate to do a home visit, the interview will be conducted over the phone.

I am satisfied that the foreseeable risks have been described and control measures put in place, so safety approval is given for the project to go ahead.

DR JOAN BRIEN
Safety Standards Officer
Appendix B: John Hunter Hospital Cover Letter
Dear Sir/Madam

I am the Clinical Nurse Consultant (CNC) of Trauma Services at the John Hunter Hospital (JHH) and am writing to give you the opportunity to contribute to a research project being conducted at the University of Newcastle.

Our records show that you were recently admitted to the JHH following a motor vehicle accident (MVA). Therefore, you may be interested in contributing to this research that explores the psychological effects of experiencing a MVA. Please read the information sheet attached that explains the project.

This project is supported by JHH and I am distributing the survey on behalf of the researchers. YOUR NAME AND CONTACT DETAILS HAVE NOT BEEN RELEASED TO ANYONE.

Whether you choose to participate or not will in no way affect your current or future treatment. If you choose to be involved, please complete the survey, place it in the prepaid envelope provided and return it via Australia Post. Your contribution would be greatly appreciated.

Yours sincerely

Ms Julie Evans
Clinical Nurse Consultant
Trauma Services
John Hunter Hospital
Appendix B1: The Maitland Hospital Cover Letter
Dear Sir/Madam

I am the Clinical Nurse Consultant (CNC) of Trauma Services at the Maitland Hospital and am writing to give you the opportunity to contribute to a research project being conducted at the University of Newcastle.

Our records show that you were recently treated at the Maitland Hospital following a motor vehicle accident (MVA). Therefore, you may be interested in contributing to this research that explores the psychological effects of experiencing a MVA. Please read the information sheet attached that explains the project.

This project is supported by the Maitland Hospital and I am distributing the survey on behalf of the researchers. Your name and contact details have not been released to anyone.

Whether you choose to participate or not will in no way affect your current or future treatment. If you choose to be involved, please complete the survey, place it in the prepaid envelope provided and return it via Australia Post. Your contribution would be greatly appreciated.

Yours sincerely

Ms Catherine McDonald CNC
Appendix B2: Phase 1 Information Sheet
The Impact of Motor Vehicle Accidents upon Psychological Well-Being

Information Sheet

You are invited to take part in this research. My name is Robert Hodgson and I am a Psychology PhD student at the University of Newcastle. This research is being conducted under the supervision of Dr Rosemary Webster and Professor Mike Startup as part of the requirements for my degree. This study examines the effects of motor vehicle accidents on psychological well-being. The purpose of this research is to gain a better understanding of the factors that assist individuals to overcome the psychological impact of their accident.

We are seeking people over the age of 18 years who have been involved in a motor vehicle accident and have received treatment at the John Hunter Hospital to participate in this research. You may have been either a driver or passenger of a car or truck, a rider or pillion passenger of a motorcycle, a pedal cyclist, or a pedestrian. The attached survey asks about your experience of being involved in a motor vehicle accident. We are interested in what factors contribute to psychological well-being following a motor vehicle accident such as emotional state before and after the accident. It will take approximately 10 minutes to complete. The information collected will be used in a thesis to be submitted for my degree. It will also be reported in scientific journals and at conferences.

- Participation in this study is voluntary. Your results will be treated respectfully and with confidentiality. Individuals will not be identifiable in any reports resulting from this study.
- You may withdraw from this study at any time and do not have to give any reason for withdrawing. If you decide to withdraw from the study, you have the option of withdrawing all data relating to you.
- Return of the survey will be considered as consent to participate in this study.
- Surveys will be stored for 7 years in a locked filing cabinet at the University of Newcastle and then destroyed. Only the researchers involved in this study will have access to the data collected. The data will not be released to anyone else except if required by law.
- Answering these questions may prompt memories of the accident and if you would like to talk about your experience, you may wish to contact Lifeline on 131114, the Psychology Clinic on 02 49215975 or your local community health service.
- We will be conducting a follow-up study. If you would like to participate in the follow-up study please complete the enclosed contact form. If you would like a copy of the results of this study please tick the appropriate box on the contact form.

If you decide to complete the survey, please place it in the prepaid envelope provided, seal it, and return it via Australia Post.

Please keep this information sheet for future reference.
Enquires about this study may be directed to Dr Rosemary Webster, School of Behavioural Science, Psychology, University of Newcastle on 02 49 215975 or Email: rosemary.webster@newcastle.edu.au

Your participation in this study would be greatly appreciated and we sincerely thank you for your time and interest.

Robert Hodgson                          Rosemary Webster

This project has been approved by the University's Human Research Ethics Committee, Approval No. H- 517-0203
The University requires that all participants are informed that if they have any complaints concerning the manner in which a research project is conducted it may be given to the researcher, or if an independent person is preferred, to the University's Human Research Ethics Officer, Research Branch, Chancellery, University of Newcastle, 2308, telephone 4921 6333.

This project has been approved by the Hunter Area Research Ethics Committee of Hunter Health, Reference 02/12/11/3.31
Should you have concerns about your rights as a participant in this research, or you have a complaint about the manner in which the research is conducted, it may be given to the researcher, or, if an independent person is preferred, to Dr Nicole Gerrand, Professional Officer, Hunter Area Research Ethics Committee, Hunter Health, Locked Bag 1, New Lambton NSW 2305, telephone (02) 49214950, email Nicole.Gerrand@hunter.health.nsw.gov.au
Appendix B3: Results Summary Request and Expression of Interest for Follow-up Study
The Impact of Motor Vehicle Accidents upon Psychological Well-Being
Expression of Interest regarding a Follow-up Study

Contact Details Form

Dear participant,

Thank you for taking the time to complete the survey. If you would like to participate in a follow-up study, that involves completing another survey that will be mailed to your postal address in 3 months time, please tick the appropriate box. An information sheet about the follow-up study is attached. To ensure your privacy, a code number has been allocated which will allow us to link the two surveys.

The results of the survey will be available in approximately 18 months – 2 years time. If you would like a copy of the results please tick the appropriate box.

☐ Yes, I would like to be informed about a follow-up study.

☐ Yes, I would like a summary of the results posted to me.

Your name: __________________________________________________________

Postal address: _______________________________________________________

Email address: ________________________________________________________

Thank you very much for your interest and contribution.
Appendix B4: Phase 1 Survey
Dear Participant,

Thank you for volunteering to be in this study. This questionnaire will take approximately 10 minutes to complete. Your responses are strictly confidential.

Please answer every question that applies to you by placing a TICK in the appropriate box or writing your response on the LINE provided, or by CIRCLING the most appropriate answer.

1. Today’s date_____/_____/_____
   Day       Month      Year

2. Date of the Accident_____/_____/_____
   Day         Month          Year

3. What time did the accident occur? _____________am/pm

4. When the Accident happened, were you a:
   Driver  [ ]  Passenger  [ ]  Motorcyclist  [ ]  Pillion Passenger on a Motorcycle  [ ]
   Pedal Cyclist  [ ]  Pedestrian  [ ]

5. Please draw a circle around the number that best describes the overall Severity of the Accident.

   1  2  3  4  5
   Very Slight  Moderate  Serious  Severe  Very Severe

6. Were you Physically Injured in the accident? Yes  [ ]  No  [ ]

7. Please draw a circle around the number that best describes the Severity of your injury/injuries.

   1  2  3  4  5
   None  Mild  Moderate  Serious  Life Threatening

8. Please draw a circle around the number that best describes to what extent you feared for your life during the accident.

   1  2  3  4  5
   Not at all  A little  Somewhat  A lot  Certain I would Die
9. **At the moment** how sad or depressed do you feel?

   1                         2                         3                         4                         5  
   Not at all              A little              Somewhat              A lot              Extremely  

10. **At the moment** how anxious or fearful are you?

   1                         2                         3                         4                         5  
   Not at all              A little              Somewhat              A lot              Extremely  

11. Please draw a circle around the most appropriate answer to the following statement: “During the 4 weeks **before the accident occurred** how much were you bothered by emotional problems (such as feeling depressed or anxious)?”

   1                         2                         3                         4                         5  
   Not at all              A little              Somewhat              A lot              Extremely  

12. Have you had **any other** Motor Vehicle Accidents? No □ (If no, go to Question 14)  
   Yes □ How Many □  

13. How would rate this accident compared to the other accidents you have experienced?

   Not as bad □    The same □    Worse □  

14. **Immediately prior to the accident** how would you describe your level of alertness?

   Felt active, wide-awake.  
   Was functioning at a high level but not at peak.  
   Felt relaxed, awake but not fully alert, responsive.  
   Felt a little foggy headed.  
   Felt foggy headed, had difficulty staying awake, was beginning to lose track.  
   Felt sleepy, would have preferred to lie down, woozy.  
   Could not stay awake, sleep onset was imminent.
15. Please answer all the statements below by ticking the choice that best describes your experiences and reactions **during the accident and immediately afterward**. If a statement does not apply to your experience, please tick “Not at all true.”

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Not at all true</th>
<th>Slightly true</th>
<th>Somewhat true</th>
<th>Very true</th>
<th>Extremely true</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I had moments of losing track of what was going on – I “blanked out” or “spaced out” or in some way felt that I was not part of what was going on.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>I found that I was on “automatic pilot” – I ended up doing things that I later realised I hadn’t actively decided to do.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>My sense of time changed – things seemed to be happening in slow motion.</td>
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</tr>
<tr>
<td>4</td>
<td>What was happening seemed unreal to me, like I was in a dream or watching a movie or play.</td>
<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>5</td>
<td>I felt as though I were a spectator watching what was happening to me, as if I were floating above the scene or observing it as an outsider.</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>6</td>
<td>There were moments when my sense of my own body seemed distorted or changed. I felt disconnected from my own body, or that it was unusually large or small.</td>
<td></td>
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<tr>
<td>7</td>
<td>I felt as though things that were actually happening to others were happening to me – like I was trapped when I really wasn’t.</td>
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<tr>
<td>8</td>
<td>I was surprised to find out afterward that a lot of things had happened at the time that I was not aware of, especially things I ordinarily would have noticed.</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>9</td>
<td>I felt confused; that is, there were moments when I had difficulty making sense of what was happening.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>I felt disoriented; that is, there were moments when I felt uncertain about where I was or what time it was.</td>
<td></td>
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</tbody>
</table>

16. Briefly describe the accident:

_________________________________________________________________________________
_________________________________________________________________________________
_________________________________________________________________________________
The following questions are about your background details.

17. Male [ ] Female [ ]

18. Date of Birth _______ / _______ / _______
   Day       Month       Year

19. Please tick One of the Boxes below which corresponds to your highest level of Education.

   Some Secondary [ ] Completed Year 12 [ ]
   (6th form)       T.A.F.E or College [ ]
   University [ ]

20. Please Tick One Box, which corresponds to your Average Yearly Income Before Tax.

   Less than $15,000 [ ] Between $15,000 and $30,000 [ ]
   Between $30,000 and $50,000 [ ] More than $50,000 [ ]

Thank you very much for your contribution.

Please fold the survey and return in the envelope provided.
If you have misplaced the envelope please return the survey to Dr Rosemary Webster, School of
Behavioural Sciences, Psychology, University of Newcastle, Callaghan, NSW, 2308.

If you want to be included in the follow-up study please complete the Contact Details Form and return it in the envelope provided.
Appendix C: Phase 2 Information Sheet
The Impact of Motor Vehicle Accidents upon Psychological Well-Being: Follow up Study

Information Sheet

You are invited to take part in this follow up study. My name is Robert Hodgson and I am a Psychology PhD student at the University of Newcastle. This research is being conducted under the supervision of Dr Rosemary Webster and Professor Mike Startup as part of the requirements for my degree. This study examines the effects of motor vehicle accidents on psychological well-being. The purpose of this research is to gain a better understanding of the factors that assist individuals to overcome the psychological impact of their accident.

The survey you previously completed examined the immediate effects of motor vehicle accidents and this follow-up study examines the longer-term effects on psychological well-being. The attached survey asks about attitudes and behaviours before the accident as well as attitudes and behaviours after the accident and how you are feeling at the moment. It will take approximately 30 minutes to complete. The information collected will be used in a thesis to be submitted for my degree. It will also be reported in scientific journals and at conferences.

- Participation in this study is voluntary. Your responses will be treated respectfully and with confidentiality. Individuals will not be identifiable in any reports resulting from this study. Please do not write your name on any of the surveys. We have used a code to link your survey responses and to ensure anonymity.
- Return of the survey will be considered as consent to participate in this study.
- You may withdraw from this study at any time and do not have to give any reason for withdrawing. If you decide to withdraw from the study, you have the option of withdrawing all data relating to you.
- Surveys will be stored for 7 years in a locked filing cabinet at the University of Newcastle and then destroyed. Only the researchers involved in this study will have access to the data collected. The data will not be released to anyone else except if required by law.
- Answering these questions may prompt memories of the accident and if you would like to talk about your experience, you may wish to contact Lifeline on 131114, the Psychology Clinic on 49215075 or your local community health service.
- If you would like a copy of the results of this follow-up study please tick the appropriate box on the enclosed contact form.

If you decide to complete the survey, return it and the contact form in the prepaid envelope provided.

Please keep this information sheet for future reference.
Appendix C1: Phase 2 Survey
Dr Rosemary Webster, School of Behavioural Sciences, Psychology, University of Newcastle

The Impact of Motor Vehicle Accidents upon Psychological Well-Being: Follow-up Study

Dear Participant,

Thank you for volunteering to be in the follow-up study. This survey will take approximately 30 minutes to complete. Your responses are strictly confidential. A code is provided to link your responses from the previous survey with this survey. Do not write your name on any pages.

Please answer every question by placing a **TICK** in the appropriate box or by **CIRCLING** the most appropriate answer.

1. When answering the following questions please recall your experience of the accident. Please tick each item, indicating how frequent these comments have been true for you during the last week. If they did not occur, please tick the “Not at all” column.

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>Rarely</th>
<th>Sometimes</th>
<th>Often</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I thought about it when I didn’t mean to.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>I avoided letting myself get upset when I thought about it or was reminded of it.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>I tried to remove it from my memory.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>I had waves of strong feelings about it.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>I tried not to talk about it.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Pictures about it popped into my mind.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>I tried not to think about it.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Any reminder brought back feelings about it.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. Did you receive ANY Treatment following the Accident? Yes □ No □ (If No, go to Question 4)

   Was this for: Emotional Problems □ Physical Problems □ Both □

3. In Total How Long did you receive Treatment? One Day □ Up to One Week □

   Up to One Month □ Between One and Three Months □ More than Three Months □
4. Overall, how well do you feel you have recovered from the accident?

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fully recovered</td>
<td>Almost fully recovered</td>
<td>Half recovered</td>
<td>Partially recovered</td>
<td>Not at all</td>
</tr>
</tbody>
</table>

5. During the last month have you been limited in any of the following activities? If so, how much? Please answer all the questions by placing a TICK in response to each question.

<table>
<thead>
<tr>
<th>No, not at all</th>
<th>Yes, sometimes or a little</th>
<th>Yes, moderately or definitely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have your health problems limited you in any of the following activities?</td>
<td>a. The type or amount of vigorous activity you can do, e.g., lifting objects, running or sports.</td>
<td>b. The type or amount of moderate activity you can do, e.g., moving a table, carrying groceries or goods.</td>
</tr>
<tr>
<td>c. Climbing stairs or walking uphill.</td>
<td>d. Bending, lifting or stooping.</td>
<td>e. Walking long distances (i.e., 1 - 2 km).</td>
</tr>
<tr>
<td>f. Eating, dressing, bathing, or using the toilet.</td>
<td>Have you had to cut down or stop any activity you used to do, such as hobbies, because of some illness or injury?</td>
<td>Have you not been able to do something that your family (or household) expected from you as part of daily routine?</td>
</tr>
<tr>
<td>Have your personal problems decreased your motivation for work?</td>
<td>Have your personal problems decreased your personal efficiency at home, school or work?</td>
<td>Has there been a deterioration in your social relations with friends, workmates or other persons?</td>
</tr>
</tbody>
</table>
Appendix D: Participant Thank You/Reminder Letter
The Impact of Motor Vehicle Accidents upon Psychological Well-Being

Participant Thank You/Reminder Letter

Dear Sir/Madam

Thank you very much for volunteering to participate in our research project examining the psychological effects of experiencing a motor vehicle accident. This letter is being sent to participants who have agreed to participate in the follow-up study. If you have returned your survey, thank you so much for your time and effort, we really appreciate it.

If you have not yet returned it there is still time to do so. If you have misplaced your survey, please telephone the Psychology Clinic on 49 217753, and leave your name on the answering machine. We will then mail you another one that you may complete and return in the pre-paid envelope provided. Your contribution to this study is important and valued and we thank you for your time and interest.

If you choose not to complete the survey, please return it in the envelope provided.

Sincerest regards

Robert Hodgson

Rosemary Webster
Appendix E: Phase 3 and Phase 4 Invitation Letter
The Impact of Motor Vehicle Accidents upon Psychological Well-Being

Invitation Letter

Dear Participant,

Thank you very much for your interest and contribution to our research project. I have recently transferred from the Master of Clinical Psychology to a PhD in Psychology and have extended the study to include a second follow-up survey and interview. We are interested in your experience of your motor vehicle accident and how it may have affected you initially and in the longer term.

We are sending you this letter so that you may have the opportunity to be part of the ongoing study and would like to invite you to participate in the second follow-up survey and the interview. **If you choose to participate in the interview you will be reimbursed for traveling expenses.** Please see the attached Information Sheets for details.

We appreciate the time and effort you have taken to support our research and we would be grateful should you choose to participate further. If you would like to participate in the second follow-up survey and/or the interview please complete the **Participant Consent Form**, and return it in the prepaid envelope provided.

Yours sincerely

Robert Hodgson
Research Student

Rosemary Webster
Research Supervisor

The UNIVERSITY of NEWCASTLE
AUSTRALIA
Appendix E1: Phase 3 Information Sheet
The Impact of Motor Vehicle Accidents upon Psychological Well-Being

Information Sheet - Follow up Study 2

You are invited to take part in this follow up study. My name is Robert Hodgson and I am a Psychology PhD student at the University of Newcastle. This research is being conducted under the supervision of Dr Rosemary Webster and Professor Mike Startup as part of the requirements for my degree. This study examines the effects of motor vehicle accidents on psychological well-being. The purpose of this research is to gain a better understanding of the factors that assist individuals to overcome the psychological impact of their accident.

The survey you previously completed examined the short-term effects of experiencing a motor vehicle accident and this second follow-up study examines the longer-term effects on psychological well-being. The attached survey asks about attitudes and behaviours after the accident and how you are feeling at the moment. It will take approximately 30 minutes to complete. The information collected will be used in a thesis to be submitted for my degree. It will also be reported in scientific journals and at conferences.

- Participation in this study is voluntary. Your responses will be treated respectfully and with confidentiality. Individuals will not be identifiable in any reports resulting from this study. Please do not write your name on any of the surveys. We have used a code to link your survey responses and to ensure anonymity.
- Return of the survey will be considered as consent to participate in the follow-up study 2.
- You may withdraw from this study at any time and do not have to give any reason for withdrawing. If you decide to withdraw from the study, you have the option of withdrawing all data relating to you.
- Surveys will be stored for 7 years in a locked filing cabinet at the University of Newcastle and then destroyed. Only the researchers involved in this study will have access to the data collected. The data will not be released to anyone else except if required by law.
- Answering these questions may prompt memories of the accident and if you would like to talk about your experience, you may wish to contact Lifeline on 131114, the Psychology Clinic on 49 215075 or your local community health service.
- If you would like a copy of the results of the second follow-up study please tick the appropriate box on the enclosed contact form.

If you decide to complete the survey, return it and the contact form in the prepaid envelope provided.

Please keep this information sheet for future reference.
Appendix E2: Phase 4 Information Sheet
Dr Rosemary Webster
Lecturer
School of Behavioural Sciences
Discipline of Psychology
University Drive, Callaghan
NSW 2308 Australia
Telephone: + 61 2 49215975
Facsimile: + 61 2 49216560
Email: Rosemary.Webster@newcastle.edu.au

The Impact of Motor Vehicle Accidents upon Psychological Well-Being

Information Sheet - Interview

You are invited to take part in the Interview. My name is Robert Hodgson and I am a Psychology PhD student at the University of Newcastle. This research is being conducted under the supervision of Dr Rosemary Webster and Professor Mike Startup as part of the requirements for my degree. This study examines the effects of motor vehicle accidents on psychological well-being. The purpose of this research is to gain a better understanding of the factors that assist individuals to overcome the psychological impact of their accident.

The interview will be conducted either in person or over the telephone. You will be asked to respond to a range of questions, some will be about the accident and others will be about how you currently feel, think and behave in general. You will also be asked about your thoughts following the accident. The interview will take approximately 1 hour to complete.

If you wish to participate you may choose to have the Interview conducted at the University of Newcastle Psychology Clinic or in your home. Alternatively, if you wish to participate, but do not want the Interview conducted in person, or if you live outside of the Newcastle/Hunter Valley area, you may choose to have the Interview conducted over the telephone. The information collected will be used in a thesis to be submitted for my degree. It will also be reported in scientific journals and at conferences.

• Participation in this study is voluntary. Your responses will be treated respectfully and with confidentiality. Individuals will not be identifiable in any reports resulting from this study.
• Return of the Participant Consent Form with the Interview box ticked will be considered consent to participate in the Interview.
• You may withdraw from this study at any time and do not have to give any reason for withdrawing. If you decide to withdraw from the study, you have the option of withdrawing all data relating to you.
• Only the researchers involved in this study will have access to the data collected, which will be identified by code not name. Information from the Interview will be securely stored for 7 years at the University of Newcastle and then destroyed. The data will not be released to anyone else except if required by law.

Answering Interview questions may prompt memories of the accident. If you would like further support to deal with your experience, you may wish to contact Lifeline on 131114, the Psychology Clinic on 49 215075 or your local community health service.

If you would like to participate in the Interview please tick the appropriate box on the Participant Consent Form, and return it the prepaid envelope provided.

Please keep this information sheet for future reference.
Appendix E3: Participant Consent Form
The Impact of Motor Vehicle Accidents upon Psychological Well-Being
Participant Consent Form

Dear participant,

Your participation in the earlier phases of this research project places you under no obligation for further involvement. However, if you would like the opportunity to participate in a second follow-up survey, that involves completing another survey that will be mailed to your postal address approximately 6 months after your accident, please tick the appropriate box.

If you would like the opportunity to participate in the interview, which will be conducted at a time and place suitable to you, approximately 6 to 9 months after your accident, please tick the appropriate box. You will be reimbursed for travel expenses. If you would like to participate in both the second follow-up survey and the interview please tick both boxes.

If you wish to participate in the interview please tick one of the 3 smaller boxes to indicate where you would like the interview to be conducted. Also, please provide a telephone contact number so that I may contact you to arrange a suitable time to conduct the interview.

☐ Yes, I would like to participate in the second follow-up survey.

☐ Yes, I would like to participate in the interview (Please tick one box below).

☐ I would like the interview to be conducted at the Psychology Clinic.

☐ I would like the interview to be conducted at my home.

☐ I would like the interview to be conducted over the telephone.

Your name: ______________________________________

Postal address: ______________________________________

Telephone No: (H) ______________________ (W) ______________

Mobile ______________________

Please indicate the most convenient time to contact you __________________ am/pm

Thank you very much for your interest and contribution.