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**BENJAMIN J. SMITH**

**DOCTORAL THESIS**

**PREVALENCE, AETIOLOGY AND MAINTENANCE  
OF POOR PSYCHOLOGICAL MORBIDITY  
FOLLOWING A MINOR ROAD TRAFFIC ACCIDENT:  
A PROSPECTIVE LONGITUDINAL STUDY**

**UNIVERSITY COLLEGE LONDON  
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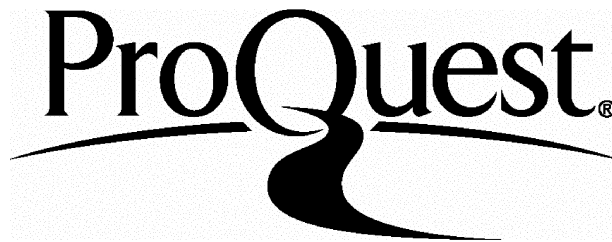
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## **ABSTRACT**

The current study aimed to investigate the prevalence, aetiology and maintenance of poor psychological morbidity following a minor road traffic accident (RTA). A prospective longitudinal research design was employed and participants completed assessments within one month of their RTA and three months later. It was anticipated that, in accordance with published empirical evidence, participants would report clinically significant levels of anxiety, depression and Post-Traumatic Stress Disorder (PTSD). Informed by recent cognitive conceptualisations of PTSD (e.g. Ehlers and Clark, 2000; Brewin et al., 1996) it was hypothesised that a number of psychological factors would predict and maintain PTSD.

It was found that in this sample of minor-RTA victims clinically significant levels of anxiety, depression and PTSD were present. Further examination revealed that PTSD could be significantly predicted by a number of independent variables. Anxiety sensitivity, immediate post-traumatic reaction and peri-traumatic dissociation were all found to predict PTSD. Negative interpretation of symptoms, rumination and thought suppression (taken together) were found to heavily mediate the relationships of all these predictive factors with follow-up PTSD. These maintenance factors were the only variables to independently and significantly predict follow-up PTSD. The results reinforce the importance of both negative attribution and avoidant coping in the persistence of PTSD and a number of clinical and theoretical implications are discussed.

## **INTRODUCTION**

### **1.1 Statement of Intention**

The introduction chapter aims to place the current research in empirical and theoretical context. First, RTAs are discussed as a potential cause of psychological trauma and diagnostic and phenomenological descriptions of traumatic sequelae are outlined (e.g. PTSD). Second, the empirical evidence regarding prevalence of psychological disorder post-RTA is described, revealing that much confusion remains concerning the natural history of many post-traumatic disorders. Third, in order to properly address the issues of prediction and maintenance of PTSD, behavioural and cognitive conceptualisations are outlined. In the context of this theory empirical evidence is described for a number of variables regarding their potential role in the development of psychological disorder post-RTA. Both environmental and psychological variables are addressed. Finally, the principal aims of the current study are re-iterated, research questions posed and hypotheses outlined.

### **1.2 Road Traffic Accidents**

Road Traffic Accidents (RTA) are an integral part of everyday life, and despite being geographically dispersed and often small in size, they are large in totality (Digallo and Parry-Jones, 1996). An RTA can lead to physical injury, mental and emotional trauma, and social disruption. The World Health Organisation (WHO) has predicted that by the year 2020 the RTA will have moved from a rank of nine to a rank of three, of leading causes of disability resulting from disease or injury

(Bland, 1996). Between 1993 and 1995 over 10,000 individuals were killed on British roads, and over 750,000 injured (Mitchell, 1997). According to Norris (1992) the lifetime frequency of being involved in an RTA is 23% in the USA.

The statistics above are important, and yet their presentation to society is rare. Horne (1999) suggests that the RTA is treated differently from other disasters. When an individual gets into a car there is little or no expectation of being involved in a serious accident, so when it occurs it is truly a shock. Paradoxically, because such accidents are so commonplace and because they normally involve small numbers of people, the societal response is much less well co-ordinated. Mitchell (1997) notes that the manner in which traffic news pertaining to an RTA is presented, differs from reporting of other negative events. Mitchell proposes that bulletins are light and friendly with the purpose of keeping traffic moving and to prevent further accidents due to congestion. It is illuminating to reflect on how society would react to some other aspect of everyday technology causing so much harm; if the telephone killed ten individuals a day, and injured a further 500 (British road injury rate, 1994), surely it would be banned (Mitchell, 1997).

Over the last decade greater attention has been paid to the social and psychological sequelae of the RTA. However, the impact of the day-to-day societal view of the RTA continues to dwarf research in this area when compared to other large scale disasters (Norris, 1992). It has been proposed by many authors (e.g. Mitchell, 1997; Norris, 1992; Di-gallo and Parry-Jones, 1996; Horne, 1999) that the impact in the long-term for many RTA survivors has been greatly underestimated. Many

RTA victims do not have their emotional and psychological reactions attended to, or measured in hospital. Horne (1999) proposes that RTA induced depression, anxiety and post-traumatic reactions go undetected and untreated, largely due to the different societal response when compared to other disastrous events.

### 1.3 Psychological Trauma

Brewin et al. (1996), define trauma as any experience that has threatened the health or well being of an individual. Trauma may involve events that are within or outside the normal range of human experience. Traumatic events often indicate the world is uncontrollable and unpredictable, for example physical or sexual assault, social humiliation, transgression of one's own moral code, disaster, accident or illness. Traumatic events may violate pre-conceived assumptions about the world, others and the self, or in some cases confirm negative assumptions. The effects of psychological trauma differ widely depending on the traumatic event, and on an individual's reaction to it. Post-Traumatic Stress Disorder is by far the most researched sequelae of psychological trauma. The development of the PTSD construct is briefly described below in order to inform discussions of the prevalence, prediction and maintenance of PTSD post-RTA, a major focus of this study.

### 1.4 Post-Traumatic Stress Disorder

The symptoms of Post-traumatic Stress Disorder (PTSD) have been described since the inception of war, but only in recent years has attention been paid to post-traumatic symptoms during peace time. Only over the past century have clusterings of trauma associated symptoms been isolated, named and broadened

phenomenologically (Tomb, 1994). Following World War I, the American Psychiatric Association initiated the formal classification of reactions to trauma by creation of the category “Gross Stress Reaction”, in the Diagnostic and Statistical Manual - Volume I, (DSM-I). Gross Stress Reaction was defined as “a reaction to severe combat or civilian catastrophe that may progress to one of the neurotic reactions.... If the reaction persists”. DSM-II minimised reactions to trauma, reducing them to a brief ‘Transient Situational Disturbance’. It is clear from these diagnostic descriptions that post-traumatic reactions were seen as a form of malingering, or the expression of another psycho-pathological disorder.

PTSD first received official recognition in the third edition of DSM (DSM-III: APA, 1980), in which it was placed among the anxiety disorders, following extensive work by Horowitz (1976) in the civilian population. The DSM-III definition of PTSD in 1980 aimed to create for the first time a specific Post-Traumatic Stress Disorder describing a consistent pattern of symptoms, following exposure to trauma of all types. To make the diagnosis DSM-III and DSM-III-R (APA, 1987) after it, demanded that there be a severe stressor ‘outside the range of usual human experience’ which generated a triad of i) intrusive re-experiencing of events, ii) avoidance responses to evidence of the trauma or generalised psychological numbing and isolation, and iii) widespread physiologic arousal not previously present.

Although DSM-IV (APA, 1994), maintains the triad of diagnostic symptoms groups from DSM-III, it differs from earlier conceptualisations of PTSD in several

important ways. DSM-IV shifted from a primary focus on the severity of the stressor to a mixture of exposure to a traumatic stressor, coupled with an individual's reaction to it. This reflects a predominant view in the literature that the perception of threat or trauma is as important as the 'objective' severity of the stressor. As shall be discussed below and addressed empirically in this study, evidence for the role of psychological risk factors in the development of PTSD following minor traumatic events is developing. Tomb (1994) notes that there is a growing body of evidence indicating a significant role for an individual's interpretation of events that seem objectively 'trivial'.

The full DSM-IV criteria for PTSD are as follows: i) An individual must have been exposed to a traumatic event in which they experienced, witnessed or were confronted with an event that involved actual or threatened death, serious injury or a threat to physical integrity; ii) An individual must also have had a response which involved 'intense fear, helplessness or horror'; iii) An individual must experience symptoms including re-experiencing of the traumatic event, whether through dreams, vivid visual flashbacks or in reaction to reminding cues, (internal or external); iv) Individuals must experience at least two of persistent arousal, anger, sleeplessness, concentration problems, hypervigilance or an exaggerated startle response; v) Finally, individuals will show persistent avoidance (cognitive or behavioural) in relation to activities or places associated with the trauma, and a numbing of general responsiveness.



In accordance with DSM-III-R, DSM-IV continues to require symptoms to be present for 'more than one month'. PTSD has also been divided into two sub-types; acute and chronic. Acute PTSD requires a symptom duration of at least one month but fewer than three months, whereas chronic PTSD requires at least three months of sufficient symptoms. Delayed onset PTSD is diagnosed if the onset of symptoms is at least six months after the traumatic event.

Horowitz (1976) and Brewin et al. (1996), note that PTSD is often characterised by an alteration between re-experiencing and avoiding trauma related memories. Memories appear rapidly, spontaneously and intrusively, and are emotionally intense. Tomb (1994) suggests that avoidance symptoms are the result of an individual's attempt to escape traumatic memories. PTSD sufferers often experience many secondary negative emotions such as sadness, anger, guilt and shame. Taylor (1999) reports the results of a factor analysis of the PTSD construct with over 500 individuals, all of whom had been exposed to a traumatic event satisfying DSM-IV criteria. Taylor found PTSD to consist of two correlated factors: i) intrusion and avoidance, and ii) hyperarousal and numbing. These two factors loaded successfully onto a higher order factor of PTSD, which itself accounted for between 13 and 38% of the variance in symptom severity. Taylor (1999) concludes that PTSD arises from a general mechanism, with contributions from two specific mechanisms.

### 1.5 Acute Stress Disorder

The core phenomenon of high levels of distress during the acute trauma phase has been formalised in DSM-IV, with the inclusion of a new diagnostic label, Acute Stress Disorder (ASD). ASD exists as an attempt to describe and define individual pathological reactions to trauma in the first month. Diagnostic criteria are as follows: i) Identical stressor (trauma) criterion as PTSD; ii) Individuals must experience at least one of the re-experiencing phenomena of PTSD; iii) Individuals must experience marked avoidance of trauma related stimuli; iv) Individuals must experience marked hyperarousal as described in PTSD; v) Either whilst experiencing or after experiencing the distressing event, an individual has three (or more) of the following dissociative symptoms: subjective sense of numbing, detachment or absence of emotional responsiveness; a reduction in awareness of surroundings; derealisation; depersonalisation; dissociative amnesia; and finally vi) Individuals experience the symptoms over a period of at least two days and not more than four weeks, within one month of the traumatic event.

ASD during the first month post-trauma can appear identical to the PTSD that occurs after one month. The exception is that in order to gain a diagnosis of ASD, an individual must exhibit dissociative symptoms which are not required for a diagnosis of PTSD. A key rationale for the inclusion of the ASD diagnosis in DSM-IV is the assumption that ASD is a predictor of PTSD. It is thought that the ASD diagnosis may help to predict those who are at risk of chronic PTSD, and for whom an early intervention may reduce long term psychopathology. The predictive

power of ASD is currently being tested empirically (described in detail below and addressed in this study).

There is widespread recognition of co-morbidity associated with PTSD/ASD. There is often a large overlap of symptoms, particularly phobic avoidance and physiological arousal (anxiety disorders) and psychogenic numbing (depression). It is well recognised that the traumas that lead to PTSD and ASD, and the enduring symptoms of these disorders, place an individual at risk of developing related but distinct psychiatric disorders such as Major Depressive Disorder, Agoraphobia and Simple Phobias. In the following section of this volume, the evidence for the prevalence of these psychological sequelae will be examined, with specific reference to the RTA.

## 1.6 The Psychological Consequences of an RTA: Empirical Evidence

### Post-Traumatic Stress Reactions

The prevalence and natural history of post-traumatic stress reactions post-RTA are unclear. Many studies investigating the prevalence of PTSD following an RTA have been confounded, as they have studied patients seeking either compensation (e.g. Culpan and Taylor, 1974) or psychological assistance (e.g. Hickling and Blanchard, 1992). Unsurprisingly, these studies range in their estimates of the occurrence of PTSD from nearly 0% (Jones and Riley, 1989), to 50% (Hickling and Blanchard, 1992). In recent years some of these methodological weaknesses have been corrected, and a number of longitudinal studies using consecutive attenders to

hospital have been published. These studies are not without their limitations but mark a more realistic approach.

Bryant and Harvey (1996) assessed post-traumatic stress symptoms in 114 RTA victims within two weeks of their accidents. Respondents had all attended hospital following their RTA, and had been admitted for at least one day. Using the Impact of Events Scale (IES), Bryant and Harvey found that over 50% of respondents reported moderate or high post-traumatic intrusions or avoidance (IES > 20). They conclude that many individuals experience the core symptoms of PTSD in the immediate aftermath of an RTA. The degree to which these responses are normative or psychopathological is unclear although Horowitz (1986), proposed that individuals adjust to trauma through intrusion and avoidance processes.

In a similar study Harvey and Bryant (1998) assessed Acute Stress Disorder (ASD) in 92 RTA victims within one month of their accidents. In addition, these respondents were followed up at six months post-RTA and re-assessed for PTSD. The authors found that ASD was diagnosable in 13% of respondents, and that 21% had sub-clinical levels of ASD. At six month follow up, 25% of respondents met DSM-IV criteria for PTSD; of those individuals diagnosed with ASD, 78% had gone on to develop PTSD, as had 60% of individuals who met sub-clinical levels for ASD. Harvey and Bryant conclude that these levels of ASD and PTSD are significant, and that the relationship between ASD and PTSD is not linear. They suggest that by reducing the 'overly rigorous' criteria for ASD (a minimum of three dissociative symptoms), ASD could more accurately describe those individuals

likely to go on to PTSD. They note that those individuals assessed to be sub-clinical for ASD, were deemed so due to not meeting the required three dissociative symptoms.

Shalev et al. (1998) report results from a prospective evaluation of PTSD following a variety of traumas. Respondents were 211 trauma survivors recruited at Accident and Emergency, and assessed at one week, one month and four months post-trauma. 85.8% of respondents had been involved in an RTA leading to their attendance at hospital. Shalev et al. found that 29.9% of respondents met criteria for PTSD at one month, and that at four months post-trauma 17.5% had PTSD. The authors used the Clinician Administered PTSD Scale (CAPS), in order to assess and diagnose PTSD. Mayou et al. (1993) assessed 188 consecutive Accident and Emergency RTA admissions using the Present State Examination. Respondents were assessed initially, at three months post-trauma, and finally after one year. Mayou et al. found that 8% of individuals had PTSD at three months, and 5% at one year. Mayou et al.'s (1993) results may be questioned due to the lack of a diagnostically sensitive assessment tool.

Blanchard et al. (1996) assessed 132 RTA victims between one and four months post-RTA, at six months and then 12 months later. Of the 132 respondents they assessed, 48 met full criteria for PTSD initially. By six months 50% of those with initial PTSD had remitted, and by 12 months over two-thirds of initial PTSD individuals were no longer diagnosable. Blanchard et al. note that in the 13 respondents with PTSD at 12 months, there was very little additional improvement

over the next six months. Feinstein and Dolan (1991), conducted research into the prevalence of PTSD in an RTA population of orthopaedic inpatients. Of 48 respondents with leg fractures, 25% met diagnostic criteria for PTSD at six weeks, as did 14.6% at six months post-RTA. Feinstein and Dolan's conclusions are strengthened by their use of clinical interviews and the Impact of Events Scale.

A prospective one year follow-up study, investigating both ASD and PTSD in an RTA population is reported by Koren et al. (1999). These authors assessed 74 injured RTA victims and a comparison group of 19 hospitalised elective orthopaedic patients. Respondents were interviewed at one week, and one, three, six and twelve months post-RTA. At one year follow up Koren et al. found 32% of the RTA group and none of the control to qualify for PTSD diagnosis. The PTSD group were noted to have experienced more frequent and severe symptoms from initial assessment onwards, and that these symptoms had worsened over the first three months. Those individuals without PTSD showed a gradual amelioration of symptoms over the first year. Koren et al. note that the 32% with PTSD at one year had associated psychiatric disorders in 71% of cases. They conclude that the sequelae of an RTA are not limited to PTSD.

Blanchard et al. (1996) report on the assessment of 158 RTA victims who sought medical attention regarding their accident. Using the CAPS as an assessment tool between one and four months post-RTA, they found that 62 respondents met DSM-III-R criteria for PTSD, and that a further 45 respondents were sub-syndromal. Blanchard et al. propose that this is yet more evidence of the association between an

RTA and subsequent post-traumatic stress symptoms. In further support of this association is research conducted by Ursano et al. (1999). These authors assessed acute and chronic PTSD in a sample of 122 RTA victims at one, three, six and 12 months post-RTA. Ursano et al. report that at one month 34.4% of RTA victims had PTSD, at three months 25.2%, at six months 18.2%, at nine months 17.4% and at one year post-RTA 14.0%. They conclude that PTSD is commonplace immediately after an RTA, and that over the first year over 60% of those initially diagnosable will remit. Of those in remission, 47% had done so by six months post-RTA. Blanchard et al. (1995), report that of 98 RTA victims who sought medical attention, 40 met criteria for PTSD at initial assessment. In support of Ursano et al. (1999), 20 of those initially diagnosable were no longer diagnosed with PTSD at six months post-RTA.

The current study employs a prospective longitudinal design and aims to further examine the prevalence of PTSD post-RTA and specifically investigate the role of minor RTAs using consecutive attenders to hospital and standardised psychological measures.

### Depression

Empirical research into post-traumatic depression has been limited despite major depression having long been associated with stressful life events, and with PTSD (Shalev et al., 1998). The co-occurrence of depression and PTSD exceeds the expected effect of simple coincidence, and lends support to an association between traumatic events and depression. In their 1998 study Shalev et al. prospectively

evaluated the onset, overlap and cause of PTSD and depression following trauma. As noted above, the authors assessed 211 attenders at Accident and Emergency (85.8% of whom were RTA victims) at one week, one month and four months post-trauma. In addition to PTSD rates quoted above, Shalev et al. report that 19.0% of respondents were suffering with major depression at one month, and 14.2% at four months post-trauma. In terms of co-morbidity, 44.5% of respondents had co-morbid depression and PTSD at one month, and 43.2% at four months post-trauma. The authors note that co-morbidity was associated with greater symptom severity and lower levels of functioning. This study provides no evidence for a chronological (or causal) progression from PTSD to depression. Evidence for the chronological progression from PTSD to depression comes from the National Co-morbidity Study (Kessler et al, 1995). The NCS found that 78.4% of respondents with co-morbid PTSD and depression, reported that the onset of their affective disorder followed that of PTSD.

Blanchard et al. (1998) propose that the overlap in diagnostic criteria for PTSD and Major Depressive Disorder may account for a degree of the observed co-morbidity. The emotional numbing symptoms of PTSD overlap substantially with the symptoms of anhedonia characteristic of depression. The authors note that sleep disturbance, concentration problems and a lack of enjoyment of activities (all symptoms of PTSD) if present, represent three of the necessary five symptoms needed for a diagnosis of depression. Blanchard et al. pose the question 'Are PTSD and Depression interrelated manifestations of some unitary construct such as 'response to trauma', or are they distinct responses to trauma?'



In an attempt to answer this question, Blanchard et al. (1998) assessed 107 RTA victims with regards to PTSD and depression. Of 62 respondents who experienced PTSD at between one and four months post-RTA, 33 also met criteria for major depression, in 27 of whom the depression commenced after the RTA. The authors report that co-morbid respondents were more subjectively distressed and were less likely to be in remission at six months. This supports Shalev et al.'s (1998) assertion that co-morbidity enhances chronicity of PTSD. Blanchard et al. conclude that PTSD and depression are different disorders, rather than different manifestations of a single disorder. They suggest that there is no evidence for raising the symptomatic threshold for depression, as those respondents with seven or eight depressive symptoms did not differ from those with the minimum requirement of five symptoms. Both Shalev et al. (1998), and Blanchard et al. (1998), conclude that PTSD and depression are independent sequelae of trauma; further investigation is needed in order to tease apart the complex interactions of trauma, depression and PTSD. The current study aims to plot the natural history of depression following minor-RTA.

### Anxiety

In their comprehensive review Taylor and Koch (1995) report that anxiety disorders (including, but not exclusively PTSD and ASD), are the most common psychological sequelae of an RTA. Bryant and Harvey (1996) report data on 114 RTA victims assessed within two weeks of their accident. They found 76% of respondents reported moderate/high state anxiety, and 63% moderate/high trait

anxiety on assessment post-RTA. Furthermore, Mayou et al. (1991), found that in a five year follow-up of RTA victims, anxiety related driving problems were persistent in 10-20% of respondents. Mayou described how these individuals typically reduced their travel, avoided cars and how some suffered from diagnosable phobic anxiety. Di-gallo and Parry-Jones (1996) suggest that individuals suffering from phobic anxiety related to an RTA, without the dramatic re-experiencing symptoms of PTSD, may be deemed to be suffering from a disorder of 'internal causation'. They suggest that PTSD provides an obvious and understandable link between cause and effect, where in contrast anxiety or depression do not. Mayou (1997), warns against the disabling effects of phobic anxiety and/or depression resulting from an RTA, especially in the light of the expected societal response to an RTA per se (see previous discussion). The current study aims to clarify the extent of anxiety present following minor-RTA.

Kuch et al. (1991, 1994) provide a definition and description of "Accident Phobia". They note that accident phobia is characterised by three features: i) Specific phobia as diagnosed in DSM-IV, ii) Onset and content related to the accident, iii) Anxiety and avoidance centring on excessive fear of repetition of the accident. Kuch et al. propose that exposure to driving/accident related stimuli may induce panic and increase somatic symptoms, and that subsequent avoidance includes both cognitive and behavioural strategies. There has been far less attention paid to accident phobia in the literature, than to PTSD, ASD or depression. One reason for this may be the currently unresolved issue of aetiology in many individuals identified as suffering with driving phobia. It is often unclear that the phobic symptoms started as a direct

result of an RTA, and there is a need for prospective longitudinal research to address this issue.

Ehlers et al. (1994) assessed 56 individuals defined as driving phobic (criteria as Kuch et al. above, without onset and content related to the accident), and 31 controls. Respondents were examined with regard to anxiety disorders (DSM-IV), agoraphobic avoidance, driving history and phobic history. 81% of driving phobics reported having had panic attacks, but only 14% met criteria for panic disorder. Interestingly, and of relevance to the accident phobia debate, 15% reported an RTA associated fear pathway (onset and content related to the RTA). 53% of driving phobics reported panic attacks as the primary reason for their phobia, and a fear of anxiety symptoms whilst driving. Driving phobics reported no more involvement in an RTA than controls. Ehlers et al. conclude that the apparently causal event was a rise in anxiety whilst driving, either triggered internally or as a transitory overreaction to a minor external event. Only a small number of respondents attributed their fears to RTA-specific conditioning.

Sartory et al. (1992), assessed and interviewed 15 individuals with self-reported driving phobia, and 15 controls. These authors found that during an in-vivo behavioural avoidance test, those individuals with reported driving phobia exhibited higher subjective anxiety and heart rate when compared to controls. The driving phobic group also exhibited higher trait and state anxiety, greater agoraphobic avoidance, higher depression scores and less perceived internal control. Sartory et al. found that a significant number of driving phobics worried they would have a

panic attack whilst driving, and attributed their fears to incidents of unexpected rises in anxiety whilst driving, rather than to an RTA.

Taylor and Deane (1999) have further examined the issue of the aetiology of driving related fears. They assessed 190 RTA victims regarding their reactions to normal driving situations, reactions to their RTA and their anxiety response patterns. Taylor and Deane found that only 27% of individuals reporting driving related fears attributed them to a direct conditioning pathway, such as an RTA. The trait driving fear pathway characterised by reports of 'I have always been this way', accounted for 25% of respondents. The authors suggest that the low figure of 27% attributing fears to a direct incident may even be an exaggerated figure. They propose that individuals involved in a memorable RTA will be more likely to ascribe driving fear acquisition to this event. Taylor and Deane conclude that non-associative pathways to driving fear development may be important. It is suggested that a series of small unconditioned stimuli slowly inflate a weak conditioned fear response, with individuals not connecting such small events with the original conditioning pathway when asked to ascribe causality to their fears in retrospect.

In a prospective follow-up study Taylor et al. (1999) assessed the stability of fear onset ascriptions and fear severity over time in a subset of 85 respondents from the Taylor and Deane (1999) study. They found that fear relevant negative thinking appeared to worsen over time, while physiological reactions and general anxiety remained stable. They conclude that, although fear severity appears stable over time, fear onset ascriptions do not. 46% of respondents changed the pathway to

which they ascribed their fear from the Taylor and Deane study, to the present one. Taylor et al. (1999), suggest that prospective longitudinal studies are needed to track fear development post-RTA, in order to develop a more accurate understanding of the associations involved. Both anxiety and driving phobia are addressed in the current research.

### 1.7 Conceptual Models of PTSD

As noted above, a major part of the current study is concerned with examining which environmental and psychological factors are involved in the development and maintenance of post-traumatic psychological distress. In order to properly investigate this issue it is important to review the theoretical underpinnings of why certain variables may be implicated in this process. The vast majority of both empirical research and theoretical conceptualisations of psychological morbidity post-trauma are concerned with PTSD, rather than depression and anxiety. As a result the current research focuses on predicting and maintaining factors of PTSD following a minor RTA. What follows is a review of relevant psychological theories of PTSD development and maintenance, in order to inform current hypotheses. For the reader who wishes to explore theoretical models more extensively than this document permits, descriptions of other psychological and biological theories of PTSD are included in the appendices (e.g. Horowitz, 1979; Janoff-Bulman, 1985, 1992; Foa et al., 1985; Dagleish, 1999; Siegel, 1995; Yehuda, 1991; van der Kolk, 1984).

### Behavioural Models

Behavioural conceptualisations of PTSD are based fundamentally on Mowrer's (1960) two-factor theory. The theory proposes that any stimulus associated with a traumatic event can become, through the process of classical conditioning, capable of eliciting a conditioned response similar to that associated with the original traumatic event. An individual experiencing such conditioned responses will learn avoidance behaviours in order to escape from, or prevent the conditioned response. In turn, these avoidance behaviours are negatively reinforced through the process of operant conditioning, making them very resistant to extinction. Avoidance behaviours effectively obstruct an individual's realisation that the conditioned stimuli have ceased to be associated with the original trauma, and hence fear is maintained. Keane et al. (1985), suggest that as time passes post-trauma, an individual may inadvertently or intentionally expose themselves to trauma related stimuli, leading to anxiety, negative affect and arousal. In this manner new sets of conditioned stimuli develop, a phenomena Keane et al. refer to as stimulus generalisation. Keane et al. (1985), propose that this model accounts for both the aetiology and maintenance of PTSD symptoms including re-experiencing, avoidance and hyperarousal.

### Cognitive models

The cognitive model of PTSD is the most developed psychological model and attempts to account for a large range of factors implicated in PTSD. All cognitive models have a resemblance. They all assert that an individual brings to a traumatic event a set of pre-existing beliefs and models of the world, of others and of

themselves. It is proposed that these mental representations are the product of the individual's prior experiences. Dagleish (1999), suggests that traumatic events introduce highly salient and often incompatible information into these pre-existing meaning structures. This trauma information must then be integrated into mental representations in order to avoid psychological chaos. Cognitive theories argue that it is this attempt to integrate trauma information into existing models that leads to the various phenomena which characterise post-traumatic stress. Successful resolution of the trauma occurs when the new information is integrated into the existing models, often by virtue of changes in these same models. Unsuccessful resolution occurs when individuals are unable to bring trauma information into accord with existing models (Dagleish, 1999).

Brewin et al. (1996) propose a dual-representation theory of PTSD, based on earlier work by Brewin (1989). It is suggested that traumatic events experienced after early childhood give rise to two sorts of memory: verbally accessible memory (VAM) and automatically/situationally accessible memory (SAM). It is proposed that sensory input is subject to both conscious and non-conscious information processing (Brewin, 1989). Non-conscious processing permits far more detailed and extensive computations than conscious processing, as it is rapid, parallel and multiple. It follows that the output of these processes will be stored verbally in different ways. Teasdale and Barnard (1993), propose that dual representations in memory of traumatic experience, are the minimum cognitive architecture within which this complex data can be understood. Hence, one representation will be conscious and verbally accessible, and a second representation will be non-

conscious and situationally accessible. Brewin et al. (1996) suggest that retrieval of non-conscious trauma memory will be non-deliberative, and will occur when triggered by either internal or external trauma related stimuli. The re-experiencing symptoms of PTSD will be experienced when SAM representations are triggered, intruding from non-consciousness into consciousness. It follows, therefore, that an individual's ability to consciously recount a traumatic event would be the function of the accessibility of VAM representations. Brewin et al. propose that successful emotional processing of trauma needs to include both VAM and SAM in order to be successful. Individuals need to consciously integrate the verbally accessible information in VAM into their pre-existing models. Brewin (lecture, London 2000), suggests that through repeated exposure and elaboration, VAMs become sophisticated, contextualised and temporal, and are then able to inhibit SAM activation. VAMs can be developed consciously to contain all SAM elements, plus reparative and protective cognitive information.

Brewin et al. account for PTSD symptoms, based on the emotional processing outlined above. They note that emotional processing after any trauma has three potential endpoints. The first is completion/integration where memories of the trauma have been fully processed, worked through and integrated with memories of self and world view. The second potential end point, according to Brewin et al. (1996) is that of chronic emotional processing. In this instance trauma may have been so severe and prolonged, or have had such a profound impact on an individual's sense of self, that the difference between prior assumptions and trauma information is too great to integrate. There is an inability to stop SAMs pervading



consciousness. The third potential end point of emotional processing is premature inhibition. Inhibition may occur as a result of sustained efforts to avoid the reactivation of unpleasant SAMs or VAMs. An avoidance schema may develop. Within this inhibited state further emotional processing cannot occur. Such a state, according to the theory, may be characterised by numbing, dissociation, phobic avoidance and somatisation.

Dalgleish (1999) asserts that a major strength of dual-representation theory is the application of a coherent cognitive architecture in which to understand personal schemas, traumatic information and the interaction between the two. The model has similarities to that of Horowitz (editing of VAMs into congruence with prior schemas), and of Foa (activation of SAM and the integration of non-trauma information). Figure 1. below provides a schematic summary of the model.

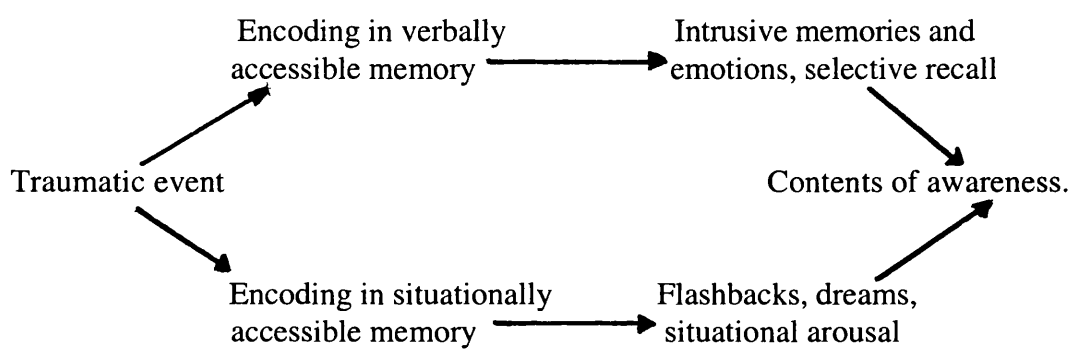


Figure 1. A schematic illustration of dual representation theory applied to PTSD (Brewin et al., 1996).

Ehlers and Clark (2000) propose that PTSD becomes persistent when an individual processes trauma information in a way that leads to a sense of current threat. The authors suggest this sense of threat arises as a consequence of, i) excessively negative appraisals of the trauma and/or its sequelae, and ii) a disturbance of autobiographical memory characterised by poor elaboration and contextualisation, strong associative memory and strong perceptual priming. It is proposed that a series of behavioural and cognitive coping strategies prevent changes in appraisal and memory post-trauma.

Ehlers and Clark (2000) assert that an individual who appraises their traumatic experience negatively and globally, will be more likely to develop PTSD. Individuals who interpret trauma as having global, negative implications for the future, e.g. 'I attract disaster', 'I am partly to blame' or 'I am not able to cope with the world', are unable to see the event as time limited and historical. These attributions are hypothesised to lead to a sense of current threat, and an individual experiencing them will be less able to place the traumatic event properly in the past.

The authors also suggest that the manner in which an individual appraises, or interprets, their post-trauma symptoms will impact upon the maintenance of PTSD. Based on the work of Ehlers and Steil (1995), the authors propose that negative appraisals such as 'I am not coping with this' or 'I must be going mad' function both to create a sense of current threat, and to make avoidance strategies more likely. In addition, they note that negative interpretations of symptoms lead to the production of negative emotions such as anxiety, depression and anger.

Ehlers and Clark (2000) also place heavy emphasis on the nature of traumatic memory. It is suggested that intense emotion, confusion or dissociation at the time of trauma make it very difficult for individuals to process the meaning of the event. Ehlers and Clark suggest that individuals with PTSD fail to process this meaning in an organised and contextualised manner, and that this leads to an emphasis on processing of the sensations experienced at the time of trauma. This sensation, or data driven processing, creates strong associative memories with many primed sensory triggers, capable of intruding into consciousness unintentionally. Ehlers and Clark note that the resulting memory trace will be poorly discriminated from other memory traces, as it will lack elaboration, context and meaning. As a result, stimulus discrimination between trauma related and non-trauma related stimuli will be poor, and intrusive memories will occur (re-experiencing symptoms of PTSD).

The question remains of how appraisals and traumatic memories are related, and how they lead to the characteristic avoidance seen in PTSD. Ehlers and Clark suggest that appraisals and memories have a reciprocal relationship. It is proposed that recall of a traumatic event will be biased by appraisals, and hence individuals will selectively retrieve information that is consistent with these appraisals. This selective retrieval prevents individuals remembering aspects of the trauma that contradict their appraisals, and thus prevents appraisal change.

Furthermore, the authors note that an inability to remember details of the event can be interpreted as evidence either of self-blame, or of some insidious post-traumatic reaction, both of which encourage a sense of current threat. Additionally, the 'here

and now' quality of memories often gives rise to a natural desire to avoid the associated emotional response, and to appraisals of a permanently altered self (Ehlers and Clark, 2000).

Based on their cognitive conceptualisation of PTSD, the authors propose a three-pronged approach to treatment. First, based on the hypothesis that traumatic memories in PTSD are poorly elaborated and inadequately integrated into time and place, elaboration of the memory is advocated in order to enhance higher order, meaning based, intentional retrieval, and to inhibit unintentional triggering. By using taped verbal accounts, or a written narrative, an individual may develop a new autobiographical version of events, integrating memories with older mental representations, and incorporating time related safety information. Second, emphasis is placed on addressing problematic attributions using cognitive therapy techniques. Ehlers and Clark note that any appraisal that gives rise to a sense of current threat needs to be modified, and that maladaptive or problematic appraisals should be examined in the light of alternative available evidence. Finally, Ehlers and Clark suggest that dysfunctional behavioural and cognitive strategies that prevent memory elaboration, exacerbate symptoms or hinder re-assessment of problematic appraisals, need to be dropped for successful recovery. These include strategies such as thought suppression and phobic avoidance.

## 1.8 Prediction and Maintenance of PTSD post-RTA

Adverse psychological reactions following an RTA are a highly prevalent problem (see this chapter, 1.6). Much progress has recently been made in trying to understand these problems with a view to treatment. An understanding of who is vulnerable, or likely, to develop mental health problems following an RTA and who is not, may provide the bases for the allocation of early intervention resources. The psychological models of PTSD described above aid our understanding of how some individuals experience psychological distress post-RTA where others do not. Foreknowledge of who is likely to develop PTSD, for instance, could allow an optimal allocation of potentially scarce mental health resources (Blanchard et al., 1996). The issue of which factors predict and mediate poor psychological morbidity post-RTA has been addressed by many authors. What follows is a detailed account of the evidence so far. Both the intuitive environmental factors (e.g. accident severity) and newer psychological considerations informed by theoretical conceptualisations of PTSD are discussed.

### Accident and Injury Severity

There is inconsistent evidence for the predictive power of accident severity and/or injury severity during RTA, as regards the subsequent development of mental health problems. Frommberger et al. (1998), assessed 179 RTA victims all of whom had been admitted to hospital as a result of their injuries. The respondents were assessed initially, and again six months later when 18.4% of the sample met DSM-IV diagnostic criteria for PTSD. The authors found that the best predictor of six month PTSD status was injury severity, although also identified baseline anxiety,

depression and ASD as good predictors. Frommberger et al. interpret their results in the context of the existing literature, and note that their finding has little support elsewhere. They suggest that their associated findings of the importance of initial psychiatric symptoms in subsequent PTSD development, fits well with literature documenting the predictive power of ASD in PTSD (e.g. Buckley et al., 1996; Blanchard et al., 1996; Brewin et al., 1999). It may be that injury severity in this sample is very closely associated with initial distress levels.

In a similar research project Blanchard et al. (1996) assessed 98 RTA victims. They found that extent and severity of injury significantly predicted the subsequent development of PTSD, as did perception of threat to life. Between them, these two variables only accounted for 12.2% of the variance. The authors suggest that although injury severity may be important in the development of PTSD, the perception and appraisal of fear and threat may be more significant in predicting subsequent disorder. Blanchard et al. (1995) note that 'in some individuals, it is the perceptions resulting from the accident rather than the seriousness of the injuries, that are important in PTSD'. This is congruent with cognitive conceptualisations of PTSD (e.g. Ehlers and Clark, 2000). Neither Mayou et al. (1993) nor Bryant and Harvey (1995) found a predictive association between injury/accident severity and subsequent post-traumatic mental illness. Di-Gallo and Parry Jones (1996) propose that the consequences of an outwardly trivial accident that attracts little or no attention, should not be overlooked. In such circumstances RTA victims may fear derision if they admit to psychological distress. This hypothesis is yet to be tested empirically.

Jaspers (1998) addresses the issue of whiplash resulting from RTA. Whiplash (cervical-acceleration injury), arises from the hyperextension and hyperflexion of the neck, and is common in car drivers/passengers hit from behind. Jaspers notes that there are incidental reports in the literature about a possible connection between whiplash and PTSD, but that there is currently no empirical evidence to support this assertion. Mayou et al. (1993) found no predictive power for whiplash as regards PTSD, as did Blanchard et al. (1996). Jaspers (1998), propose a non-predictive association between whiplash and PTSD, to try and account for its co-occurrence. Jaspers proposes that the pain resulting from the hypertonia of the neck is a form of respondent pain that becomes chronic under the influence of operant processes. It is conceivable, according to the author, that post-traumatic stress affects this process, by preserving both the hypertonia and the operant process. It appears, therefore, that evidence for the role of accident/injury severity in the development of PTSD post-RTA is weak.

### Litigation

It has been widely believed that litigation, and its settlement, play a large role in the natural history of psychological symptoms and disability following an RTA. Potentially pejorative terms such as 'accident neurosis', 'compensation neurosis' and 'litigation neurosis' have appeared for many years in the literature. In a recent study Blanchard et al. (1998) set out to empirically test the predictive and mediating power of litigation in psychological symptoms post-RTA. The authors assessed 132 RTA victims initially and at one year follow-up. 67 respondents were involved in litigation at initial assessment and exhibited significantly higher post-traumatic



stress scores, and had suffered more severe injuries than the 65 respondents not involved in litigation. At one year follow-up, 18 litigants had settled and the remaining 49 litigants remained pending. All three groups had improved in major role function and had reduced post-traumatic stress symptoms by the follow-up stage. The litigants group (whether settled or not), did however exhibit significantly less reduction in anxiety and depression scores over the first year.

Blanchard et al. (1998) suggest that the failure to find a difference between settled and pending groups challenges the long held assumption of RTA victims 'malingering' in order to benefit from litigation settlements. Mayou et al. (1993) also found that there were no effects of initiating or settling litigation on psychological status. Blanchard et al. attempt to explain the raised post-traumatic stress symptoms at baseline, and anxiety and depression scores at follow-up. In terms of initial distress it may be that those more distressed choose to seek compensation, or alternatively that initiating litigation is in itself stressful, and keeps the traumatic event 'in mind'. It may be that the need to confront the traumatic event through interview and in written form during litigation, thwarts an individual's characteristic efforts at avoidance and results in a resurgence of intrusive ideation and increased arousal. Blanchard et al., (1998) argue that litigation, and the delays and frustrations of the judicial system, may further traumatise victims leading to anxiety and depression. It is suggested that victims are often left feeling that the legal process comes before their interests and well being. If this is the case litigation may provide the stimuli which some individuals find aversive and is

congruent Ehlers and Clark (2000), in that appraisal of trauma and its sequelae may perpetuate symptoms.

### Responsibility for the RTA

A related idea to that of litigation is responsibility. Delahanly et al. (1997) assessed a group of RTA victims two weeks after their accidents and subsequently followed them up. They found that 19% of respondents who attributed responsibility for the RTA to themselves met criteria for PTSD at two weeks, whereas 29% of respondents who attributed responsibility elsewhere had PTSD. At follow up, 43% of the self-responsible PTSD group had remitted, while only 23% of the other-responsible PTSD group had remitted. In a similar study, Hickling et al. (1999) found that six months after an RTA, remission rates for PTSD differed dependent on attribution of responsibility, in 158 RTA victims. Hickling et al. found that 100% of a self-responsible PTSD group had remitted by six months, whereas only 43% of an other-responsible group had remitted. Both Delahanly and Hickling conclude that those with PTSD who blame themselves for the RTA are less symptomatic initially, and recover more rapidly over six months, than those with PTSD who blame a third party for the accident.

Hickling et al. (1999) suggest that blaming another individual leads to a sense of victimisation. Further, that protracted dealings with the legal system that implicitly blame the victim for their continued symptoms and a greater sense of vulnerability for future RTAs, might contribute to post-traumatic symptoms in some 'other-blaming' individuals. On one level these results seem counter-intuitive, but Hickling

et al. assert that it depends on the extent of self-blame. They note that there is a significant difference between behavioural self-blame such as 'I was driving too fast' and characterological self-blame such as 'I am a very poor driver': the former is readily correctable whereas the latter is not. Hickling et al. (1999), further hypothesise that individuals who fiercely blame others for an RTA may experience a shattering of assumptions as described by Janoff-Bulman (1985; 1992). An individual who firmly and inflexibly (see Dalglish, 1999), believes 'I *always* keep my car under *strict* control and *never* break the rules; everyone *must* do the same', may be at risk following an RTA of experiencing a large disparity between incoming information and prior assumptions. Similarly, an individual who already believes 'I am a bad person' may have their beliefs confirmed by an attribution of self-responsibility such as 'I am a very bad driver'. It may be that Hickling et al. did not sample any individuals with complementary negative pre-existing beliefs and negative self-responsibility attributions.

### Immediate Post-Traumatic Reaction

The assumption that immediate post-traumatic stress symptoms are a precursor to long term post-traumatic problems is widely held. The core phenomena of high levels of distress during the acute trauma phase was formalised in DSM-IV with the introduction of the ASD diagnosis. A key rationale for the inclusion of ASD in DSM-IV was the assumption that ASD would predict PTSD. This assumption has recently been empirically tested. Blanchard et al. (1996) assessed 182 RTA victims initially and at one year follow-up. The authors were interested in predicting which respondents would remit from post-traumatic stress symptoms by one year, and

which would not. They found that three variables (initial scores on irritability, foreshortened future and vulnerability during the RTA) combined to correctly identify 79% of remitters/non-remitters at one year follow-up. Furthermore, four variables combined to predict 64% of the variance in the degree of PTSD at one year follow-up; presence of alcohol abuse, axis II disorder at initial assessment, initial hyperarousal score and initial avoidance score. Blanchard et al. (1996) suggest therefore, that immediate post-traumatic reactions exhibit predictive power of PTSD status and severity at one year post-RTA.

Barton et al. (1996) compared a group of RTA victims diagnosed with ASD and a group without ASD. They found that the ASD group were different in their pre-trauma functioning, and had a history of more previous psychological dysfunction than the non-ASD group. The authors report that in their study ASD did not predict more severe PTSD symptoms at follow-up (according to the CAPS), and that ASD exhibited no prognostic significance in recovery from RTA induced PTSD. This is interesting as the ASD group were retrospectively identified to have experienced more psychological problems in the past. Barton et al. conclude that these results call into question the predictive power of the ASD diagnosis as regards subsequent PTSD.

In contrast to Barton et al. (1996), Brewin et al. (1999), report results supporting the ASD diagnosis as an accurate precursor to PTSD. In a group of 157 crime victims interviewed within one month and at six months post-crime, the authors found a prevalence of ASD at 19% and PTSD at 20%. They report that ASD

diagnosis correctly classified 83% of the subsequent PTSD group, and the regression analysis revealed that ASD and high levels of re-experiencing and arousal symptoms made independent contributions to predicting PTSD. Brewin et al. conclude that in their sample of crime victims there is clear support for the predictive role of the ASD diagnosis, and for the proposed symptom thresholds, (they found that increasing symptom thresholds did not increase accuracy of predictions), suggested in DSM-IV. They note that the predictive utility of ASD was confirmed, and that high re-experiencing and arousal symptoms made an equal independent contribution. It may be that a simple count of re-experiencing or arousal symptoms is a highly efficient method of predicting PTSD (Brewin et al., 1999). A unique role for dissociation (part of ASD), in predicting PTSD was not found. This will be discussed in detail later in this volume.

Koren et al. (1999) present results from their study investigating the predictive power of ASD for PTSD in a group of 74 injured RTA victims. They found that existence of ASD immediately after the accident was the best predictor of PTSD, better than accident/injury severity. They suggest that the development of PTSD can be predicted as early as one week post-RTA on the basis of the existence and severity of early symptoms. The authors extend their discussion to the utility of the one month cut off point for transition from ASD to PTSD. It is suggested that the first three months, rather than just the first month, are the critical period for the gradual development of PTSD; Koren et al. report that the initial differences between respondents on severity of symptoms intensifies over three months, and note that between-time correlations peak at this point representing symptom

crystallisation. It is proposed that a three month cut off point for ASD to PTSD be investigated. Koren et al. (1999) therefore support the predictive power of initial post-traumatic distress in the development of chronic PTSD.

Harvey and Bryant (1998) assessed 92 RTA victims within one month of their accident and six months later. They found that 78% of respondents diagnosed with ASD went on to develop PTSD and that 60% of respondents who met sub-clinical levels for ASD also later developed PTSD. Further analysis revealed that only a subset of ASD symptoms predict PTSD; acute numbing, depersonalisation, reliving and motor restlessness. Harvey and Bryant note that overall ASD diagnosis was better at predicting the absence of PTSD than its presence (high negative predictive power). It is suggested that ASD diagnosis is therefore not an adequate predictor of the presence of PTSD, and furthermore it is not clear whether the dissociative or the post-traumatic stress symptoms of ASD are predicting PTSD (or its absence). In a similar study not using the ASD diagnostic criteria, Blanchard et al. (1997) assessed 145 RTA victims between one and four months post-RTA, and six months later. It was found that four variables, including severity of initial symptoms, degree of initial physical injury, physical recovery and trauma in a close family member accounted for 84% of the variance of remitters/non-remitters from PTSD at six month follow-up. Blanchard et al. report that initial CAPS score was the overall best predictor of diagnostic status at six month, accounting for 17% of the variance. This supports the assumption that initial distress predicts subsequent PTSD and that the severity of that initial reaction is of importance.

The importance of autobiographical memory in PTSD has been discussed by Ehlers and Clark (2000), Dalgleish (1999), and Brewin et al. (1996). In a recent study Harvey et al. (1998) have examined the role of autobiographical memory in ASD and its role in predicting subsequent PTSD. Harvey et al. (1998) found that in a group of RTA victims diagnosed with ASD, respondents found it more difficult to report memories of trauma than respondents without ASD. This difficulty accounted for 25% of the variance of PTSD occurrence at six month follow-up. Harvey et al. (1998) hypothesise that individuals minimise their awareness of traumatic memories to alleviate the associated discomfort, and that such an impaired access to traumatic memory will impede trauma recovery. The authors suggest that autobiographical memory in ASD may be poorly organised, elaborated and contextualised (see Ehlers and Clark, 2000 for a similar description of memory in PTSD). Foa and Kozak, (1996) note that adaptive reactions to trauma require a capacity to integrate corrective information in order to modify threat based schemas. Individuals with ASD, according to Harvey et al. (1998), will find this difficult, as they exhibit poor recall of both trauma and of general autobiographical specific memories. In conclusion, this study provides more evidence for, i) the predictive role of ASD in PTSD, and ii) the specific role of disrupted autobiographical memory in post-traumatic stress reactions, as highlighted by cognitive models (Ehlers and Clark, 2000; Dalgleish, 1999; Brewin et al., 1996).

### Anger

A number of recent studies have examined the role of anger in post-traumatic mental health problems. In a prospective study Riggs et al. (1992) found that

feelings of anger predicted chronic PTSD in individuals who had been assaulted. Also studying victims of violent crime, Andrews et al. (2000) investigated the role of anger in the development of PTSD. Andrews et al. found that after controlling for gender, education and injury severity, anger (along with shame), was a consistent predictor of baseline PTSD scores. Both anger and shame were seen to make independent contributions to PTSD total scores and to the three sub-scales of re-experiencing, avoidance and hyperarousal. At six months follow-up, Andrews et al. (2000) report that anger and shame were significant in predicting PTSD once the controlled variables were accounted for. Furthermore, at this follow-up stage anger was found to significantly predict re-experiencing symptoms but not avoidance. The authors hypothesise that anger may function to block the processing of fear, or contribute directly to PTSD symptoms such as irritability. Foa et al. (1995) propose that the activation of anger may allow victims to avoid feelings of anxiety and thereby impede the processing of distressing feelings, seen as necessary for good post-traumatic psychological adjustment.

Ehlers et al. (1998) conducted a prospective longitudinal study assessing 967 consecutive patients attending hospital after an RTA, initially, three months later and again at one year post-accident. They found the prevalence of PTSD to be 23.1% at three months and 16.5% at one year. The authors found that chronic PTSD was related to some objective measures of trauma severity, perceived threat, and dissociation during the RTA, to female gender, previous emotional problems and to litigation. Ehlers et al. report that a number of maintaining psychological factors including anger cognitions enhanced the accuracy of this prediction of



PTSD. Furthermore, anger cognitions helped to identify cases of delayed onset PTSD at one year. In discussing their findings Ehlers et al. note anger to be very common in victims of trauma. They suggest that anger functions to maintain a current sense of threat post-trauma, directly leading to the perception of threat and preventing an individual from seeing the RTA as an isolated negative event in the past. It is proposed that anger's role in delayed onset PTSD reinforces its role as a maintaining factor. These findings support the role of cognitive-affective variables in predicting and maintaining post-traumatic symptoms outlined in Ehlers and Clark (2000).

### Thought Suppression

In 1989, Wegner reported that efforts to suppress a thought may produce a subsequent increase in its frequency. Wegner described this as a 'rebound effect', and outlined a theory of 'ironic control'. Ironic control theory proposes that the degree of mental control an individual enjoys will be significantly influenced by the presence of cognitive load. Wegner (1989) suggests that during attempted thought suppression an operating process directs attention towards a thought other than the unwanted one and concurrently a monitoring process searches for failures to achieve the desired suppression. It is proposed that identification of failed suppression by the monitor reactivates the operating process, arguably having the ironic effect of sensitising an individual to the very thoughts which are targeted for avoidance (Wegner, 1994). This ironic effect is hypothesised to be more marked with additional cognitive load (e.g. anxiety) which impedes the effectiveness of the operating process leading to greater sensitisation. Such competition for working

memory will enhance the activities of the monitor. Wegner (1989) uses ironic control theory to explain intrusions and cognitive avoidance in PTSD. It is suggested that the anxiety characteristic of PTSD can be conceptualised as cognitive load, which decreases working memory capacity (Eysenck, 1982). This same anxiety leads to prompting of spontaneous thought suppression of anxiety provoking thoughts, setting in motion ironic control sensitising an individual to the thoughts, increasing their frequency and associated anxiety levels; a vicious cycle develops.

Ironic control theory has been tested empirically. McNally and Ricciardi (1998), found that negatively valent thoughts may respond differently to neutral thoughts, following attempts to suppress them. The authors report that subjects in a 'neutral thought' group experienced a decline in thoughts previously targeted for suppression, whereas subjects asked to first suppress a personally relevant negative thought experienced a three-fold increase in its frequency of occurrence when later given permission to express it. Harvey and Bryant (1998) exposed 72 non-clinical participants to a distressing film, a positive film and a neutral film. They found that the expected exaggerated rebound effect occurred following intentional thought suppression in all three exposure groups. Harvey and Bryant suggest that the failure of the distressing film to produce greater rebound effects in thought occurrence may be due to it not being personally meaningful. It would appear therefore, that negative personally meaningful thoughts are susceptible to the rebound effect of thought suppression.

Harvey and Bryant (1999) investigated Wegner's (1989) assertion that anxiety will mediate and enhance a rebound effect following thought suppression. The authors hypothesised that thought suppression would impede participants' (N=96), performance on cognitive tasks, that individuals using thought suppression would ironically experience more intrusive thoughts, and that these effects would be exaggerated in anxious individuals. Harvey and Bryant (1999) report that thought suppression leads to impairment in cognitive tasks, an increase in intrusive thoughts, and that these differences were mediated by anxiety. They note that high anxiety participants appear to spontaneously suppress thoughts. It may be that this is a learned behaviour related to trait anxiety, or alternatively a state effect related to stressful situations. There is a need for further research examining whether such cognitive strategies are characterological or situation specific (e.g. related to traumatic events). Harvey and Bryant conclude that there is evidence to support the mediating role of anxiety in the rebound effect of thought suppression.

A number of recent studies have examined thought suppression and anxiety in post-traumatic populations, in order to further explore their roles in the development of PTSD. Warda and Bryant (1998) investigated thought control strategies in 40 RTA victims, 20 with a diagnosis of ASD and 20 without. They found that ASD individuals engaged in significantly higher levels of cognitive self-punishment (e.g. 'Don't be so stupid. Don't think that') and worry and that attempted suppression of traumatic memories in this group was associated with increased intrusions. This result is congruent with Wells and Davies (1994) who reported that punishment and worry were associated with controlling unwanted thoughts. Furthermore, Harvey

and Bryant (1998) investigated thought suppression in 48 RTA victims, 24 with a diagnosis of ASD and 24 without. The ASD individuals reported higher ratings of anxiety, frequency of trauma related thoughts, and attempted suppression of trauma related thoughts than non-ASD individuals. These findings provide more evidence for an association between cognitive avoidance and increased intrusion in trauma survivors. It also appears that ASD individuals spontaneously engage in thought suppression (Harvey and Bryant, 1998).

Ehlers and Steil (1995) observed that PTSD persistence was predicted by catastrophic negative appraisal of trauma related intrusions, which in turn emerged as a unique predictor of behavioural and cognitive avoidance of the intrusions. The authors propose that negative appraisal may increase motivation to suppress such thoughts, which interferes with natural habituation processes and leads to increased thought frequency. Therefore, the occurrence of long term intrusions may be well predicted by a tendency to suppress immediately after trauma, which is influenced by negative beliefs about the event. In support of this theory Davies and Clark (1998) found that a pre-existing tendency to thought-suppress, combined with negative affect, predicted more frequent intrusions in a non-clinical sample in whom trauma was induced. Negative affect following trauma is hypothesised to be associated with negative attributions of the traumatic event. Davies and Clark (1998) suggest that the sensitisation to target thoughts that occurs post-trauma, and following thought suppression, creates a new set of reminders. They note the similarity between this and Keane et al.'s (1985) assertion that higher order

conditioning may account for the large number of triggering stimuli, which come to elicit post-traumatic memories in PTSD.

Ehlers et al. (1998) in their investigation of 967 RTA victims found that thought suppression enhanced the accuracy with which objective measures of trauma predicted chronic PTSD. Ehlers and Steil (1998), have also shown that suppression of intrusive recollections are correlated with PTSD severity following an RTA. Ehlers and Clark (2000) propose that thought suppression functions to maintain a sense of current threat post-trauma. It is suggested that thought suppression may prevent a change in negative appraisals of the trauma and/or its sequelae, as well as a change in the nature of traumatic memory. Also it may function to increase the frequency of intrusions directly, in accordance with the ironic control theory (Wegner, 1989). Finally, Wenzlaff and Wegner, (1998) show evidence that thought suppression at or immediately after trauma may impede encoding into episodic memory. The authors argue that suppression of certain elements of trauma leads to their enhanced accessibility, thereby undermining the associative links of these items to other items. This fits with Ehlers and Clark's (2000) conceptualisation of traumatic memory as poorly elaborated and discriminated, and Foa et al.'s (1995) assertion that traumatic memory is fragmented.

#### Negative Interpretations of Trauma and Sequelae

Individuals differ widely in the meaning they assign to their experiences. Ehlers and Steil (1995) observed that RTA victims differed in the meaning they attached to the occurrence and content of intrusive recollections of their accidents, and to the RTA

itself. The authors discuss how some individuals see intrusive memories as a normal part of recovery, where others interpret them negatively, for example as an indication that they are going mad. Ehlers and Steil suggest that such negative interpretations are significant in maintaining intrusions as they will determine how distressing intrusions are and the extent to which an individual engages in strategies (e.g. thought suppression and behavioural avoidance) to control them. This avoidance is then implicated in a lack of change in the meaning of the trauma and of the intrusive memories themselves.

Ehlers et al. (1998) found negative interpretations of intrusions to enhance the accuracy with which objective measures of trauma predicted chronic PTSD. Negative interpretations predicted a proportion of the variance of PTSD at one year that could not be explained by PTSD severity at three months. The authors suggest that this underlines the importance of negative interpretations of intrusions as a maintaining factor in PTSD; negative interpretations of intrusions explain a large proportion of the variance of the distress caused by intrusions that is not explained by the frequency of intrusions. Ehlers et al. also found (as noted above), that negative interpretations of intrusions explained a large proportion of the variance of strategies that are intended to control the intrusions, but ironically maintain them. This provides empirical evidence to support the theory proposed by Ehlers et al. (1995) that intrusions and avoidance post-trauma are a function of an individual's idiosyncratic beliefs about the trauma, and the meaning of their initial PTSD symptoms.

Dunmore et al. (1999) investigated predictor and maintenance factors in PTSD in a group of 92 assault victims. They found that the following factors were associated with the onset and maintenance of PTSD; appraisal of aspects of the assault (appraisal of emotions, mental defeat, mental confusion), appraisal of the sequelae of the assault (appraisal of symptoms, perceived negative responses from others, permanent change), dysfunctional strategies such as avoidance, and beliefs impacted upon by the assault. Previous evidence exists implicating a link between negative appraisal of events and initial symptoms in PTSD (e.g. Foa and Riggs, 1993; Dunmore et al., 1997). Dunmore et al. (1999) suggest that victims who believe they will go mad unless they control their intrusive thoughts will make intentional efforts to suppress them, with paradoxical effect. They suggest that other individuals may attempt to 'undo' the traumatic event as a result of its retrospective negative evaluation, using techniques such as worry and rumination. Dunmore et al. (1999) propose that the importance of cognitive factors (including a negative interpretation of events and sequelae) is in the maintenance of a sense of threat. For individuals who interpret their emotional responses as signs of being unstable, out of control, or sick, these emotions are likely to represent a threat to their sense of self (Dunmore et al., 1999). This study provides further empirical support to the role of interpretation, attribution and meaning in the development and maintenance of PTSD, as postulated by Ehlers and Clark (2000).

A recent study examining coping strategies and responses to intrusive memories in a cohort of 56 Ambulance Service workers, was conducted by Clohessy and Ehlers (1999). It was found that 21% of the participants were suffering with PTSD. The

authors found that PTSD severity was predicted by wishful thinking and mental disengagement during incidents, negative interpretation of intrusive memories and maladaptive responses to these memories (rumination, suppression, dissociation). The results are consistent with the hypothesis that responses to trauma that prevent emotional processing (Rachman, 1980) maintain PTSD. These results also lend support to Ehlers and Steil (1995) and Ehlers and Clark (2000), who assert a role for negative interpretations of post-traumatic intrusions in PTSD. Clohessy and Ehlers (1999) note that negative interpretations of intrusions explained the distress caused by intrusions independent of intrusion frequency (as in Dunmore et al., 1999).

Warda and Bryant (1998) investigated cognitive bias in a group of 34 RTA victims, 17 of whom were diagnosed with ASD and 17 of whom were not. They found that ASD participants exaggerated both the probability of negative events occurring and the potential adverse costs of these events, where non-ASD participants did not. Foa et al. (1985), suggest that activation of the fear network in PTSD leads to attentional bias for threat intrusive thoughts of trauma, and exaggerated beliefs about trauma. Foa and Kozak (1986) note that over-estimations of probabilities of threatening events and elevated stress are critical in the maintenance of anxiety. Harvey et al. (1998) report that individuals with PTSD post-RTA exhibit cognitive bias for negative memories. The results of Warda and Bryant (1998) therefore support information processing models of PTSD and the importance of attribution and cognitive appraisal post-trauma. Indeed, the authors found that avoidance symptoms were more closely associated with the perceived cost of negative events,



than with intrusions. Thus, individuals are not simply avoiding aversive intrusions; perceptions and attributions also lead to avoidance. This emphasis on appraisal fits with the cognitive conceptualisation of PTSD suggested by Ehlers and Clark (2000). Of importance here is the direction of causality; is cognitive bias in PTSD a response to traumatic experience or a cognitive style that predisposes to developing PTSD? Kuyken and Brewin (1995) propose that childhood traumas and experiences are related to cognitive styles that predispose individuals to negative reactions to subsequent stress. In support of this predisposition theory 30% of the ASD group and 6% of the non-ASD group in Warda and Bryant's (1998) study had a psychiatric history. The conceptualisation of cognitive style as a trait variable fits with the general cognitive model (Beck, 1976).

There exists evidence of the importance of peri-traumatic perceptions and cognitive appraisals in the development of subsequent PTSD. Harvey et al. (1996) conducted experimental research investigating conscious and pre-conscious processing of threatening information in a sample comprising RTA- PTSD, RTA non-PTSD, and non-RTA participants. Harvey et al. report that PTSD participants showed greater interference on tasks of naming threat words even when words were presented at a level that does not allow conscious recognition. The results indicate that preferential processing of threat-related information in PTSD occurs at a pre-conscious stage of processing. The same interference in pre-conscious processing did not occur in either of the other groups. Harvey et al. (1996) conclude that pre-conscious processing of threat may be specific to PTSD. It remains to be seen whether this is due to pre-conscious activation of fear networks developed in

PTSD, or to a predisposition to pre-consciously attend to threat due to past experiences. Empirical evidence exists in support of peri-traumatic perception of threat and its role in PTSD. Bryant and Harvey (1996) found that intrusive symptoms and avoidance symptoms in a group of RTA victims were best predicted by perception of fear during the RTA. Green et al. (1993) found that initial perceptions of an RTA as life threatening predicted PTSD at one year post-accident and Ehlers et al. (1998) report that perceived threat to life was one of four variables most predictive of PTSD in a cohort of 967 RTA victims. The importance of peri-traumatic, as well as post-traumatic, appraisal fits with Ehlers and Clark's (2000) conceptualisation of PTSD.

### Rumination

Wells and Butler (1997) describe how self-referent negative rumination is characteristic of excessive anxiety and clinically significant worry and how it is implicated in Generalised Anxiety Disorder (GAD). Rumination (i.e. dwelling on negative events and repeated questioning of them), has also been implicated in the maintenance of PTSD. Ehlers and Steil (1995) suggest that ruminating on aspects of a traumatic event and/or its' sequelae (i.e. 'How things could have been different, if only...' and 'Why did this happen to me?'), may prevent changes in negative appraisals, as well as changes in the nature of trauma memory. Also, rumination, like thought suppression, might directly increase the frequency of reliving symptoms. In support of these hypotheses, Ehlers and Steil (1998) have shown that rumination correlates with the severity of PTSD in a group of RTA victims. Similarly, Ehlers et al. (1998) in their investigation of 967 RTA victims found

rumination to enhance the predictive power of objective measures of trauma, as regards PTSD. Rumination at three months post-RTA was found to predict PTSD at one year and also to help identify cases of delayed onset PTSD.

Rumination has also been identified as a consequence of negative interpretations of intrusions, themselves implicated in the maintenance of PTSD (Dunmore et al., 1999). The authors propose, in line with Ehlers and Clark (2000), that rumination is one of a number of unhelpful consequences of negative attributions of trauma and its sequelae. Victims who believe that events or sequelae are to be feared, or to be suppressed or altered retrospectively, may engage in ruminative processes. Ehlers and Steil (1998) suggest that such a strategy leads to a sense of current threat, impedes emotional processing and maintains trauma as a contemporary experience, exacerbating PTSD symptoms. Furthermore, Clohessy and Ehlers (1999) found rumination to be one of a number of variables correlated with PTSD severity in a group of Ambulance Service workers. They understand the role of rumination to be in maintaining distress by blocking emotional processing. This information is congruent with results on the role of worry in maintaining generalised anxiety (Davey and Tallis, 1994).

#### Attitude Towards Emotional Expression

Rachman (1980) suggested that the suppression of strong emotions could impede the processing of emotionally upsetting experiences. Similarly, Pennebaker (1982) hypothesised that a failure to confront traumatic events results in poor health. Williams (1993) suggested that inhibited emotional expression constitutes a

vulnerability for psychological disorder following trauma. Joseph et al. (1994) reported preliminary results of a measure to assess attitudes towards emotional expression (see Research Methodology for a detailed account). Joseph et al. (1994), define attitudes as cognitions (beliefs about emotional expression) and behaviours (tendencies to act in certain ways regarding emotional expression). The attitudes towards emotional expression construct is seen as a trait measure and is hypothesised to predict poor psychological outcome post-trauma (Williams, 1993). Specific cognitions implicated include the belief that one should keep one's feelings under control, that expressing emotions may lead to certain consequences such as rejection and that emotional expression is a sign of weakness. Williams (1993) suggests that a negative attitude towards emotional expression can be seen as a fear of the consequences of expressing emotion.

Williams (1993) examined the idea that individual differences exist in attitude towards emotional expression (AEE) and that these underlie coping behaviour and the failure/propensity to confide in others. Using a preliminary 4-item version of the AEE scale, the authors found AEE was associated with greater psychological distress in survivors of the 'Herald of Free Enterprise' ferry disaster at eight years post-trauma. Bryant and Harvey (1995) investigated predictors of post-traumatic symptoms in a group of 55 RTA victims, one year post-accident. Significant levels of intrusive and avoidance symptoms were reported by 20% of participants and an avoidant coping style and litigation accounted for 41% of the variance in intrusion scores. Bryant and Harvey suggest that an avoidant coping style and a negative attitude towards emotional expression are associated. Indeed, Joseph et al. (1994)

found a good association between the 20-item AEE and the Ways of Coping Questionnaire (Folkman and Lazarus, 1988). Joseph et al. report concurrent validity ( $r=.53, p<.001$ ) in a sample of 180 undergraduates indicating that high AEE scores were associated with low social support seeking. Solomon et al. (1988) have also reported an association between avoidant coping style and PTSD. They suggest that avoidance may mediate negative adjustment following trauma and an association between avoidance and poor help-seeking behaviour. A role for cognitive and behavioural avoidance of both traumatic memories and reminding stimuli (e.g. confiding traumatic information and the associated emotions) in the development of PTSD is congruent with Horowitz's assertion that adjustment is facilitated by habituation. The success of exposure based therapies in PTSD lends further support to this argument and for the role of attitudes to emotional expression in the development and maintenance of such disorders.

Brown and Grover (1998) assessed 594 police officers for exposure to trauma, 'just world' beliefs, availability of social support and negative AEE and examined their relationship to GHQ scores. The most 'at risk' officers to psychiatric symptoms according to the GHQ, had a profile of low 'just world' beliefs, low social support and negative AEE. Brown and Grover found this profile to predict GHQ scores in both high and low stress contexts. This result provides support for the role of AEE in the development of psychological distress post-trauma. Furthermore, the finding that exposure to objective measures of trauma was not associated with GHQ scores is consistent with previous findings (e.g. Bryant and Harvey, 1995), that fail to indicate a linear relationship between trauma severity and stress response

(McFarlane, 1988). It would be wrong to assert that AEE functioned outside a social context (i.e. available social support), but evidence points to an emphasis of individual subjective interpretation of trauma and its sequelae, as proposed by Ehlers and Clark (2000).

In a follow-up of Williams (1993) study with survivors of a ferry disaster, Joseph et al. (1997) assessed a group of 37 survivors at five years post-trauma. They found that negative AEE at three years predicted PTSD at five years and that this association remained even when perceptions of helplessness during trauma and PTSD symptoms at three years, were partialled out. Joseph et al. (1997) propose therefore, that negative AEE, in the form of relatively robust and consistent rigidly applied rules for living, acts to block the emotional processing of traumatic information. The process of integrating and assimilating stressful experiences may be blocked by characterological attitudes that result in a fear of the consequences of expressing one's emotions.

### Anxiety Sensitivity

In 1978 Goldstein and Chambless suggested that panic disorder patients learned to fear the symptoms of anxiety via interoceptive classical conditioning of internal physical sensations. Internal bodily cues become the conditioned stimuli for the conditioned response of anxiety and panic. Goldstein and Chambless (1978) therefore proposed that the 'fear of fear' loop characteristic of panic and anxiety developed as a consequence of an initial panic attack. In 1986 Clark further developed the role of cognition in the aetiology and maintenance of panic. Clark

(1986) suggested that panic attacks arise from the catastrophic misinterpretation of certain bodily sensations, driven by a fear of these anxiety related bodily sensations. Clark's theory of panic suggests that misinterpretation of sensations as dangerous generates anxiety, further exacerbating these feared sensations of arousal such as cardiac and respiratory activity. A vicious cycle develops that culminates in a panic attack. Therefore, Clark's model of panic differs from the behavioural conceptualisation of Goldstein and Chambless, in its emphasis of cognitive misappraisal driven by a fear of autonomic arousal.

Based on Clark's model, Reiss (1991) developed an expectancy theory of anxiety sensitivity (AS). Reiss (1991) defined AS as the fear of anxiety sensations arising from beliefs that the sensations have harmful somatic, psychological or social consequences. The theory proposes that AS functions to amplify fear and anxiety reactions and plays an important role in the aetiology and maintenance of anxiety disorders. Reiss conceptualised AS as a trait mechanism, with innate and acquired mechanisms determining an individual's disposition, or enduring tendency to become frightened by anxiety related sensations. Specific learning experiences including observation, traumatic events and verbal instruction are hypothesised to interact with biological factors to create an enduring trait of AS (Taylor, 1995). AS, therefore, refers to the extent to which an individual believes that autonomic arousal can have harmful consequences. Expectancy theory proposes that AS is a precursor to, not a consequence of, panic.

The construct of AS has been likened to trait anxiety, although psychometric studies indicate that the Anxiety Sensitivity Index (Peterson and Reiss, 1987) and trait measures of anxiety are hierarchically organised, although the causal nature of this organisation remains to be determined. Reiss (1991) suggests that AS (and other fundamental fears, i.e. injury sensitivity and fear of negative evaluation) are causes of trait anxiety. Schmitt et al. (1997) found AS to predict the development of panic symptoms under conditions of stress in a group of Air Force Cadets above and beyond a measure of trait anxiety.

Ehlers (1996) examined the role of AS in maintaining panic in a group of panic patients. Ehlers reports that AS at initial assessment was related to maintenance of panic disorder in untreated panic patients, maintenance of spontaneous panic attacks in infrequent panickers, and first occurrence of panic attacks in simple phobics/controls. AS accounted for more variance than did previous panic status and all other predictor variables. Schmitt et al. (1997) investigated the role of AS as a premorbid risk factor for the development of anxiety pathology, in 400 non-clinical Air Force Cadets undergoing stressful and intense basic training, over a five week period. AS was found to predict the development of spontaneous panic attacks even after controlling for a history of panic attacks and for trait anxiety. 20% of high AS participants experienced a panic attack during their five week training, compared to only 6% of the rest of the sample. Schmidt et al. note that overall the cadets had low AS ratings and assert that panic can be induced even in individuals with very few arousal-related fears when substantial stressors are involved. The authors also found AS to predict depression, a result which requires



replication and examination before any firm conclusions can be drawn. Schmidt et al. (1997) conclude that AS acts as a cognitive trait that places individuals at risk of developing psychopathology in the context of stress. Maller and Reiss (1992) found AS to be highly predictive of panic over a three year period in previously non-panic undergraduates; AS individuals were found to be five times more likely to develop panic. Taylor (1995) notes that this study is the only prospective non-clinical trial and lends credence to the role of AS as a dispositional factor in the aetiology of anxiety disorders.

McNally et al. (1999) investigated the associations between AS and cognitive bias for threat seen in panic disorder, in order to ascertain whether AS was associated with interpretative bias, explicit memory bias or attentional bias favouring the processing of threat information and to investigate if these factors would be pre-morbid to panic. This approach makes the assumption that 'cognitive risk for panic' is a multi-faceted construct, of which AS is one part. McNally et al. (1999) failed to find an association between AS and information processing bias in a non-clinical sample. There are a number of explanations for this result; i) AS and information processing bias are a part of panic and not a precursor to it; ii) AS is a precursor to panic (as supported elsewhere), but information processing bias is a part of panic, or iii) examination of this non-clinical sample failed to identify an existent relationship between AS and information processing bias as precursors to panic (as supported elsewhere, and posited by Clark (1986) and Reiss (1991)).

Further evidence for the role of autonomic arousal and its' appraisal in the development of post-traumatic symptoms was found by Shalev et al. (1998). In an assessment of 211 trauma victims recruited in hospital, Shalev et al. found elevated heart rates in A&E to be specifically associated with a later diagnosis of PTSD. The authors propose that early autonomic activation may be perceived by some individuals as fearful in themselves (AS). Freedman et al. (1999) suggest that such heightened responsivity, or heightened unconditional response, might be responsible for an initial step in the development of PTSD. Initial response may trigger a process of learned conditioning, which is further moderated by biological processes into a persistent and chronic arousal state (Freedman et al., 1999).

Blanchard et al. (1996) monitored heart rate and blood pressure in an experimental study with a group of 105 RTA victims, exposed to descriptions and video footage of RTA. They found that heart rate was good at distinguishing PTSD individuals from non-PTSD individuals. 37 of the 48 participants who had PTSD initially and at one year follow-up, could have their status predicted by heart rate. Furthermore, heart rate was able to predict 11 of the 16 participants who did not remit by one year, and 26 of the 33 who did (sensitivity 64.7% and specificity 83.9%). Blanchard et al. hypothesise that individuals who still experience a high heart rate on exposure to reminders of RTA may be experiencing a conditioned emotional response (Keane et al., 1985), exacerbated by AS. Alternatively, these individuals may have a more entrenched fear structure (Foa et al., 1995), a set of unelaborated, poorly discriminated and easily triggered traumatic memories (Ehlers and Clark, 2000), situationally accessible memories (Brewin et al., 1996), or a strong

associative level arousal (Daggleish, 1999). It remains to be seen whether AS is implicated in the development of such traumatic representations in memory.

In a recent study, Federoff et al. (2000) analysed the predictive role of AS and beliefs about harmful events, in the development of PTSD symptoms. The authors found AS and pain severity to be significant predictors of PTSD, and for those participants receiving cognitive-behavioural therapy for their PTSD, it was found that reductions in AS and pain severity predicted reductions in PTSD. This provides evidence for the role of AS as a significant cognitive risk factor for exacerbating and maintaining PTSD symptoms. Federoff et al. (2000) propose that many PTSD symptoms are arousal-related sensations such as palpitations, intrusive thoughts, intrusive memories, respiratory distress and hyperarousal. Individuals who believe arousal-related sensations are dangerous (AS individuals), are more likely to catastrophically misinterpret these sensations (Clark, 1986), e.g. intrusive thought misinterpreted as an impending threat of insanity, leads to greater anxiety, arousal, and a greater likelihood of thought suppression, and therefore (ironically), a greater incidence of intrusive thoughts. The authors suggest that a traumatic event, such as an RTA, may act as a conditioning episode that amplifies already existent AS beliefs. This is congruent with Ehlers and Clark's (2000) cognitive model of PTSD, which places emphasis on the importance of beliefs about symptoms in maintaining threat and distress.

Federoff et al. (2000) discuss the implications of failing to find that beliefs about harmful events were predictive of PTSD. They note that many therapies of PTSD

incorporate work on identifying and restructuring beliefs directly related to a traumatic event, such as an RTA. However, if beliefs about symptoms and arousal sensations are more powerful predictors, then treatments may be improved using methods to reduce AS. It may be that controlled exposure allows an individual to learn that arousal sensations are innocuous, explaining the success of such treatments. Federoff et al. conclude that AS is useful for assessing individuals at risk of developing PTSD and for measuring response to treatment. There is a need for prospective studies to examine the role of AS over time, in the course and chronicity of PTSD.

### Dissociation

van der Kolk et al. (1996) describe dissociation as the compartmentalisation of experience. The authors outline three levels of dissociation. Primary dissociation refers to the difficulty, during a traumatic experience, that many individuals have in integrating the totality of the event into consciousness. Sensory and emotional elements of the event may not be integrated into personal memory and identity and may remain isolated from ordinary consciousness. van der Kolk et al. (1996) suggest that in primary dissociation experience is split into its isolated somatosensory elements, without integration into a personal narrative. Brewin et al. (1996) note that traumatic events give rise to two sorts of representations; conscious, mental representations and unconscious, non-verbal, situationally accessible representations. Primary dissociation may result in formation of these situationally accessible memories (SAMS), as described by Brewin.

Secondary dissociation is defined by van der Kolk et al. (1996) as a dissociation between the observing ego and experiencing ego. Individuals report leaving their bodies during trauma and observing what happens from a distance. van der Kolk et al. (1996) suggest that this process allows pain and distress to be minimised by putting individuals out of touch with their feelings and emotions. Marmar et al. (1994), have termed this dissociative state peri-traumatic dissociation and propose that during the unfolding of traumatic events individuals will report alterations in the experience of time, place and person, which confer a sense of unreality on the event as it is occurring. The authors note that bewilderment, confusion and disorientation are commonly experienced during traumatic events and that in non-clinical samples exposure to catastrophic stress may trigger transient dissociative phenomena.

Tertiary dissociation is described by van der Kolk et al. (1996) as the trauma-induced development of distinct ego states. These ego states contain the traumatic experience and consist of complex identities with distinct cognitive, affective and behavioural patterns. Individuals experiencing tertiary dissociation states often report chronic and intense sexual, physical and psychological abuse, often starting from a very early age.

A number of studies have investigated the role of dissociation, particularly secondary/peri-traumatic dissociation, in the development of post-traumatic stress symptoms. Noyes and Kletti (1977) surveyed 101 RTA victims and assault victims. They found that 72% of respondents experienced feelings of unreality and altered experience of the passage of time during the event, 57% experienced automatic

movement, 52% a sense of detachment, 56% depersonalisation, 34% detachment from their body, and 30% of respondents experienced derealisation. Holen (1993) in a long term prospective study of survivors of a North Sea oil rig disaster, found that the level of reported dissociation during the trauma predicted subsequent PTSD. Koopman et al. (1994) examined predictors of post-traumatic stress symptoms in a group of 187 fire survivors. The authors found dissociation at the time of trauma to predict subsequent post-traumatic stress symptoms, over and above anxiety and the subjective experience of a loss of personal autonomy. Shalev et al. (1996) conducted a prospective study of 51 injured trauma survivors. They found peri-traumatic dissociation to predict 29.4% of the variance of PTSD scores at six month follow-up. Marmar et al. (1994), note that there is a growing body of evidence suggestive of an important role for peri-traumatic dissociation as a risk factor for PTSD.

Despite the evidence supportive of a role for peri-traumatic dissociation in the development of PTSD, a selection of studies have failed to identify a relationship. Barton et al. (1996) failed to illustrate the significant role of ASD in subsequent PTSD development in a group of RTA victims. As dissociative symptoms are a major part in ASD diagnostic criteria, Barton et al. assert that there is no evidence from their sample of a predictive role of dissociation in subsequent development of PTSD. The authors suggest that much of the evidence in support of a predictive role for peri-traumatic dissociation in the development of PTSD is from non-RTA samples. They propose that an RTA may not induce a level of dissociation that more severe disasters are capable of (e.g. oil rig disaster, catastrophic fire).

Therefore, their failure to illustrate the predictive role of dissociation in PTSD development with an RTA victim sample may be due to low levels of dissociation (Barton et al., 1996). It is also of importance that Barton et al. measured dissociation at the post-trauma stage, rather than peri-traumatic dissociation as in previous studies.

Brewin et al. (1999) reported further results regarding the predictive power of the ASD diagnosis for subsequent PTSD. As discussed earlier, the authors reported that overall ASD diagnosis correctly classified 88% of subsequent PTSD cases, from a sample of 157 crime victims. Of importance was Brewin et al.'s (1999) finding that dissociation at the time of trauma did not play a unique role in this prediction and that ASD diagnosis was by far the strongest precursor to subsequent PTSD. It may be that dissociative experiences, subjective levels of anxiety during trauma (Shalev et al., 1996) and initial post-traumatic stress symptoms interact to predict PTSD. Harvey and Bryant (1998) also reported results of relevance to the predictive power of dissociation in PTSD. In their prospective study of 92 RTA victims they found that although ASD was a good predictor of PTSD, there was no independent role for dissociation. The authors reported that dissociation had high negative predictive power; most non-PTSD individuals had low levels of dissociation, as did PTSD individuals. Harvey and Bryant's (1998) results concur with Brewin et al. (1999) and Barton et al. (1996), in bringing into question the independent predictive role of dissociation in the development of PTSD. It may be, as asserted by Barton et al. (1996), that the dissociation experienced by most RTA victims is too low to predict the PTSD in these samples. Finally, Harvey and Bryant

(1999) failed to find a predictive role for dissociative experiences in a group of 62 RTA victims, assessed for the severity of their ASD reactions.

Evidence for the predictive role of dissociation in the development of post-traumatic stress symptoms is mixed. What is well documented is the occurrence of varying degrees of dissociative experiences during traumatic events, including the RTA (e.g. Noyes and Kletti, 1977). Harvey et al. (1998) report evidence that dissociative experiences during trauma can disrupt the functioning of autobiographical memory, and note that difficulties in trauma memory retrieval accounted for 25% of the variance of PTSD occurrence at six month follow-up post-RTA. Cognitive conceptualisations of PTSD also emphasise the disruption of information processing during trauma (e.g. Ehlers and Clark, 2000; Brewin et al., 1996) and propose that this disruption gives rise to poorly discriminated, poorly elaborated and fragmented traumatic memory (Ehlers and Clark, 2000). It may be that peri-traumatic dissociation during certain events and for certain individuals, further disrupts processing of traumatic information to the extent that measures of dissociation are found predictive of PTSD. In other circumstances, possibly less catastrophic and fearful, dissociation may exist at low levels during trauma and may fail to be predictive of PTSD. Primary dissociation, as defined by van der Kolk (1996), may be responsible for enough cognitive disturbance during trauma to lead to the situationally accessible trauma memories proposed by Brewin et al. (1996), or the poorly discriminated and elaborated memory traces of Ehlers and Clark (2000). Secondary/peri-traumatic dissociation and a severe dislocation of person, place or time accompanied by a distancing of emotion, may not only be rare in RTA



(e.g. Barton et al., 1996) but not necessary for poor encoding of traumatic memory and the hypothesised consequences of this in the development of PTSD.

### 1.9 Principal Aims

There remains confusion in the literature regarding why some RTA victims experience prolonged psychological distress, where others do not and to what severity RTA victims experience this distress. It is the aim of the current study to further examine those hypotheses which have clear theoretical underpinnings and a growing body of empirical evidence. By employing a prospective longitudinal design, a sample of consecutive attenders to hospital and psychometrically standardised research measures, this study aims to make an important contribution to the existing literature. Additionally, the current study aims to examine driving/passenger phobia, anxiety and depression, as well as the well researched PTSD and to make an original contribution to the literature, as regards the natural history of phobic, anxious or depressive reactions to RTA (an area currently in need of investigation, Di-Gallo and Parry-Jones, 1996). The dispositional measures of AEE and AS have not been investigated in tandem, nor has AEE been applied to an RTA population previously. It is expected that applying these proven measures to the current research population will enable further confirmation of their utility in predicting potential psychological disorder. Ehlers et al. (1998) stands as the only methodologically robust illustration of the mediating role of negative interpretations, thought suppression, anger and rumination in the development of PTSD post-RTA, as hypothesised by Ehlers and Clark, (2000). The study aims to further examine this mediating role and to investigate a possible relationship

between anxiety sensitivity, negative interpretations of events and symptoms and subsequent thought suppression.

#### 1.10 Research Questions

The following research questions compliment the current literature (as reviewed) and directly inform the specific hypothesis detailed over the page.

- Do individuals involved in a minor RTA experience clinically significant psychological distress?
- Do elements of an individual's personality predict their vulnerability to developing PTSD post-RTA?
- Do elements of the RTA itself predict an individual's vulnerability to developing PTSD?
- Does the immediate post-traumatic reaction of an individual predict their vulnerability to developing PTSD in the long-term?
- Does the manner in which an individual perceives and interprets the RTA and its sequelae predict their vulnerability to developing PTSD in the long term?

- Does the manner in which an individual copes during the aftermath of an RTA (cognitively and behaviourally) predict their vulnerability to developing PTSD in the long term?

### 1.11 Hypotheses

The current study aims to address a number of specific hypotheses derived from both theoretical conceptualisations of PTSD and recent empirical evidence. The principal aims are to test hypotheses relating to the prevalence of adverse psychological reactions post-RTA and to predicting and maintaining variables implicated in such reactions. It is expected that a better understanding of who develops psychological problems post-RTA and of how and why these problems develop and persist, will aid health professionals in allocating both proactive and reactive resources to patients. Hypotheses are therefore as follows:

- It is predicted that individuals involved in an RTA will experience clinically significant post-traumatic psychological distress. RTA victims are anticipated to present with ASD at a rate congruent with previous research findings (Bryant and Harvey, 1996 = 13%; Shalev et al., 1998 = 29.9%; Ursano, 1999 = 34%). It is also anticipated that PTSD rates will be lower than ASD rates, in accordance with previous research findings (Shalev et al., 1998 = 17.5%; Ursano, 1999 = 25.2%; Feinstein and Dolan, 1991 = 25%), and according to cognitive conceptualisations of post-traumatic stress (Dagleish, 1999). RTA victims are expected to experience depressive symptoms (Shalev et al., 1998 = 19% at one month post-RTA, and

14.2% at four months post-RTA), state anxiety (Bryant and Harvey, 1996), and driving phobia related specifically to RTA (Ehlers et al., 1994 = 15%).

- It is predicted that individuals who exhibit a negative attitude towards emotional expression will experience PTSD post-RTA. A predictive role of AEE in PTSD is congruent with cognitive-behavioural explanations of information processing (Rachman, 1980; Pennebaker, 1982; Williams, 1993) and with Horowitz's conceptualisation of PTSD. Empirical evidence for a role of AEE in PTSD development (Brown and Grover, 1998; Joseph et al., 1994; Williams et al., 1994; Bryant and Harvey, 1995), is anticipated to be confirmed.
- It is predicted that individuals who exhibit high anxiety sensitivity will experience PTSD post-RTA. A predictive role of AS in PTSD is congruent with Ehlers and Clark's (2000) cognitive model, which places emphasis on the importance of beliefs about symptoms in maintaining threat. Empirical evidence for a role of AS in anxiety and panic (Ehlers, 1995; Schmitt et al., 1997; Maller and Reiss, 1992) and PTSD development (Shalev et al., 1998; Federoff et al., 2000) is anticipated to be confirmed.
- It is predicted that individuals who exhibit high peri-traumatic dissociation during and immediately after RTA, will experience PTSD. This hypothesis is informed by Ehlers and Clark (2000), whose cognitive conceptualisation of PTSD predicts that peri-traumatic dissociation will lead to a disturbance in autobiographical memory for trauma, and poorly elaborated, discriminated and contextualised memories.

Empirical evidence for a role of peri-traumatic dissociation in PTSD development (Holen, 1998; Koopman et al., 1994) is expected to be confirmed.

- It is predicted that individuals who exhibit high levels of perceived threat during and immediately after RTA, will experience PTSD. This hypothesis is congruent with a shift from a primary focus on the severity of a stressor in PTSD, to a mixture of exposure to a traumatic stressor and an individual's reaction to it, reflected in DSM-IV. Evidence for a role of perceived threat in the development of psychological distress (Green et al., 1993; Harvey et al., 1996; Bryant and Harvey, 1996; Ehlers et al., 1998) is expected to be confirmed.
- It is predicted that individuals who exhibit high levels of immediate post-traumatic symptoms will experience PTSD at three month follow-up .The hypothesis is informed by conceptualisations of PTSD that place emphasis on the importance of avoidance symptoms (Ehlers and Clark, 2000; Brewin et al., 1996; Foa et al., 1996) as initial avoidance prevents alterations in traumatic memory and in cognitive attributions of trauma and its sequelae. Empirical evidence for the role of initial symptoms in PTSD persistence (Blanchard et al., 1996; Brewin et al., 1999; Koren et al., 1999; Harvey and Bryant, 1998; Blanchard et al., 1997) is expected to be confirmed.
- It is predicted that individuals who exhibit high anxiety sensitivity will catastrophically misinterpret RTA and its sequelae negatively. This hypothesis is informed both by Reiss' (1994) expectancy theory, and Clark's (1986) cognitive

model of panic, which both predict that a fear of anxiety-related sensations will lead to misinterpretation of the autonomic arousal of anxiety, as fearful and negative. It is anticipated that empirical evidence for this relationship will be established.

- It is predicted that individuals who exhibit negative interpretations of trauma and its sequelae, will experience PTSD. A mediating role for negative interpretations in post-traumatic symptoms is congruent with Ehlers and Steil's (1995) assertion that PTSD becomes persistent when an individual interprets trauma in a way that leads to a sense of current threat. Empirical evidence for the role of negative interpretations in PTSD (Ehlers et al., 1998; Clohessy and Ehlers, 1999; Warda and Bryant, 1998) is expected to be confirmed. The role of negative interpretations of RTA and its sequelae is hypothesised to be a mediating role, between pre, peri and immediate post-traumatic predictors, and subsequent PTSD persistence. It is anticipated that further empirical evidence for a mediating role of negative interpretations will be established.
- It is predicted that individuals who exhibit high levels of anger regarding the RTA will experience PTSD. A mediating role for anger is congruent with Foa et al.'s (1995) assertion that anger acts as avoidance of feared stimuli and Andrews et al. (2000) who suggest that anger blocks the processing of fear and contributes directly to the symptoms of PTSD. Ehlers and Steil (1995), assert that anger leads to a current sense of threat post-RTA. Empirical evidence for the role of anger in post-RTA psychological distress (Riggs et al., 1992; Andrews et al., 2000; Ehlers et al., 1998) is expected to be confirmed. The role of anger is conceptualised as a

mediator between pre, peri and immediate post-traumatic predictors, and subsequent PTSD persistence. It is anticipated that further empirical evidence for a mediating role of anger will be established.

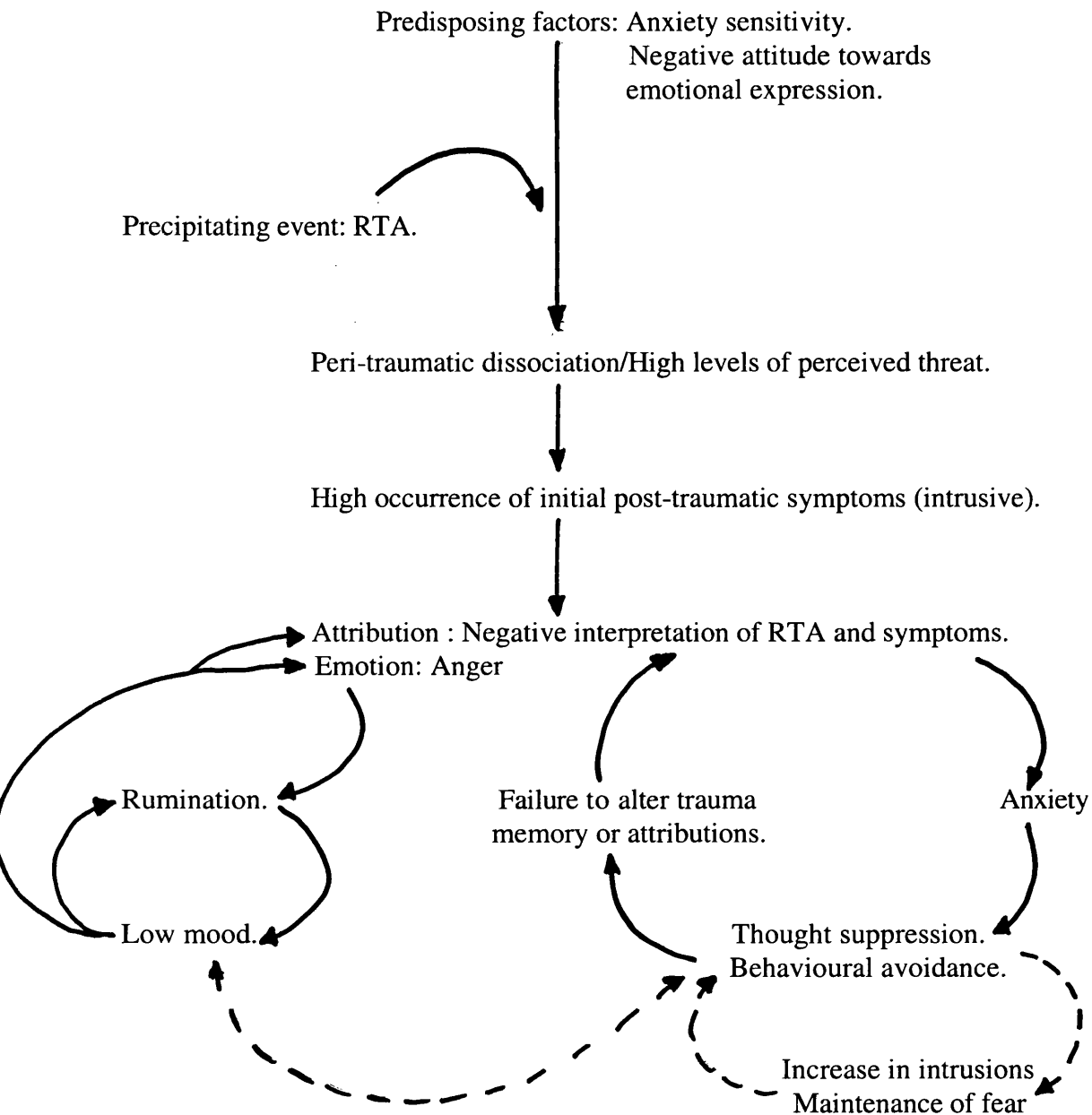
- It is predicted that individuals who engage in thought suppression post-RTA will experience PTSD. This hypothesis is informed by ironic control theory (Wegner, 1989) and by Ehlers and Steil (1995) who suggest that thought suppression will lead to long term intrusions into consciousness. Empirical evidence for a role of thought suppression in PTSD (Warda and Bryant, 1998, Harvey and Bryant, 1998; Davies and Clark, 1998; Ehlers et al., 1998) is expected to be confirmed. The role of thought suppression is conceptualised as a mediator between pre, peri and immediate post-traumatic predictors, and subsequent PTSD persistence. It is anticipated that further empirical evidence for a mediating role of thought suppression will be established
- It is predicted that individuals who engage in behavioural avoidance of driving/passenger post-RTA will experience PTSD. This hypothesis is informed by conceptualisations of PTSD that implicate avoidance in the development and maintenance of PTSD (Horowitz, 1979; Foa et al., 1995; Brewin et al., 1996; Dagleish, 1999; Ehlers and Clark, 2000). It is anticipated that empirical evidence for this relationship will be established. The role of behavioural avoidance is conceptualised as a mediator between pre, peri and immediate post-traumatic predictors, and subsequent PTSD persistence.

- It is predicted that individuals who engage in rumination post RTA will experience PTSD. This hypothesis is informed by Ehlers and Steil (1995) who suggest that rumination prevents change in the nature of traumatic memory and in trauma-related attributions. Empirical evidence for a role of rumination in PTSD (Ehlers and Steil, 1998; Ehlers et al., 1988; Dunmore et al., 1999; Clohessy and Ehlers, 1999) is expected to be confirmed. The role of rumination is conceptualised as a mediator, between pre, peri and immediate post-traumatic predictors, and subsequent PTSD persistence. It is anticipated that further empirical evidence for a mediating role of rumination will be established.
- It is predicted that individuals who exhibit negative interpretations of trauma and its sequelae will also exhibit thought suppression and rumination. This hypothesis is informed by Ehlers and Clark (2000) who suggest that negative interpretations of trauma and its sequelae will lead to anxiety and the subsequent avoidance of anxiety generating thoughts (thought suppression). Furthermore, evidence exists from the thought suppression literature that anxiety induces thought suppression (e.g. Harvey and Bryant, 1999) and from rumination literature that anxiety induces ruminative processes (e.g. Davey and Tallis, 1994). Empirical evidence for a role of negative interpretations in thought suppression (Dunmore et al., 1997:1999, Ehlers et al., 1998) and rumination (Ehlers et al., 1998) is expected to be confirmed.

The hypotheses above are summarised as a flow diagram in Figure 2. below.



Figure 2. A schematic representation of the current hypotheses.



The variables above are hypothesised to fulfil the following functions:

- Inhibition of emotional processing post-RTA: AEE, thought suppression, behavioural avoidance.
- Disruption of autobiographical memory: peri-traumatic dissociation, perceived threat.
- Maintenance of a sense of current threat: negative interpretations, rumination, anger, thought suppression, behavioural avoidance, AS.

The combination of the following factors make the current study unique in its approach;

1. Investigation of minor RTA
2. Prospective longitudinal research design
3. Psychometrically standardised measures of all dependent variables and most independent variables
4. Analysis of the prevalence and natural history of anxiety and depression
5. Examination of both prediction and maintenance of PTSD
6. Empirical analysis of the cognitive model of PTSD (Ehlers and Clark, 2000)

## **RESEARCH METHODOLOGY**

### **2.1 Design**

A prospective longitudinal research design was employed.

### **2.2 Participants**

Participants were adults aged between 16 and 65 years who had been involved in an RTA and who had subsequently attended Accident and Emergency at the Whittington Hospital, North London. Participants can have either been in cars, on motorcycles or bicycles. Individuals were excluded from the research if they had suffered a head injury or severe physical injuries. Pedestrians were also excluded from the research.

Participants (N=50) were a self-selected sample. Their age range was 18 to 62 years with a mean age of 34.70 (standard deviation = 10.42). 25 of the participants were male and 25 were female. Of the 50 participants, 47 classified themselves as White-British and the remaining three as Black-British. All participants were fully qualified drivers/motorcyclists (with the exception of participants involved in bicycle accidents). The range of years since qualification was 1 to 45 with a mean of 11.76 (standard deviation = 9.48). 56% of participants reported having been involved in a previous RTA of which the range was one to eight (mode=two previous accidents). 28% of participants indicated that they had seen a therapist or counsellor in the past.

Of the 50 participants, 48% were travelling in cars, 32% on motorcycles and 20% on bicycles. 44% of these forms of transport were 'written off' by insurance companies following the RTA. Of the remaining vehicles, 26% were described as markedly damaged and the remaining 30% as having incurred minor damaged.

Participants reported to have been alone in/on the vehicle in 90% of cases, 6% accompanied by family and 4% with friends. 18% of participants experienced bone injuries, 48% soft tissue injuries and 34% no injuries. 90% of participants reported that no one else had been injured in the RTA and 10% reported soft tissue injury to others. In response to a question 'Who was responsible (for the RTA) in your opinion?' 90% of participants indicated the third party. Only 4% attributed full responsibility to themselves. Finally, 80% of participants indicated at baseline that they had begun, or intended to initiate, litigation regarding their RTA.

There is no descriptive information available for individuals who chose not to participate in the research. Only those individuals who met the inclusion criteria (described above) were approached. Overall, 352 individuals were suitable for participation in the time-frame available and were approached regarding the research.

### 2.3 Procedures

In May 1999, following the submission of a detailed research protocol and ethics application, ethical approval for the research was granted by the Whittington Hospital (see appendices). Research procedures comply with the conditions of ethical approval.

In liaison with the Accident and Emergency department at the Whittington Hospital the primary researcher (current author) viewed computer printouts of patients who had attended following an RTA. Between June 1999 and December 1999 (seven months) the primary researcher visited the hospital weekly in order to access this information. As noted above, during this seven month period 352 individuals were assessed to have met the inclusion criteria.

All 352 individuals were written to within a week of their RTA inviting them to take part in the research, explaining its rationale and what would be involved (see appendices). Of those 352 individuals contacted, 60 responded by returning the cut-off slip on their invitation letter. This is a response rate of 17%. The sixty individuals who responded were then contacted by the primary research worker either by telephone, facsimile, e-mail or again by post, dependent on the contact information they had provided. As a result of this second round of contacts 50 individuals completed baseline assessment within one month of their RTA. The remaining ten individuals failed to complete baseline assessment within one month of their RTA and were therefore excluded.

Baseline assessments were, for the most part, conducted in person by the primary researcher either at an individuals' home, place of work or at University College London. Of the 50 baseline assessments completed, 40 were conducted in person. The remaining ten were completed by individuals in their own time and returned by post. Baseline assessment involved the collection of the following information; demographics; accident details; cognitive appraisal of the RTA and its consequences; post-RTA driving and passenger behaviour; two measures of PTSD; two measures of anxiety; one measure of depression; anxiety sensitivity; attitude towards emotional expression and peri-traumatic dissociative experiences (see Measures section for detailed account). Participants were asked to complete the assessment document themselves. The researcher monitored the participants' progress throughout the assessment. Participants were encouraged to elaborate on any questions/answers that they felt pertinent to their experience. Baseline assessments took place between July 1999 and January 2000. All participants completed a consent form after reading the information sheet regarding the research and before completing baseline assessment.

Follow-up assessments were completed three months after baseline (therefore up to a maximum of four months post-RTA). Follow-up assessment involved the collection of the following information; cognitive appraisal of the RTA and its consequences; post-RTA driving and passenger behaviour; two measures of PTSD; two measures of anxiety and one measure of depression (see Measures section for a detailed account). Of the 50 participants who completed baseline assessment 39 also completed three month follow-up. The 11 baseline participants who failed to

complete three month follow-up did not respond to either telephone, facsimile , e-mail or postal contact when contacted three months later. Follow-up assessments took place between October 1999 and May 2000. None of the 39 participants who completed both baseline and follow-up assessment were engaged in any post-traumatic psychological therapy.

## 2.4 Measures

What follows is a detailed description of the measures employed.

### Demographics

A short questionnaire (see appendices) including items pertaining to demographics, psychiatric history, history of previous accidents and trait anxiety was developed for the specific purposes of this research. The demographic items solicited information on age, gender, occupation, ethnicity and marital status. Psychiatric history was assessed by asking 'Have you ever seen a therapist or counsellor in the past?'. Participants were asked to indicate if they had had previous RTAs and if so how many. Finally, an item worded 'Do you think you are the sort of person who usually worries a lot?' was included to provide information on participants' self-perception of trait anxiety.

### RTA Information

A short questionnaire (see appendices) including items pertaining to personal injury and injury to others, damage to property, perception of responsibility for the RTA and perceived threat to ones' own and others' lives, was developed. Participants

were asked to indicate the nature and extent of injuries incurred to themselves or others and to describe any damage to property. Perception of responsibility was assessed by asking 'Who was responsible in your opinion?'. Finally, two items worded 'Did the thought go through your mind, 'this is it, I'm going to die!?' and 'Do you think others' lives were threatened?' were included to provide information on participants' perceived threat to life.

### Cognitive Appraisal

An 11 item questionnaire (see appendices) including items pertaining to cognitive appraisal of the RTA and its' consequences, was developed for the specific purposes of this research. Informed by the work of Ehlers and Steil (1995) and Ehlers et al. (1998), this measure was developed in order to assess the meaning individuals attached to their experiences. Ehlers and Steil (1995) proposed that negative interpretations of intrusive thoughts, rumination, thought suppression and anger may function to maintain a current sense of threat post-trauma. It is hypothesised that this prevents an individual from seeing trauma as an isolated negative event in the past. Ehlers et al. (1998) found that negative interpretation of intrusions, persistent medical problems and rumination at three months post-RTA predicted PTSD at 12 months. Ehlers et al. (1998) conceptualise these findings as a set of factors that appear to maintain distress post-RTA. This 11 item questionnaire therefore includes a number of possible maintaining factors derived from these theoretical considerations. The questionnaire taps information on perceived threat, pre-accident psychiatric health, anger, medical problems, financial problems, negative interpretations of intrusions, rumination and thought suppression.



Perceived threat was determined by asking participants to rate how frightening the accident was on a likert scale ranging from one (not frightening) to four (extremely frightening). Pre-accident psychiatric health was determined by asking participants whether they were suffering from emotional problems prior to the RTA on a likert scale ranging from one (not at all) to three (a lot).

Two aspects of anger were assessed; initial anger reaction and anger related to intrusive recollections. Participants were asked to indicate how angry they felt after the accident on a scale ranging from one (not at all) to four (extremely). In addition, participants were asked to indicate how angry they felt when they remembered the accident on a scale ranging from one (not at all) to four (extremely). Finally, they indicated how often they had anger-related thoughts ('Others have harmed me') when they had recollections of the accident using a likert scale ranging from one (never) to four (always).

Persistent medical problems were determined by asking participants to what extent they were physically recovered from the RTA on a scale ranging from one (fully recovered) to three (still major problems). Persistent financial problems were determined by asking participants what the financial impact of the RTA had been on a scale ranging from one (none) to three (major impact).

Negative interpretations of intrusive recollections were determined by asking participants to indicate how often they thought 'I must be going mad' or 'I'll never get over this', when having recollections of the RTA. They rated this on a likert

scale ranging from one (never) to four (always). Ehlers and Steil (1995) note that these are common examples of negative interpretations.

Two aspects of rumination were assessed. Trait worry (as described in the Demographics section), and rumination about intrusive recollections of the RTA. Participants rated how often they dwelled on memories of the accident on a likert scale ranging from one (never) to four (more than once a day). Similarly, participants rated how often they thought ‘Why did this happen to me?’ when recollections occurred, on a likert scale ranging from one (never) to four (always). The rumination about recollections score was the mean of these two items.

Thought suppression was determined by asking participants to indicate how often they pushed memories of the RTA out of their mind when they occurred. A likert scale ranging from one (never) to four (always) was used.

#### Driving and Passenger Behaviours/Cognitions

A 16-item questionnaire (see appendices) designed to assess driving and passenger phobia was developed for the specific purposes of this research. This measure was derived from, and designed around, the work of Kuch et al. (1991, 1994). Kuch et al. define the construct known as accident phobia as follows; accident phobia has three features, i) DSM diagnosable simple phobia, ii) onset and content of this phobia related specifically to the RTA, and iii) anxiety symptoms and avoidance behaviours centred on excessive fears of repetition of the accident. All items on the questionnaire are measured by either a ‘Yes’ or ‘No’ answer.

Anxiety symptoms were determined by asking participants i) are you anxious about travelling in cars?, ii) when you are driving do you experience any physical signs of anxiety, such as muscular tension, quick-shallow breathing, palpitations or sweating?, iii) do you find you feel anxious at the thought of travelling in a car? and iv) when you are a passenger do you experience any physical signs of anxiety, such as muscular tension, quick shallow breathing, palpitations or sweating?

Avoidance behaviours/cognitions were determined by asking participants i) have you driven since the accident?, ii) do you avoid driving when it is not essential?, iii) do you avoid driving under certain conditions, such as at night, on certain roads, in the wet or in heavy traffic?, iv) have you been a passenger in a car since the accident?, v) do you avoid being a passenger when it is not essential?, vi) do you avoid being a passenger under certain conditions, such as at night, on certain roads, in the wet or in heavy traffic?, vii) do you distract yourself when travelling as a passenger? and viii) do you find yourself back seat driving?

Excessive fear of repetition of the RTA was determined by asking participants i) when you are driving are you worried another accident will happen?, ii) do you think the probability of another accident occurring is high?, iii) when you are a passenger are you worried another accident will happen?, and iv) when you are a passenger do you think the probability of another accident occurring is high?

Taylor and Koch (1995) note that this definition (Kuch et al. 1991;1994), of accident phobia requires that the phobic stimulus be avoided or endured with

intense anxiety or distress. Complete driving avoidance is therefore not required. The authors suggest that accident phobia may be underestimated when driving avoidance is used as the sole criteria. Hence, anxiety symptoms and excessive fears are included in our measure.

### Impact of Event Scale–Revised

The original Impact of Event Scale (IES) was developed as a measure of the emotional sequelae of extreme stress by Horowitz et al. (1979). This 15-item measure describes emotional reactions to a traumatic event to which the respondent is asked to indicate on a four-point scale, ranging from zero (not at all) to five (often), how frequently each reaction has been experienced in the last week. The IES is used to yield a seven item measure of intrusion and an eight item measure of avoidance, as regards traumatic stimuli.

The Impact of Event Scale–Revised (IES-R) was developed by Weiss and Marmar (1997) and contains all original 15 items from the IES. The IES-R was devised as a self-report measure of the three broad domains of response to traumatic stress; intrusive phenomena, avoidant phenomena and hyperarousal phenomena. The seven new items pertaining to hyperarousal are what differentiates the IES from the IES-R. Weiss and Marmar (1997) note that the IES and IES-R are not derived from a narrow theoretical orientation, but from observation of stress response. Weiss also asserts that the IES and the IES-R are comparable due to the overlap of 15 items and two of the three factors.

Internal consistency of the IES has been determined in many psychometric reviews. Horowitz et al. (1979) reported high internal consistency of the intrusion and avoidance subscales (Cronbach's alpha coefficient for intrusion=.79, and for avoidance=.82). Weiss et al. (1995) reports data from a study of 439 emergency service workers using the IES-R. Cronbach's alpha coefficients were stated as .85 (intrusion), .85 (avoidance) and .77 (hyperarousal).

Briere and Elliot (1998) report on the concurrent validity of the IES. In a non-clinical sample of 505 members of the general public, asked to report on a recent upsetting event, the authors assessed the generalisability of the IES across instruments. They report high concurrent validity when the IES is compared to the Trauma Symptom Inventory (TSI) and the Los Angeles Symptom Checklist (LASC).

The generalisability of the IES-R across occasions (test-retest) is reported on by Weiss et al. (1995). Weiss et al. describe a study of 88 victims of the Northridge Earthquake and reports test-retest reliability as .47 (intrusion), .40 (avoidance) and .51 (hyperarousal). Figures from a study of 318 emergency workers are also reported, with test-retest reliability as .56 (intrusion), .51 (avoidance) and .59 (hyperarousal).

The ability of the IES to accurately predict PTSD has been addressed by a number of authors. Feinstein and Dolan (1991) report that in their study IES scores one week post-trauma were highly predictive of PTSD at four months post-trauma.

These authors report sensitivity of this prediction (the rate of true positives) and specificity (the rate of true negatives) as 100% and 87% respectively. Shalev et al. (1996) however, report less convincing results. They found initial IES scores to be predictive of follow-up PTSD with a sensitivity of 92.3% and specificity of 34.2%. Shalev et al. (1996) conducted a Receiver Operator Characteristic analysis (ROC) on the IES and other psychological measures. ROC analysis allows measures to be compared with each other in terms of their predictive power. Shalev et al. report that the IES is significantly better than chance at predicting PTSD both at one week, and one month post-trauma. However, the IES did not perform significantly better than non-trauma measures (State-Trait Anxiety Inventory–Spielberger, 1983) in predicting PTSD. Shalev et al. (1996) suggest that both trauma and non-trauma measures in their study were assessing a general level of psychological distress. The original aim of the IES was to pick up distress characteristic of PTSD, not to diagnose it. For the purposes of the current research IES is not seen as an indicator of PTSD diagnosis.

#### Self-Rating Scale for PTSD

The Self-Rating Scale for PTSD (SRS-PTSD: DSM-IV) was developed by Carlier et al. (1998). It was designed as an abridged version of the Structured Interview for PTSD (SI-PTSD: DSM-IV) which measures the presence and severity of PTSD symptoms from both a current and lifetime perspective. The 17 items of the SRS-PTSD correspond to the diagnostic criteria for PTSD (DSM-III-R: APA, 1987) and cluster around three factors; re-experiencing, avoidance and hyperarousal. Carlier et al. (1998) report the results of a principal component analysis to be consistent

with the sub-grouping of items into re-experiencing, avoidance and hyperarousal. These three factors together explained 57% of the variance. There are four items pertaining to re-experiencing, seven items pertaining to avoidance and six items of hyperarousal. Scoring of each then ranges from zero to two, an item being rated as 'present' if scored at one or above. Respondents meet diagnosis for PTSD if they have at least one re-experiencing item, three avoidance items and two hyperarousal items endorsed as present.

Internal consistency is reported by Carlier et al. (1998) for the 17 items of the SRS-PTSD. They found Cronbach's alpha coefficient of .96. Additionally, the separate factor clusters exhibited good internal consistency (re-experiencing=.88, avoidance=.88 and hyperarousal=.93).

Carlier et al. (1998) report on the concurrent validity of the SRS-PTSD. They report the results of Pearson's correlation coefficients comparing the SRS-PTSD with the SI-PTSD. Correlations for overall score were  $r(136)=.89, p<.001$ , for re-experiencing  $r(136)=.72, p<.001$ , for avoidance,  $r(136)=.72, p=.001$ , and for hyperarousal  $r(136)=.85, p<.001$ .

The SRS-PTSD was assessed by the same authors for its' generalisability across raters (inter-rater reliability). Two independent raters reviewed data regarding PTSD diagnosis on the basis of the SRS-PTSD. Excellent agreement was found between raters (Cohen's  $K = .98$ ).

The stated ability of the SRS-PTSD to predict diagnosis of PTSD as determined by structured interview was also determined by Carlier et al. (1998). Using the SI-PTSD as a comparison the authors found the sensitivity of the SRS-PTSD to be 86% and specificity to be 80%. Specificity and sensitivity were combined to form a likelihood ratio of the test (Likelihood Ratio Positive=4.30; Likelihood Ratio Negative=.18). This was taken as an indicator of good predictive power of the SRS-PTSD in predicting DSM diagnosable PTSD.

### Hospital Anxiety and Depression Scale

The Hospital Anxiety and Depression Scale (HADS) was developed by Zigmond and Snaith (1983). It was originally designed for use in general hospitals and consists of two subscales, one measuring anxiety (A-Scale) and one measuring depression (D-Scale), which are scored separately. The HADS was designed as a present-state measure and consists of seven anxiety items and seven depression items. Zigmond and Snaith (1983) designed the A-Scale to measure the state of anxious mood, restlessness and anxious thoughts. It is of importance to note that the many somatic symptoms of anxiety are not reflected in the HADS A-Scale. Turner and Lee (1998) suggest that since many trauma victims experience physical pain and other somatic complaints, the HADS is useful in not including these factors as indicators of psychological disorder. The D-Scale of the HADS is focused largely, but not entirely, upon the state of loss of interest and diminished pleasure response. Turner and Lee (1998) note that this lowering of hedonic tone is recognised as a reliable guide for biologically originating depression. Each of the items on the HADS is scored on a likert scale from zero to three. The maximum



score in each subscale is therefore 21. Zigmond and Snaith (1983) propose the following for interpreting scores; 0–7 normal range, 8–10 mild, 11–14 moderate, 15–21 severe.

Internal consistency was determined by Zigmond and Snaith (1983) on data from 50 respondents. The authors found Cronbach's alpha coefficient of .76 for the A-Scale and .60 for the D-Scale. Moorey et al. (1991) report data from a study of 568 individuals suffering from cancer. They report Cronbach's alpha coefficients of .93 for the A-Scale, and .90 for the D-scale.

The concurrent validity of the HADS was determined by Zigmond and Snaith (1983). In a comparison with a five-point psychiatric rating scale of anxiety and depression, using 100 medical outpatients, the authors report significant correlations of .54 for the A-Scale and .79 for the D-scale. In their review Turner and Lee (1998) note that the concurrent validity for the HADS has also been reported in psychiatric patients (Bramley et al., 1988), in a heterogeneous group of patients with physical illness (Aylard et al., 1987), and in patients attending a genito-urinary clinic (Barezak, 1988).

Zigmond and Snaith (1983) report on the generalisability of the HADS across occasions (test-retest) in a group of healthy respondents. They report significant correlations over time of .89 for the A-Scale and .92 for the D-Scale.

The construct validity of the HADS was assessed by Moorey et al. (1991) using 568 cancer patients. The authors conducted a factor analytic study and found there to be two separate factors of anxiety and depression, as asserted by Zigmond and Snaith 1983). These two factors accounted for 53% of the variance. Leung et al. (1993) and Bedford et al. (1997) report bi-dimensional factors of anxiety and depression, in agreement with Moorey et al. (1991).

### Beck Anxiety Inventory

The Beck Anxiety Inventory (BAI) was developed by Beck and Steer (1987) as a measure of state anxiety. The BAI is a 21 item self-report measure comprising 14 items pertaining to somatic aspects of anxiety and seven items reflecting subjective aspects of anxiety, such as fears. The measure is rated on a four point scale ranging from zero to three and respondents are asked to refer to the past week, including today, when answering each item. The BAI does not contain items reflecting avoidance and phobia. Beck and Steer (1987) designed the BAI with the explicit intention of making it highly discriminated from depression measures (particularly the 21 item Beck Depression Inventory).

Beck and Steer (1999) review the psychometric evidence regarding the BAI, in both clinical and non-clinical populations. The authors suggest that studies have generally found high internal consistency (Cronbach's alpha coefficient >.90). Beck and Steer also report adequate test-retest reliability ( $r_s >.60$ ) for the BAI. Creamer et al. (1995) assessed the BAI using a sample of 326 undergraduate students and report high internal consistency (Cronbach's alpha coefficient .91).

The concurrent validity of the BAI is reported on by Beck and Steer (1999). They note that a number of studies have investigated this and report moderate to high concurrent validity ( $r > .50$ ) with other self-report and clinical rating scales.

The construct validity of the BAI has been a topic worthy of extensive investigation. As noted above, Beck and Steer (1987) designed the BAI around two factors; somatic anxiety and subjective anxiety. Beck et al. (1988) conducted a principal component analysis and found the BAI to reflect two correlated ( $r = .56$ ) dimensions in a normative sample. The first factor was somatic and the second subjective and psychological. Creamer et al. (1995) found that a normal sample of undergraduates failed to exhibit the bi-dimensional factor structure of the BAI, when a principal component analysis was conducted. This same sample, when under stress, however, did exhibit the two factor structure asserted by Beck. Additionally, Hewitt and Norton (1993) found a bi-dimensional factor structure (somatic/cognitive) in the BAI. This fits with conceptualisations of anxiety where somatic and cognitive elements are important (e.g. Barlow, 1988).

There remain some discrepancies around the bi-dimensional factor structure of the BAI. Cox et al. (1996) conducted a factor analysis using the BAI and found three factors; dizziness, catastrophic cognitions/fear and cardiorespiratory distress. They report that these three factors are highly correlated with factors comprising the Panic Attack Questionnaire (PAQ), Norton et al. (1986) and highlight similarities between BAI items and DSM-IV criterion for panic disorder. Cox et al. (1996)

propose that the heavy physiological loading (14 items; Beck and Steer, 1987) on the BAI mean it is compounded with, or actually measures, panic attacks.

Lang (1971) suggests that anxiety is made up of four components; affective, cognitive, behavioural and physiological and therefore that psychometric attempts to measure anxiety should address all four components. Creamer et al. (1995) note that in an attempt to maximise discrimination from depression, the BAI has dispersed with elements of construct validity (anxious affect and behaviour). The BAI may therefore underestimate anxiety in individuals who show low levels of physiological anxiety, in the context of high levels of behavioural avoidance and affective distress. In the context of the current research, the BAI complements the non-somatic HADS A-Scale.

### Anxiety Sensitivity Index

The Anxiety Sensitivity Index (ASI) was developed by Peterson and Reiss (1987). The ASI was designed around and directly influenced by Reiss' (1991) expectancy theory of anxiety sensitivity. The theory posits that anxiety sensitivity is a dispositional fear of anxiety related sensations, which arises from beliefs that these sensations have harmful somatic, psychological or social consequences. Reiss proposes that anxiety sensitivity amplifies fear and anxiety reactions and plays an important role in the aetiology and maintenance of anxiety disorders. The ASI is a 16 item self-report measure that assesses two aspects of anxiety sensitivity; beliefs about the dangerousness of anxiety sensations and fears of these sensations.

Respondents complete the ASI by rating the extent to which they agree with items on a scale ranging from 1= very little, to 5= very much.

Psychometric analysis of the ASI has been concentrated on assessing the factor structure of the measure. There has recently been much discussion in the literature regarding the unifactoral vs. multifactoral structure of the ASI. Taylor (1995) proposes that the ASI is unifactoral at the higher order level (a high order anxiety sensitivity factor according to Reiss' definition) and that three lower order factors load onto this higher order factor (multifactoral at the lower level). Taylor suggests that in accordance with Reiss these lower order factors are the three consequences of anxiety; i) somatic harm (e.g. death); ii) psychological harm (e.g. insanity) and iii) social harm (e.g. ostracism).

Zinbarg et al. (1997) recommend formalising the three lower order factors into three ASI subscales for clinical and research purposes. These authors determined concurrent validity by comparing profiles of subscales with profiles of participants who had different principal anxiety disorder diagnoses. The Panic Disorder group attained significantly higher scores than did each of the other participant groups on the ASI somatic fears subscale. The highest mean on the ASI social fears subscale was attained by the social phobia group.

### Attitudes towards Emotional Expression

The Attitudes towards Emotional Expression measure (AEE) was developed by Joseph et al. (1994). The AEE was designed in the context of research indicating that inhibited emotional expression constitutes a vulnerability for psychological disorder following trauma (Williams, 1993). The AEE is a 20 item self-report measure assessing cognitions (beliefs about emotional expression) and behaviours (tendencies to act in certain ways regarding emotional expression). Respondents are asked to indicate how they endorse each item on a scale ranging from 1=disagree very much, to 5=agree very much.

Joseph et al. (1994) made a preliminary investigation of the internal consistency of the AEE. The authors report high internal consistency (Cronbach's alpha coefficient=.90) in a sample of 180 undergraduates. Joseph et al. conducted a principal component analysis in order to examine further the internal consistency and factor structure of the AEE. They propose that at the higher order level the AEE is unifactorial with four lower order factors loading onto this higher order factor. The four factors are as follows; i) behavioural style (e.g. 'when I am upset I bottle up my emotions'); ii) beliefs about meaning (e.g. 'turning to someone else for advice or help is a sign of weakness'); iii) beliefs about expression (e.g. 'I should always have complete control over my emotions'), and iv) beliefs about consequences (e.g. 'my bad feelings will harm other people if I express them'). All four subscales were positively associated (lowest  $r=.33$ ,  $p<.001$ ) suggesting a higher order AEE factor. Each subscale had loadings higher than .47 on their respective factors and lower than .46 on all other factors, confirming the assertion by Joseph et

al. (1994) that the subscales represent different constructs. The authors report the following Cronbach's alpha coefficients for each subscale factor; i) behavioural style =.88, ii) beliefs about meaning=.85, iii) beliefs about expression=.82 and iv) beliefs about consequences=.70.

Concurrent validity (generalisability across instruments) was preliminarily determined by Joseph et al. (1994). They examined the association between the AEE and the Ways of Coping Questionnaire (Folkman and Lazarus, 1988), a six item scale which describes efforts to seek informational, tangible and emotional support. With their sample of 180 undergraduates the authors report good convergent validity ( $r = .53, p < .001$ ) indicating that high scores on the AEE were associated with low social support seeking. There remains a need for further validation when comparing AEE to behavioural measures of inhibition. Similarly, further information regarding test-retest reliability and the predictive validity of the AEE are needed.

#### Peri-Traumatic Dissociative Experiences Questionnaire

The Peri-Traumatic Dissociative Experiences Questionnaire (PDEQ) was developed by Marmar et al. (1994). The PDEQ is a ten item self-report measure designed to assess acute dissociative responses at the time the traumatic event is unfolding. Marmar et al. (1994) describe peri-traumatic dissociation as an alteration in the experience of time, place or person, conferred as a sense of unreality on the event as it occurs. They note that bewilderment, confusion and disorientation are commonly experienced during traumatic events and that in non-clinical samples exposure to

catastrophic stress may trigger transient dissociative phenomena. The PDEQ comprises items measuring derealisation, amnesia, out of body experience and altered time perception. It is rated by respondents on a scale from 1=not at all true, to 5=extremely true, in relation to a specific traumatic event.

There has to date been little psychometric evaluation of the PDEQ. Internal consistency was determined by Marmar et al. (1994) in a study of 251 Vietnam veterans (retrospective). The authors report item to scale correlations ranging from .41 to .56, and Cronbach's alpha coefficient=.80. Shalev et al. (1996) report item to scale correlations ranging from .31 to .78, and Cronbach's alpha coefficient=.79. In the only prospective study involving the PDEQ to date Shalev et al. (1996) report item to scale correlations ranging from .31 to .62, Cronbach's alpha coefficient=.77.

Marmar et al. (1996) studied 367 emergency service workers using the PDEQ and the Dissociative Experience Scale (DES), Bernstein and Putnam (1986). They found the two measures to be strongly associated and predictive of PTSD. In a study of 77 female Vietnam veterans Tichenor et al. (1994) also report a strong association between the PDEQ and DES. Marmar et al. (1996) extrapolate from these results that the PDEQ exhibits both high internal consistency and strong concurrent validity. There is a need for further psychometric assessment of the PDEQ particularly regarding its' generalisability across measures, test-retest



reliability and predictive validity. A principal component analysis would provide useful information regarding construct validity.

## **RESULTS**

The results chapter aims to describe the statistical procedures undertaken and the subsequent output of these procedures. First, data preparation will be described. Second, prevalence results are presented for both dependent and independent variables. Third, results of multivariate statistics addressing the prediction of PTSD symptoms are presented. Fourth, further analysis of the mediation of predictive relationships is reported on. Finally, a summary of main findings is presented.

### **3.1 Data Preparation**

The normality of distribution around the mean was analysed for all variables. It was found that a number of variables exhibited unacceptable skewness and kurtosis for the planned parametric statistical analysis. Square root transformations were performed in order to correct these distributions in most cases. For the independent variables of anxiety sensitivity (ASI) and attitudes towards emotional expression (AEE) it was clear that a normal distribution was disrupted by an outlier (participant number 10 in both cases). It was judged that the extremely high scores on these measures were important to include in the analysis, as the theoretical underpinnings of the study anticipate them to be important predictors of PTSD. Therefore, rather than exclude them from parametric analysis, these scores were reduced artificially to one point higher than the next-highest-participant-score, in accordance with procedures outlined by Hirsch and Riegelman (1996). Table 1. below shows the results of the analysis of normality and the alterations in z-statistics resulting from transformations.

Table 1. Results of the analysis of normality and subsequent transformations; z-statistic < 1.96 represents a normal distribution (Hirsch and Riegelman, 1996).

Variable	Skewness	Kurtosis	Transformed Skewness	Transformed Kurtosis
AEE	3.76	7.03	0.19	0.88
Anger (B)	1.15	-0.51		
Anger (F)	1.59	1.16		
ASI	2.75	2.15	1.07	-0.90
BAI (B)	4.65	3.04	1.19	-0.91
BAI (F)	5.28	4.61	2.11	0.10
Neg. Int. (B)	4.32	2.07	3.37	0.34
TS (B)	2.52	-1.11	1.89	-1.63
Neg. Int. (F)	5.03	3.70	4.51	2.26
TS (F)	3.04	0.59	2.24	-0.55
Driv. (B)	0.45	-1.40		
Driv. (F)	0.58	-1.89		
HAD ANX (B)	1.83	-0.67		
HAD DEP (B)	4.11	2.19	0.91	-1.59
HAD ANX (F)	1.75	-0.63		
HAD DEP (F)	4.41	2.53	2.05	-0.85
IES AVOID (B)	2.35	-1.00	0.26	-2.01
IES HYP (B)	2.77	0.03	-0.17	-1.00
IES INTR (B)	3.33	2.19	0.32	0.20
IES TOTAL (B)	2.44	0.08	-0.26	-0.75
IES AV/IN (B)	2.18	-0.27	-0.39	-0.91
IES AVOID (F)	3.83	1.24	.62	-1.12
IES HYP (F)	3.94	1.15	1.99	-0.81
IES INTR (F)	3.46	0.49	1.42	-0.91
IES TOTAL (F)	3.84	0.88	-1.68	-0.22
IES AV/IN (F)	3.78	0.93	1.35	-0.60
Pass. (B)	1.26	1.59		
Pass. (F)	2.09	-0.89		
PDEQ.	0.60	-1.13		
P. Threat	3.96	2.56	2.07	0.04
RUM (B)	2.28	-0.13	1.24	-0.70
RUM (F)	3.26	1.09	2.32	0.05
Prev. Acc.	4.07	1.91	1.11	1.79
Property	0.82	2.41		
SRS AVOID (B)	4.86	3.63	1.00	-0.94
SRS HYP (B)	2.62	-0.60	0.11	-1.72
SRS INTR (B)	1.23	-0.60		
SRS TOTAL (B)	3.43	1.41	0.32	0.06
SRS AVOID (F)	4.64	2.50	2.26	1.34
SRS HYP (F)	5.22	4.86	2.20	-0.45
SRS INTR (F)	4.08	3.11	0.23	-0.29
SRS TOTAL (F)	4.49	2.92	1.86	-0.13
Prior prob.	3.73	0.94	3.15	-0.31
Phys. prob. (B)	0.03	0.75		
Fin. Prob. (B)	-0.97	-1.54		
Phys. Prob. (F)	0.42	-0.58		
Fin. Prob (F)	-0.79	-1.36		

Internal consistencies (Cronbach's alpha) were calculated for all measures used on this population and are as follows;

AEE = .90  
Anger = .56  
ASI = .74  
BAI = .95  
Driving Questionnaire = .73  
HAD ANX = .86  
HAD DEP = .90  
IES-total score = .94  
IES-avoidance and intrusion subscales = .92  
PDEQ = .85  
Perceived threat = .38  
Passenger Questionnaire = .85  
SRS PTSD = .91

Anger and perceived threat are the only variables with less than acceptable internal consistency.

### 3.2 Prevalence Rates of Dependent Variables

#### PTSD

According to the SRS-PTSD:DSM-IV the following diagnostic results were obtained from the population; baseline (N=49), 14.3% of individuals met DSM-IV diagnostic criteria, follow-up (N=39), 12.8% of individuals met DSM-IV diagnostic criteria.

Analysis of the group of participants who completed baseline assessment but did not respond to follow up (drop-out group) indicated that 42.8% of those individuals diagnosed with PTSD at baseline dropped out.

#### Psychological Distress Characteristic of PTSD

According to the IES-R the following levels of psychological distress were identified in the population. Intrusion and avoidance subscale totals have been added in order to make use of existing normative data available for the IES. The

number of participants at baseline was 50 and at follow-up 39. The raw scores for the added intrusion and avoidance subscales, at both baseline and follow-up, covered the range described in the normative data (<40<sup>th</sup> percentile to >99<sup>th</sup> percentile; Briere and Elliot, 1998). Of the population, 46% at baseline and 23.2% at follow-up scored 'moderately' (IES  $\leq$  20) and 24% at baseline and 17.9% at follow-up scored 'highly' (IES  $\leq$  35). These clinical cut-off points were taken from Robbins and Hunt, 1996; Bryant and Harvey, 1996. The mean score for the intrusion and avoidance subscales added was 22.26, standard deviation=17.55 at baseline and 14.82 (standard deviation=18.57) at follow-up.

The mean score for the intrusion and avoidance sub-scales added in the drop-out group was 21.54, standard deviation=16.65.

The Hyperarousal subscale of the IES-R had a mean score of 10.88, standard deviation=8.38 at baseline. At follow-up mean=5.90, standard deviation=9.32.

The total score of the IES-R (intrusion+avoidance+hyperarousal) had a mean=33.04, standard deviation=25.70, at baseline. At follow-up, mean score=20.72, standard deviation=26.52.

### Anxiety

The HADS anxiety subscale has a maximum score of 21. Zigmond and Snaith (1983) suggest scores of 0-7 are normal range, 8-10 mild, 11-14 moderate and 15-21 severe. At baseline, 64% of the population scored in the normal range on the HADS anxiety. Of those 36% whose scores were clinically significant, 12% had mild anxiety, 18% moderate anxiety and 6% severe anxiety. At follow-up, 66.7% of the population scored in the normal range. Of the 33.3% whose scores were clinically significant, 15.4% had mild anxiety, 12.8% moderate anxiety and 5.1% severe anxiety, (baseline N=50, follow-up, N=38).

The BAI has a maximum score of 63. Beck and Steer (1987) suggest scores of 0-7 represent minimal anxiety, 8-15 mild anxiety, 16-25 moderate anxiety and 26-63 severe anxiety. At baseline (N=50), 60% of the population scored 'minimally'. Of the 40% whose scores were clinically significant, 18% had mild anxiety, 10% had moderate anxiety and 12% had severe anxiety. At follow-up (N=39), 73.7% of the population scored in the minimal range. Of those 26.3% whose scores were clinically significant, 18.1% had mild anxiety, 27% moderate anxiety and 10.5% severe anxiety.

### Depression

The HADS depression scale has the same scoring system as the HADS anxiety scale, and utilises the same clinical cut-offs (Zigmond and Snaith, 1983). At baseline (N=50), 82% of the population scored in the normal range on the HADS depression scale. Of the 18% whose scores were of clinical significance 8% had mild depression, 6% moderate depression and 4% severe depression. At follow-up (N=39), 79.5% of the population scored in the normal range. Of the 20.5% whose scores were clinically significant, 10.2% had mild depression, 2.6% moderate depression and 2.7% severe depression.

### Behavioural Avoidance

Behavioural avoidance refers to individual's reporting having avoided using their car, motorcycle or bicycle following RTA, at both baseline and three month follow-up. 22.9% of the population engaged in behavioural avoidance at baseline (N=50), and 15.8% at follow-up (N=39).

### Fear of Driving/Riding

As described in the research methodology chapter, a questionnaire investigating i) anxiety about travelling in cars/on bikes, ii) symptoms of anxiety whilst driving/riding, and iii) avoidance of cars/bikes, was devised based on the work of Kuch et al. (1991). This measure is therefore unstandardised and shall be reported on by way of item by item analysis. At baseline (N=43), 67.4% of the population endorsed an item relating to worries that another accident would occur. At follow

up assessment (N=36), 66.7% of the population endorsed this item relating to worry. A separate item relating to anxiety about travelling in cars/on bikes was endorsed by 51% of the population at baseline (N=49) and 42.1% at follow-up (N=38). Items relating to avoidance were endorsed at a lower rate 31.8% / 30.2% baseline, and 35.1% / 43.2% follow-up. Symptoms of anxiety whilst driving were reported by 37.2% at baseline and 36.1% at follow up.

### Fear of Being a Passenger

As described in the research methodology chapter, a questionnaire investigating passenger behaviour was developed. This was based on work by Kuch et al. (1991). This is an unstandardised measure and shall therefore be analysed item-by-item.

At baseline (N=38), 57.9% of the population endorsed an item relating to 'back seat driving'. At follow-up assessment (N=37), 54.1% endorsed this item. An item relating to worries that another accident will occur whilst being a passenger was endorsed by 50.0% of the population at baseline (N=38) and 48.6% at follow-up (N=37). Measures of avoidance and anxiety symptoms were endorsed at rates below 40%.

## 3.3 Prevalence Rates of Independent Variables

### Perceived Threat

As described in the research methodology chapter, perceived threat was measured by three separate unstandardised items. These items were then grouped to produce a measure of perceived threat which was measured at baseline. Raw scores at baseline (N=50), had a mean of 8.42, standard deviation=2.17 (maximum score=10; minimum score=1). 48% of the population scored <2 raw score indicating extremely low levels of perceived threat. 24% of the population scored between 5 and the maximum 10 raw score indicating moderate to high levels of perceived threat.

### Anger

As described in the research methodology chapter, anger was measured by three separate unstandardised items. These items were then grouped to produce a measure of anger which was measured at both baseline and follow-up. Raw scores at baseline (N=50), had a mean of 6.42, standard deviation=2.25 (maximum score=12; minimum score=3). 10% of the population scored the minimum 2, and 34% scored <5. 54% of the population scored between 5 and 10, and only 4% scored 11-12 raw score for anger at baseline. At follow-up (N=38) the population mean score was 6.05, standard deviation=2.00. 39.5% scored <5, 57.8% 5-10, and 2.6% between 11 and 12 for anger at follow-up.

### Prior Emotional Problems

At baseline an assessment of individuals' emotional status prior to the RTA was conducted, as described in the research methodology chapter. 66% of the population reported to be suffering from no emotional problems prior to their RTA, 28% to be suffering 'a little' and 6% 'a lot' (N=50).

### Physical Recovery from RTA

The extent to which the population were recovered from their RTA injuries was measured both at baseline (N=50) and follow-up (N=38). At baseline, 16% of the population reported no physical problems related to RTA, 70% reported minor problems and 14% major problems. At follow-up, 34.2% reported no problems, 57.9% minor problems and 7.9% major problems.

### Financial Impact

The financial impact of RTA was measured at baseline (N=50) and follow-up (N=38). At baseline 18% of the population reported to have suffered no financial impact of RTA, 44% a minor impact and 38% a major impact. At follow-up 18.4% reported no impact, 4.7% minor impact and 36.8% major impact.



### Negative Interpretation of Symptoms

The frequency with which the population negatively interpreted their post-traumatic recollections was measured at baseline (N=50) and follow-up (N=38). At baseline, 60% of the population reported never to negatively interpret recollections, 26% reported that they sometimes did, 8% often and 6% always. At follow-up, 76.3% reported never to negatively interpret recollections, 10.5% sometimes, 10.5% often and 2.6% always.

### Rumination

As described in the research methodology chapter, rumination was measured by two separate unstandardised items. These items were then added to produce a total for rumination which was measured at baseline and follow-up. Raw scores at baseline (N=50), had a mean of 4.18, standard deviation=1.57 (maximum score=8, minimum score=2). 10% of the population scored the minimum 2, and 68% scored <4 (low). 28% scored between 5 and 7 and only 4% of the population scored the maximum 8. At follow-up (N=38), the population mean score was 3.66, standard deviation=1.71. 26.8% of the population scored the minimum 2, 78.9% scored <4 (low) and only 15.9% scored between 5 and 7, with 5.3% scoring the maximum 8.

### Thought Suppression

The frequency with which the population suppressed thoughts of the RTA was measured at baseline (N=50) and follow-up (N=38). At baseline, 48% of the population reported never to thought suppress regarding their RTA, 26% reported that they sometimes did, 8% often and 18% always. At follow-up, 52.6% of the population reported never to thought suppress regarding their RTA, 18.9% sometimes, 10.5% often and 7.9% always.

### Anxiety Sensitivity

Anxiety sensitivity was measured using the ASI at baseline (N=50). The maximum score on the ASI is 80 and the minimum score 16. The population recorded a mean score of 32.76, standard deviation=11.47. The range of scores was 16 to 72.

### Attitude Towards Emotional Expression

Attitude towards emotional expression was measured using the AEE at baseline (N=50). The maximum score on the AEE is 100 and the minimum 20. The population recorded a mean score of 49.88, standard deviation=12.15. The range of scores was 24 to 99.

### Peri-Traumatic Dissociation

Peri-traumatic dissociation was measured using the PDEQ at baseline (N=49). The maximum score on the PDEQ is 50 and the minimum 0. The population recorded a mean score of 23.53, standard deviation=9.54. The range of scores was 2 to 42.

### 3.4 Prediction of Psychological Distress Characteristic of PTSD

The Impact of Event scale was developed as a measure of post-traumatic psychological distress (see research methodology chapter). As outlined in the research hypotheses and questions, it was anticipated that a number of the independent variables measured would predict psychological distress at follow-up. Preliminary correlational analysis indicated that the following independent variables were not significantly related to the impact of events scale at follow-up (IES-F) and were therefore not controlled for in the multiple regression analyses;

Injury to others:  $r=-.148$ ,  $p=.368$

Litigation:  $r=-.156$ ,  $p=.342$

Responsibility for the accident:  $r=-.270$ ,  $p=.095$

Anger:  $r=.148$ ,  $p=.369$

Perceived threat:  $r=.255$ ,  $p=.116$

Behavioural avoidance:  $r=-.220$ ,  $p=.184$

Damage to property:  $r=-.202$ ,  $p=.215$

Injury to self:  $r=-.059$ ,  $p=.721$

A series of multi-variate analyses were completed using the Impact of Event scale (intrusion and avoidance subscales) as the dependent variable. What follows are the results of multiple regression analyses carried out to determine the relative contributions of the independent variables in predicting IES at follow-up (IES-F).

In accordance with current evidence and theoretical conceptualisations of PTSD, it was expected that anxiety sensitivity would predict PTSD symptoms at follow-up. Multiple regression analysis indicated that anxiety sensitivity predicted IES-F at nearing significant levels ( $R^2=0.07$ ,  $F=3.105$ ,  $p=0.086$ ,  $ASI\ Beta=0.278$ ). Although anxiety sensitivity just failed to predict IES-F at the  $p<0.05$  significance level the result shows that the prediction is likely to be accurate in over 91% of cases. The R value indicates that anxiety sensitivity accounts for only 7% of the variance in IES-F scores.

As detailed in the introduction chapter, Ehlers and Steil (1995) and Ehlers and Clark (2000) propose that PTSD symptoms will become persistent, or maintained, when an individual interprets these symptoms negatively, ruminates about their trauma and engages in cognitive avoidance (e.g. thought suppression). It was therefore expected that these three variables would function to maintain distress in the RTA population and mediate the predictive relationship between predisposing/immediate post-traumatic variables and subsequent PTSD symptoms. In order to maintain a good ratio between the number of participants in the analyses and the independent variables, a correlational analysis was conducted on the hypothesised mediating/maintaining factors. In accordance with the theory proposed by Ehlers and Clark (2000) it was anticipated that these variables would be strongly related. The results of this correlational analysis are presented in Table 2. below.

Table 2. Results of correlational analyses between hypothesised mediating/maintaining variables.

	<b>Negative interpretatio</b>	<b>Ruminatio</b>	<b>Thought suppressio</b>
<b>Negative interpretatio</b>	1.000 ( 50) p= .	0.6211 ( 50) p= .000	0.2911 ( 50) p= .040
<b>Rumination</b>	0.6211 ( 50) p= .000	1.000 ( 50) p= .	0.3826 ( 50) p= .006
<b>Thought suppression.</b>	0.2911 ( 50) p= .040	0.3826 ( 50) p= .006	1.000 ( 50) p= .

It is clear that these three variables are highly related. As a result of this close inter-relationship it was decided to collapse these variables into one mediating/maintaining variable. This helps to maintain a good ratio between participants and variables and reduces the probability that any mediation/maintenance functions identified statistically are the result of the high correlations between independent variables. As a check of internal consistency, Cronbach's alpha was calculated for the new collapsed variable ( $\alpha=.686$ ). This indicates an acceptable level of internal consistency.

In order to address the issue of potential mediation between anxiety sensitivity and IES-F, further multivariate analyses were conducted. Multiple regression analysis indicated that anxiety sensitivity predicted the new collapsed variable (subsequently to be referred to as maintenance factors) significantly ( $R^2 = 0.28$ ,  $F=18.65$ ,  $p=0.000$ , ASI Beta=0.529). The R value indicates that anxiety sensitivity accounts for 28% of the variance in maintenance factors scores. Further multiple regression analysis indicated that maintenance factors predicted IES-F significantly ( $R^2 = 0.31$ ,  $F=16.88$ ,  $p=0.000$ , maintenance factors Beta=0.559). This R value shows that maintenance factors account for 31% of the variance in IES-F scores. When anxiety sensitivity and maintenance factors were taken together they also predicted IES-F significantly ( $R^2 = 0.31$ ,  $F=8.25$ ,  $p=0.001$ , ASI Beta=0.029, ASI  $t=0.188$ , ASI  $p=0.852$ , maintenance factors Beta=0.546, maintenance factors  $t= 3.523$ , maintenance factors  $p=0.001$ ).

The multivariate analyses above indicate that there are strong predictive relationships between anxiety sensitivity and maintenance factors and between both variables and IES-F. Of importance is the reduction in ASI Beta from 0.278 to 0.029 when the function of maintenance factors on IES-F are partialled out. This effect indicates that the relationship between anxiety sensitivity and IES-F is strongly mediated by maintenance factors. The maintenance factors significance level of 0.001 when anxiety sensitivity is partialled out indicates that maintenance factors predict IES-F over and above, or independent of, anxiety sensitivity. The predictive role of anxiety sensitivity on IES-F is not independent of maintenance

factors. This is congruent with a mediating role between anxiety sensitivity and IES-F of the maintenance factors.

In accordance with theoretical considerations outlined in the introduction chapter, it was hypothesised that anxiety sensitivity would significantly predict negative interpretations of symptoms. Linear multiple regression analyses indicated that this was the case ( $R=.08$ ,  $F=4.567$ ,  $p=0.037$ ) and that anxiety sensitivity accounted for 8% of the variance in negative interpretations.

In accordance with theoretical conceptualisations and empirical evidence, it was anticipated that a negative attitude towards emotional expression would predict PTSD symptoms at follow-up. Multiple regression analysis indicated that, contrary to expectations, there was no significant predictive relationship between AEE and IES-F ( $R^2 = .07$ ,  $F=2.901$ ,  $p=.0969$ , AEE Beta=-.269). As a result of this lack of prediction, further analysis regarding the potential mediation of a relationship between AEE and IES-F was not conducted.

In accordance with theoretical conceptualisations of PTSD and a growing body of empirical evidence, it was anticipated that immediate post-traumatic reactions would predict PTSD symptoms at follow-up. Multiple regression analysis indicated that Impact of Event scores at baseline (IES-B) predicted IES-F significantly ( $R^2 = 0.25$ ,  $F=12.51$ ,  $p=0.001$ , IES-B Beta=0.502). The R value indicates that IES-B accounts for 25% of the variance in IES-F scores.

In order to address the issue of potential mediation between IES-B and IES-F, further multivariate analyses were conducted. It was established through multiple regression analysis that IES-B predicted maintenance factors significantly ( $R^2 = 0.57$ ,  $F=63.91$ ,  $p=0.000$ , IES-Beta=0.755). This R value shows that IES-B accounts for 57% of the variance in maintenance factors scores. Similarly, it had already been established that maintenance factors significantly predicted IES-F (see anxiety sensitivity results). When IES-B and maintenance factors were taken together they also predicted IES-F significantly ( $R^2 = 0.33$ ,  $F=9.01$ ,  $p=0.000$ , IES-B Beta=0.206,

IES-B  $t=1.050$ , IES-B  $p=0.300$ , maintenance factors Beta=0.410, maintenance factors  $t=2.091$ , maintenance factors  $p=0.043$ ).

The multivariate analyses above indicate that there are strong predictive relationships between IES-B and maintenance factors and between both variables and IES-F. Of importance is the reduction in IES-B Beta from 0.502 to 0.206 when the function of maintenance factors on IES-F are partialled out. This effect indicates that the relationship between IES-B and IES-F is strongly mediated by maintenance factors. The maintenance factors significance level of 0.043 when IES-B is partialled out indicates that maintenance factors predict IES-F over and above, or independent of, IES-B. The predictive role of IES-B on IES-F is not independent of maintenance factors. This is congruent with a mediating role between IES-B and IES-F of the maintenance factors.

As described in the introduction chapter anxiety sensitivity is conceptualised as a predispositional variable, whereas IES-B is a trauma-induced state. The analyses so far have separated out this dispositional factor from the psychological status (IES-B) of participants when completing the ASI. Therefore, the extent to which the relationship between anxiety sensitivity and IES-F is contaminated by the psychological status of participants at baseline (IES-B) remains unclear. In order to address this issue of potential contamination of ASI scores by IES-B, further multivariate analyses were conducted. Multiple regression analysis indicated that ASI and IES-B were significantly related ( $R^2 = 0.30$ ,  $F=20.87$ ,  $p=0.000$ , ASI Beta=0.550). It had already been established that ASI significantly predicted IES-F, and that IES-B also significantly predicted IES-F (see previous analyses). When ASI and IES-B were taken together, they also predicted IES-F significantly ( $R^2 = 0.25$ ,  $F=6.12$ ,  $p=0.006$ , ASI Beta=0.039, ASI  $t=0.239$ , ASI  $p=0.812$ , IES-B Beta=0.483, IES-B  $t=2.919$ , IES-B  $p=0.006$ ).

This analysis indicates that there are strong predictive relationships between ASI and IES-B and between both variables and IES-F. Of importance is the reduction in ASI Beta from 0.278 to 0.039 when the function of IES-B on IES-F is partialled

out. This effect indicates that the relationship between ASI and IES-F is not only strongly mediated by maintenance factors (as shown previously) but is strongly mediated by IES-B. The significance level of IES-B predicts IES-F over and above, or independent of, ASI. The predictive role of ASI on IES-F has again been proven to be dependent on other variables. This is congruent with a contamination of ASI scores by baseline distress and a mediating role for IES-B between predispositional AS and IES-F.

In accordance with theoretical conceptualisations of PTSD and a growing body of empirical evidence, it was expected that peri-traumatic dissociation would play a role in PTSD symptoms at follow-up. Multiple regression analysis indicated that PDEQ scores predicted IES-F significantly ( $R^2 = 0.16$ ,  $F=7.06$ ,  $p=0.012$ , PDEQ Beta=0.405). The R value indicates that PDEQ accounts for 16% of the variance in IES-F scores.

In order to assess the issue of potential mediation between PDEQ and IES-F, further multivariate analyses were conducted. It was established through multiple regression analysis that PDEQ predicted maintenance factors significantly ( $R^2 = 0.30$ ,  $F=20.29$ ,  $p=0.000$ , PDEQ Beta=0.559). This R value indicates that PDEQ accounts for 30% of the maintenance factors scores. It had already been established that maintenance factors significantly predicted IES-F. When PDEQ and maintenance factors were taken together, they also predicted IES-F significantly ( $R^2 = 0.32$ ,  $F=8.46$ ,  $p=0.000$ , PDEQ Beta=0.162, PDEQ  $t=1.004$ , PDEQ  $p=0.322$ , maintenance factors Beta=0.469, maintenance factors  $t=2.901$ , maintenance factors  $p=0.006$ ).

The analysis indicates that there are strong predictive relationships between PDEQ and maintenance factors and between both variables and IES-F. Of importance is the reduction in PDEQ Beta from 0.405 to 0.162 when the function of maintenance factors on IES-F is partialled out. This effect indicates that the relationship between PDEQ and IES-F is strongly mediated by maintenance factors. The maintenance factors' significance level of 0.006 when PDEQ is partialled out indicates that



maintenance factors predict IES-F over and above, or independent of PDEQ. The predictive role of PDEQ on IES-F is not independent of maintenance factors. This is again congruent with a mediating role between peri/pre/post-traumatic variables and IES-F of the maintenance factors.

In order to understand how the independent variables of ASI, PDEQ, IES-B and maintenance factors predict IES-F when taken together, further multivariate analysis was conducted. In light of the analysis presented above, it was anticipated that the variables taken together would significantly predict IES-F. Also, that pathways between variables would be strongly mediated and that no independent predictor of IES-F would emerge; this finding would be congruent with a conceptualisation of PTSD as a disorder resulting from both predispositional and maintaining factors.

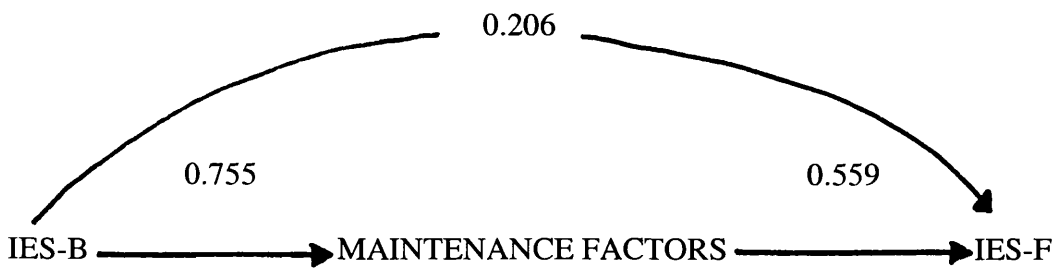
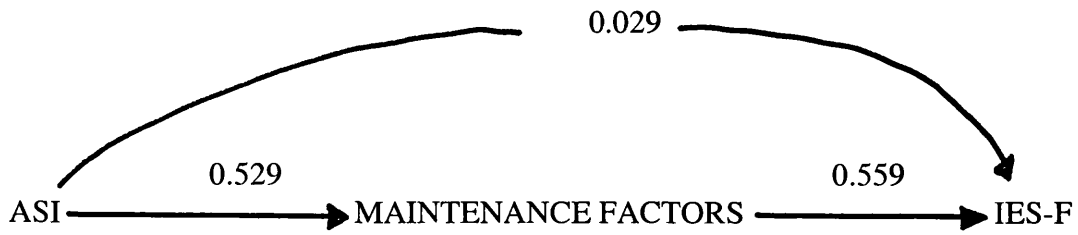
Multiple regression analysis indicated that when ASI, PDEQ, IES-B and maintenance factors were taken together, they significantly predicted IES-F. Table 3. below illustrates the results of this analysis in detail. Overall, the regression showed  $R^2 = 0.34$ ,  $F=4.27$ ,  $p=0.006$ . The R value indicates that taken together these variables account for 34% of the variance in IES-F scores.

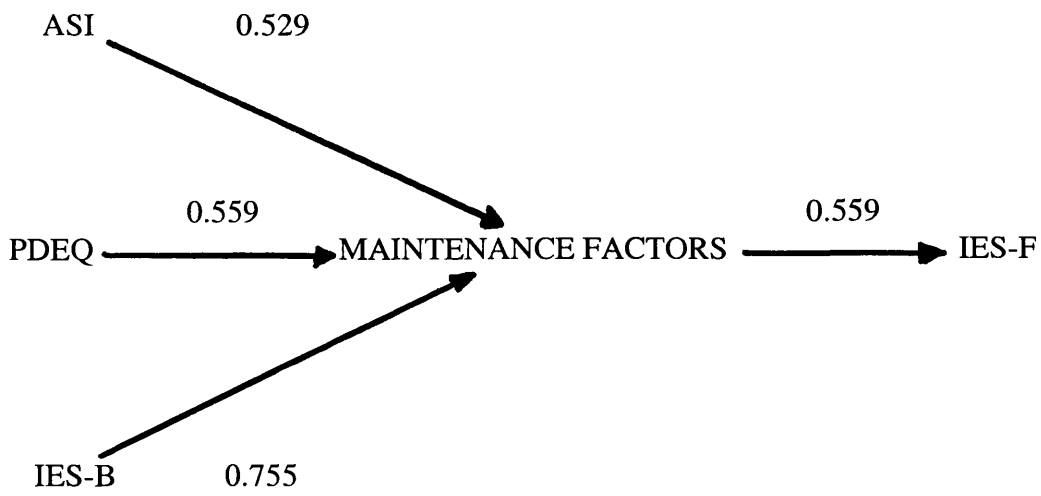
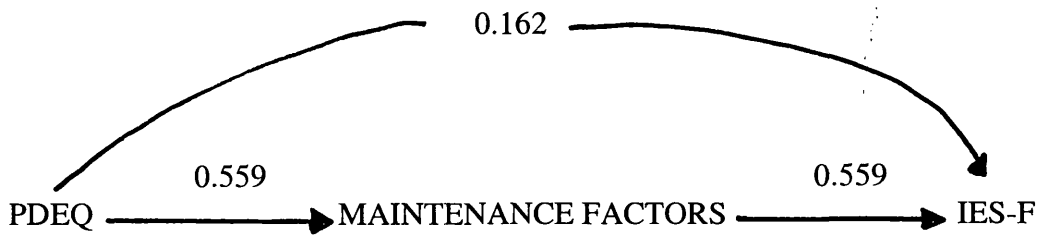
Table 3. Multiple regression analysis predicting IES-F from ASI, PDEQ, IES-B and maintenance factors.

<b>Variable</b>	<b>Beta</b>	<b>t-score</b>	<b><i>p</i></b>
ASI	-0.26	-0.163	0.871
PDEQ	0.141	0.841	0.406
IES-B	0.187	0.880	0.385
Maintenance factors.	0.358	1.677	0.103

Of importance are the reductions in Beta values for all variables from their overall relationship with IES-F to their relationship with IES-F when the function of all other three variables is partialled out. These are respectively; ASI reduced from 0.278 to -0.026, PDEQ reduced from 0.405 to 0.141, IES-B reduced from 0.502 to 0.187, and maintenance factors reduced from 0.559 to 0.358. These effects strongly indicate that the relationships between each variable and IES-F are strongly mediated by other variables. Although none of the variables are shown to act independently of one another, maintenance factors retains a robust Beta of 0.358,  $p=0.103$  when all three variables are partialled out. As noted above, maintenance factors were seen to predict IES-F independent of ASI, PDEQ and IES-B when investigated singularly. Figure 3. below represents a summary of the above statistical analysis.

Figure 3. A graphical representation of prediction and maintenance of IES-F.





### 3.5 Prediction of Diagnostic Status (SRS-PTSD: DSM-IV)

The Self-Rating Scale for PTSD (SRS-PTSD:DSM-IV) measures the symptoms of PTSD corresponding to the 17 diagnostic criteria laid down in DSM-IV and provides a diagnosis of PTSD. As outlined in the introduction chapter, it was anticipated that a number of the independent variables measured would predict PTSD status at follow-up.

Preliminary correlational analysis indicated that the following independent variables were not significantly related to SRS-PTSD and were therefore not controlled for in the multiple regression analyses;

Injury to others:  $r=.311, p=.053$

Litigation:  $r=-.047, p=.767$

Responsibility for the accident:  $r=.313, p=.052$

Anger:  $r=-.052, p=.751$

Perceived threat:  $r=-.324, p=.064$

Behavioural avoidance:  $r=.149, p=.371$

Damage to property:  $r=.212, p=.195$

Injury to self:  $r=.134, p=.415$

A series of logistic regression analyses were completed using the SRS-PTSD as the dependent variable. It was necessary to conduct logistic analysis as the aim was to predict the likelihood of falling into a category of PTSD diagnosis, rather than to predict individual scores as in multiple (linear) regression. What follows are the results of logistic regression analyses carried out to determine the relative contributions of the independent variables in predicting SRS-PTSD at follow-up (SRS-F).

In accordance with theoretical conceptualisations and empirical evidence detailed in the introduction chapter, it was anticipated that anxiety sensitivity would predict PTSD diagnostic status at follow-up. It had already been established that anxiety sensitivity predicted IES-F at a level nearing significance (see previous analysis).

Logistic regression analysis indicated that ASI did not significantly predict SRS-F as expected (Chi sq.=.547 , Chi  $p=0.459$ ,  $B=-0.0343$ ,  $p=0.460$ ). As a result of this lack of prediction further analysis regarding the potential mediation of a relationship between ASI and SRS-F was not conducted.

In accordance with theoretical conceptualisations and empirical evidence detailed in the introduction chapter, it was anticipated that a negative attitude towards emotional expression would predict PTSD diagnostic status at follow-up. Logistic regression analysis indicated AEE did not significantly predict SRS-F as expected (Chi sq.=.376 , Chi  $p=0.539$ ,  $B=0.0331$ ,  $p=0.547$ ). As a result of this lack of prediction, further analysis regarding the potential mediation of a relationship between AEE and SRS-F was not conducted. This result is congruent with the failure to find a predictive relationship between AEE and IES-F in this population.

In accordance with theoretical conceptualisations of PTSD and a growing body of empirical evidence, it was expected that peri-traumatic dissociation would play a significant role in PTSD diagnostic status at follow-up. It had already been established that PDEQ predicted IES-F significantly. Logistic regression analysis indicated that PDEQ scores predicted SRS-F at borderline significance (Chi sq.=3.795, Chi  $p=0.051$ ,  $B=-0.1102$ ,  $p=0.084$ ,  $ExpB=0.895$ ).

In order to address the issue of potential mediation between PDEQ and SRS-F, further multivariate analyses were conducted. It had already been established through multiple regression analysis that PDEQ significantly predicted maintenance factors. A logistic regression found also that maintenance factors significantly predicted SRS-F (Chi sq.=5.631, Chi  $p=0.017$ ,  $B=-1.389$ ,  $p=0.032$ ,  $Exp.B=0.249$ ). When PDEQ and maintenance factors were taken together they also predicted SRS-F (Chi sq.=6.502, Chi sig.=0.038).

The analysis indicates that there are strong predictive relationships between PDEQ and maintenance factors and between both variables and SRS-F. Of importance is the increase in PDEQ-B from  $-0.1102$  to  $-0.069$ , and PDEQ Exp-B from  $0.895$  to

0.932, when the function of maintenance factors on SRS-F is partialled out. This effect indicates that the relationship between PDEQ and SRS-F is strongly mediated by maintenance factors. Neither PDEQ nor maintenance factors significantly predict SRS-F over and above, or independent of, each other (PDEQ WALD sig.=0.317, maintenance factors WALD sig.=0.112, maintenance factors Chi  $p$  < 0.10). The predictive role of PDEQ mediated by maintenance factors as regards SRS-F is congruent with the analysis of PDEQ, maintenance factors and IES-F, reported above.

In accordance with theoretical conceptualisations of PTSD and a growing body of empirical evidence, it was anticipated that PTSD diagnostic status immediately after RTA would predict SRS-F. Logistic regression analysis indicated that SRS-B scores predicted SRS-F significantly (Chi sq.=3.754, Chi sig=0.052, B=2.335,  $p$ =0.045). It had already been established that IES-B predicted IES-F significantly.

In order to address the issue of potential mediation between SRS-B and SRS-F, further multivariate analyses were conducted. Multiple regression analysis indicated that SRS-B predicted maintenance factors significantly ( $R^2=0.19$ ,  $F=11.25$ ,  $p=0.001$ , SRS-B Beta=-0.439). It had already been established that maintenance factors significantly predicted SRS-F (see analysis above). When SRS-B and maintenance factors were taken together they also predicted SRS-F significantly (Chi sq.=7.411, Chi sig.=0.024).

The analysis indicates that there are strong predictive relationships between SRS-B and maintenance factors and between both variables and SRS-F. Of importance is the reduction in SRS-B from 2.335 to 1.715, and SRS-B Exp B from 10.333 to 5.559, when the function of maintenance factors on SRS-F is partialled out. This effect indicates that the relationship between SRS-B and SRS-F is strongly mediated by maintenance factors. Although neither SRS-B nor maintenance factors significantly predict SRS-F over and above, or independent of, each other, maintenance factors' independent role in SRS-F is nearing significance (SRS-B WALD sig.=0.186, maintenance factors WALD sig.=0.071, maintenance factors



Chi  $p=<0.10$ ). This effect of a heavy mediation of maintenance factors between SRS-B and SRS-F is congruent with results reported above on the mediation of maintenance factors between IES-B and IES-F in the same population.

In order to better understand how the independent variables of PDEQ, SRS-B and maintenance factors predict SRS-F when taken together and to further investigate any potential independent predictors of SRS-F, further multivariate analysis was conducted. In light of the results presented above it was anticipated that the variables taken together would significantly predict SRS-F and that no independent predictor of SRS-F would emerge.

Logistic regression analysis indicated that when PDEQ, SRS-B and maintenance factors were taken together they significantly predicted SRS-F. Table 4. Below illustrates the results of this analysis in detail. Overall, the regression showed Chi sq.=7.942, Chi  $p=0.047$ .

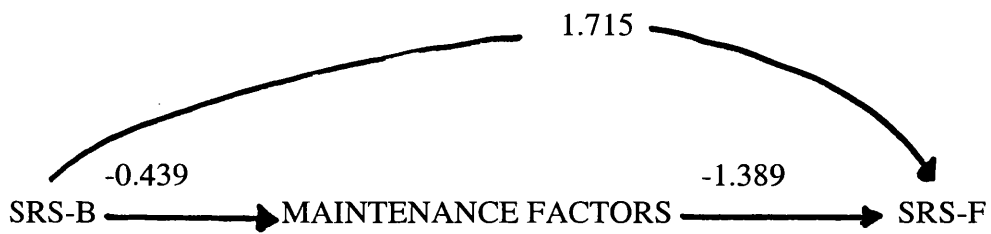
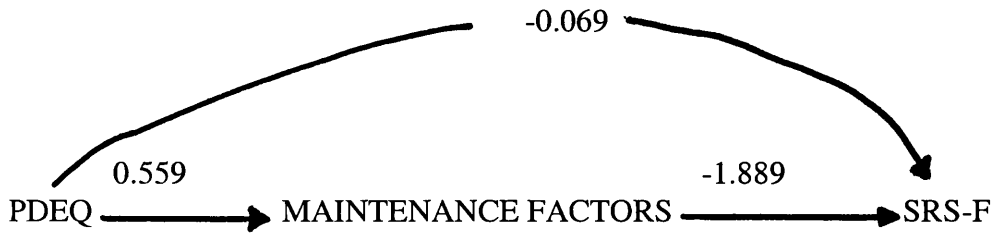
Table 4. Logistic regression analysis predicting SRS-F from PDEQ, SRS-B and maintenance factors.

<b>Variable</b>	<b>WALD sig</b>	<b>B</b>	<b>Exp B</b>
PDEQ	0.398	-0.0653	0.936
SRS-B	0.322	1.3054	3.689
Maintenance factors.	0.148	-1.0268	0.358

Of importance are the changes in B values, for all three variables, from their overall relationship with SRS-F, to their relationship with SRS-F when the function of both other variables is partialled out. These are respectively; PDEQ increased from  $-0.1102$  to  $-0.0653$ , SRS-B reduced from  $2.335$  to  $1.3054$ , and maintenance factors reduced from  $-1.389$  to  $-1.0268$ . These effects indicate that the relationships between each variable and the SRS-F are strongly mediated by the two other variables.

The WALD significance values for all three independent variables indicate that no variable alone predicts SRS-F over and above, or independent of, the other two variables. Figure 4. below, represents a summary of the above statistical analysis.

Figure 4. A graphical representation of prediction and maintenance of SRS-F.



### 3.6 Summary of Main Findings

14.3% of the population at baseline and 12.8% at follow-up, were diagnosed with PTSD.

24% of the population at baseline and 17.9% at follow-up, scored  $\geq 35$  on the IES ('high').

24% of the population at baseline and 17.9% at follow-up, scored either moderate or high on the HADS anxiety scale.

22% of the population and 13.2% at follow-up, scored either moderate or high on the BAI.

10% of the population at baseline and 10.3% at follow-up, scored either moderate or high on the HADS depression scale.

14% of the population at baseline reported negative interpretations of their post-traumatic recollections, either often or always.

26% of the population at baseline reported to suppress RTA related thoughts, often or always.

Anxiety sensitivity significantly predicts IES-F and this relationship is strongly mediated by maintenance factors (negative interpretations, rumination, thought suppression).

The relationship between anxiety sensitivity and IES-F is also strongly influenced by IES-B.

IES-B significantly predicts IES-F and this relationship is strongly mediated by maintenance factors.

There was no predictive relationship between a negative attitude towards emotional expression and IES-F.

Peri-traumatic dissociation significantly predicts IES-F and this relationship is strongly mediated by maintenance factors.

Taken together, ASI, PDEQ, IES-B and maintenance factors significantly predict IES-F.

There was no predictive relationship between anxiety sensitivity and SRS-F.

There was no predictive relationship between negative attitude towards emotional expression and SRS-F.

SRS-B significantly predicts SRS-F and this relationship is strongly mediated by maintenance factors.

PDEQ significantly predicts SRS-F and this relationship is strongly mediated by maintenance factors.

Taken together SRS-B, PDEQ and maintenance factors significantly predict SRS-F.

Maintenance factors is the only independent variable to exhibit independent prediction of IES-F and SRS-F, over and above other variables.

## **DISCUSSION**

### **4.1 Statement of Intention**

The discussion chapter aims to investigate both the clinical and theoretical meaning of the results in the current study. First, a discussion of the prevalence findings aims to place the current study in the context of empirical evidence and specifically to address any differences that may be due to minor RTA. Second, prediction of PTSD symptoms and diagnosis is examined and placed in the context of theoretical and empirical work. Third, the issue of PTSD maintenance is addressed and results are interpreted with cognitive conceptualisations of PTSD (e.g. Ehlers and Clark, 2000; Brewin et al., 1996) in mind. Fourth, both theoretical and clinical implications of the current study are outlined. Finally, methodological limitations and strengths are discussed and related to the clinical utility of the findings and implications for future research are described.

### **4.2 Interpretation of Prevalence Findings**

The identification of clinically significant anxiety, depression and post-traumatic stress symptoms in this study is congruent with findings reported elsewhere. As noted in the introduction chapter, a number of prospective longitudinal studies using consecutive attenders to hospital post-RTA have recently been published and lend themselves to comparison with the current research. The current finding that 14.3% of the population at baseline and 12.8% at 3 months follow-up were diagnosed with PTSD is consistent with a number of empirical and theoretical explanations. Shalev

et al. (1998) report prevalence levels of PTSD at 1 month post-trauma of 29.9%, and 17.5% at 4 months follow-up. Feinstein et al. (1991) report post-RTA PTSD in 25% of their sample at 6 weeks and 14.6% at 6 months. Ursano et al. (1999), report 34.4% PTSD at 1 month post-RTA and 25.2% at 3 months. The prevalence rates found in the current study are lower than those reported elsewhere, but appear to follow a similar trend of reduction in PTSD over time. PTSD rates found in our sample of minor-RTA are expected to be lower than those in a population of more severe RTA, as levels of dissociation and disruption in autobiographical memory implicated in the aetiology of PTSD (e.g. Ehlers and Clark, 2000; Brewin et al., 1996), should be reduced. The pattern of a reduction in PTSD over time following trauma, replicated in the current research, lends credence to the hypothesis of a non-linear relationship between initial symptoms and PTSD at follow-up (e.g. Harvey and Bryant, 1998). As noted in the Research Methodology chapter, none of the participants completing follow-up assessment had received any post-traumatic psychological therapy.

As reported in the results chapter, 24% of the population at baseline and 12.9% at 3 months follow-up exhibited 'high' scores on the IES ( $\geq 35$ ). This finding lends support to the pattern of a reduction in psychological distress over time, congruent with theoretical conceptualisations of trauma that emphasise emotional processing (e.g. Horowitz, 1976; Brewin et al., 1996; Ehlers and Clark, 2000; Dalgleish, 1999). The reduction in distress over time also indicates a non-linear relationship between initial distress and follow-up symptoms. Bryant and Harvey (1996) reported that 50% of an RTA population scored moderate or high on the IES two



weeks post-accident. They note that many individuals experience the core symptoms of PTSD in the aftermath of an RTA. Current findings compliment those of Bryant and Harvey (1996), despite the population being limited to minor RTA. Therefore, the evidence from the SRS-PTSD and IES taken together indicate significant levels of post-traumatic stress in this population.

The identification in the current study of 10% of the population at baseline and 10.3% at follow-up, to be experiencing moderate or high depression, fits with previous empirical evidence. Shalev et al. (1998) report post-traumatic depression in 19% of their sample at 1 month and 14.2% at 4 months follow-up. The lower rates of depression found here may be associated with lower levels of post-traumatic stress symptoms described above. Blanchard et al. (1998) propose that the overlap in diagnostic criteria for PTSD and depression is substantial and this may therefore explain the lower prevalence rates. Alternatively, it may be that individuals prone to depression report lower levels in minor RTA than major RTA, due to the lower levels of post-traumatic symptoms available for negative interpretation. Further analysis of the nature of individuals experiencing post-traumatic depression may allow exploration of ideas proposed by Dalglish (1999), that it is people with primarily damaged models of themselves, whose schemas are confirmed by trauma, who develop post-traumatic depression. This analysis was beyond the remit of the current study.

Taylor and Koch (1995) propose that anxiety disorders (including, but not exclusively PTSD and ASD), are the most common psychological consequence of

RTA. As reported in the results chapter, 24% of the population at baseline and 17.9% at 3 months follow-up, scored either moderate or high on the HADS anxiety scale. Similarly, using the BAI the population recorded 22% moderate/high anxiety at baseline and 13.2% at follow-up. In comparison to rates reported by Bryant and Harvey (1996) using the Spielberger State Anxiety Inventory of 76% moderate/high state anxiety within 2 weeks of an RTA, the results from this study are low. Again this may in part be due to the nature of trauma investigated (generally low-impact, low speed, urban RTA), and is congruent with lower levels of PTSD also found in this population. The rates of state anxiety reported by Bryant and Harvey (1996) are extremely high and derived from a different measure, and it is important to note that the lower rates reported here represent clinically significant levels of anxiety.

Fear, avoidance and anxiety related to driving/riding and to being a passenger, were reported using an unstandardised measure and analysed item-by-item. As reported in the results chapter, 22.9% of the population engaged in behavioural avoidance of driving/riding at baseline, as did 15.8% at follow-up. There is no available empirical evidence with which to compare these results, although these prevalence rates seem significant and important. Care must be taken in interpreting behavioural avoidance results however, as many individuals did not drive/ride in the aftermath of the RTA due to damage to their vehicles. A more accurate measurement of conscious and willing avoidance was gained from the item assessing specific avoidance of driving/riding conditions. 31.8% at baseline and 30.2% at follow-up reported specific avoidance as a result of RTA. This sort of avoidant coping has been proposed by many authors to be significant in impeding the emotional processing of

a traumatic event and in maintaining a current sense of threat (e.g. Brewin et al., 1996; Ehlers and Clark, 2000). Anxiety symptoms whilst driving were reported by 37.2% at baseline and 36.1% at follow-up and at similar rates whilst being a passenger. This prevalence is higher than the BAI and HADS anxiety results and indicates that in the context of specific trauma related stimuli, anxiety levels rise in even a minor RTA population. Fears about travelling in cars/on bikes were endorsed by the population at a higher rate than either anxiety symptoms or avoidance (67.4% at baseline, 66.7% at follow-up). This result may represent a tendency in the population to worry about the possibility of future accidents in the light of their recent experience. Whether this worry is of clinical significance is not clear, although it appears to lead to lower levels of actual avoidance and anxiety symptoms than the extent of worry may imply. The high rate of worry measures being endorsed may indicate a shift in schema post-RTA. Individuals who report that they are worried another accident will occur, or that the probability of another RTA is high, may be exhibiting a shift in their assumptions/expectations post-RTA. According to Dalgleish (1999) this can be conceptualised as the healthy and adaptive incorporation of new salient trauma information into existing mental representations. Only in some circumstances will this process of incorporation fail, leading to PTSD.

#### 4.3 Interpretation of the Prediction of PTSD / Post-Traumatic Stress Symptoms

As a result of extensive multivariate analyses (see results chapter), it has been possible to successfully identify a number of predictive factors for PTSD. The first of these is anxiety sensitivity. The predispositional factor of anxiety sensitivity was

found to almost significantly predict the severity of post-traumatic stress symptoms (IES) at 3 months follow-up. This result compliments empirical evidence which implicates anxiety sensitivity in the aetiology and maintenance of panic disorder (Ehlers, 1995; Schmitt et al., 1997) and PTSD (Federoff, 2000). Ehlers (1995) suggested that anxiety sensitivity is a cognitive trait that places an individual at risk of developing psychopathology in the context of stress. Furthermore, Shalev et al. (1998) found early autonomic arousal post-trauma to predict PTSD. The authors hypothesise that some individuals may perceive this autonomic activation as fearful and that it may be this predispositional fear of anxiety the triggers this process. Federoff et al. (2000) reported that anxiety sensitivity to be predictive of PTSD in a sample of RTA victims. The current study compliments the results of Federoff et al. (2000) and lends support to the predictive role of anxiety sensitivity in PTSD. As detailed in the introduction chapter, many of the initial post-traumatic symptoms experienced by most RTA victims are arousal related sensations, such as palpitations, respiratory distress, intrusions and hyperarousal. As proposed by Ehlers and Clark (2000), it is the individual's interpretation of these symptoms that is integral in the persistence, or not, of PTSD. Anxiety sensitivity, or the belief that anxiety/arousal related sensations are harmful, may predispose an individual to catastrophically misinterpret their initial symptoms, thereby increasing anxiety and the greater likelihood of further persistent PTSD symptoms. The result reported in the results chapter that anxiety sensitivity significantly predicts negative interpretation of symptoms supports this hypothesis. The current finding therefore supports the role of anxiety sensitivity in the aetiology of PTSD and the cognitive conceptualisation of PTSD suggested by Ehlers and Clark (2000).

In accordance with theoretical conceptualisations of PTSD and developing empirical evidence, it was found that both the severity of post-traumatic symptoms (IES), and PTSD diagnostic status at baseline, significantly predicted severity of symptoms and diagnostic status, respectively, at follow-up. This finding provides empirical support for the long held assumption that immediate post-traumatic stress symptoms are a precursor to long term PTSD. The findings from the current study compliment reports proposing a significant relationship between initial and long term distress (e.g. Blanchard et al., 1996; Brewin et al., 1999; Koren et al., 1999; Blanchard et al., 1997; Harvey and Bryant, 1998). The predictive role of initial post-traumatic stress symptoms in long term PTSD also lends support to conceptualisations of PTSD that place importance on disruptions in autobiographical memory (e.g. Brewin et al., 1996; Ehlers and Clark, 2000; Dagleish, 1999). Harvey et al. (1998) propose that individuals who experience disruptions in autobiographical memory and subsequent intrusive memories, will minimise their awareness of these memories in order to alleviate the associated distress. This cognitive avoidance will impair access to traumatic memory and impede emotional processing leading to the persistence of PTSD symptoms. This issue of the maintenance of PTSD will be discussed in some detail later in this chapter. Foa et al. (1996) note that adaptive reactions to trauma require a capacity to integrate corrective information into available traumatic memory; individuals who experience initial distress may impede this process as they attempt to block out the associated distress.

The third predictive variable found to significantly predict both severity of symptoms and PTSD diagnosis at follow-up, was peri-traumatic dissociation. This result provides complimentary empirical support for findings reported elsewhere, implicating dissociation in the development of PTSD (e.g. Holen, 1993; Koopman et al., 1994). The literature regarding the role of dissociation in predicting PTSD, is however, mixed. There are studies (e.g. Barton et al., 1996) that fail to find a significant role for dissociation in PTSD and authors claim that dissociative levels in RTA are too low to impact upon disruption in autobiographical memory. Although this study has not directly investigated the specific role of dissociation on memory during trauma, it remains that peri-traumatic dissociation significantly predicts PTSD in this sample of minor RTA victims. This finding is congruent with conceptualisations of PTSD that implicate the disruption of information processing during trauma in PTSD (e.g. Ehlers and Clark, 2000; Brewin et al., 1996). These authors propose that this disruption gives rise to poorly discriminated, poorly elaborated and fragmented traumatic memory, which requires emotional processing in order to incorporate it into existing mental representations. The result in the current study, that a disruption in the experience of reality (dissociation), is significantly associated with subsequent PTSD, supports theories of PTSD that emphasise disruptions in peri-traumatic information processing.

Having identified anxiety sensitivity, immediate post-traumatic reaction and peri-traumatic dissociation as significant predictors of PTSD at 3 months follow-up, further multivariate analyses were employed to investigate any independent effects. As noted in the results chapter, anxiety sensitivity, immediate post-traumatic

reactions and peri-traumatic dissociation, when taken together, significantly predict the severity of PTSD symptoms. None of the variables exhibit an independent predictive role for PTSD. Similarly, when immediate post-traumatic reaction and peri-traumatic dissociation are taken together, they significantly predict PTSD diagnostic status. Neither of the variables exhibit an independent predictive role. This failure to find independent pathways predicting PTSD from a group of predisposing, peri and immediate post-traumatic variables is congruent with a multifaceted aetiology of PTSD. Recent conceptualisations of PTSD (e.g. Ehlers and Clark, 2000) describe many levels of aetiological and maintaining functions in PTSD and the disorder itself comprises three distinct groups of symptoms.

One hypothesis driven predictive variable that failed to significantly predict either severity of post-traumatic symptoms, or PTSD diagnostic status, was negative attitude towards emotional expression. It had been hypothesised, in line with theoretical considerations (Rachman, 1980; Williams, 1993) that a tendency to inhibit emotions and beliefs that emotional expression was harmful, would impede natural trauma recovery and lead to PTSD. In this sample of minor RTA victims this prediction did not hold, contrary to a body of empirical evidence implicating negative attitude towards emotional expression in the development of PTSD in more severe disasters (e.g. Brown and Grover, 1998; Williams et al., 1994; Joseph et al., 1997).

In attempting to understand this result it is important to consider exactly the hypothesised function of an inhibited emotional style, in the development of PTSD.

Williams (1993) proposes that individuals characterised by negative AEE will fail to emotionally process traumatic memory and that this will lead to persistence of unprocessed memory intruding into consciousness, leading to an increase in avoidance and the continuing failure to process trauma. Trauma will fail to become an isolated incident in the past, as negative AEE will prevent processing of poorly elaborated, poorly contextualised (Ehlers and Clark, 2000), situationally accessible (Brewin et al., 1996), memories. It has been discussed above that a minor-RTA may not lead to the kind of disruption in autobiographical memory proposed to require verbal post-traumatic emotional processing. If this were the case, negative AEE would not be a problem as traumatic memory would not require verbal processing in order to be incorporated into existing mental representations. Therefore, one possible explanation of our finding is that negative AEE is not a predictive factor of PTSD in the absence of disruption in autobiographical memory severe enough to require thorough verbal emotional processing. This is supported by the work of Barton et al. (1996) who failed to find a role for dissociation in post-RTA PTSD. Barton cited the low traumatic impact of many RTAs. However, the finding in the current research, that dissociation is predictive of PTSD would appear to fit poorly with this explanation. Dissociation is measured on a scale that lends itself to degrees of dissociation; our result may reflect a relationship between degree of dissociation and degree of PTSD, despite the level of dissociation being low. It may be that negative AEE only becomes significant at a point beyond the levels of dissociation, or disruption in autobiographical memory, recorded in our population.



Another hypothesised predictor of PTSD that failed to show a significant relationship was perceived threat. In accordance with the argument above, it may be that minor RTA leads to a low level of perceived threat. In such circumstances this variable may not be found to be significantly predictive of PTSD, where it has been in studies including more serious RTA.

The failure to find a significant relationship between accident/injury severity, litigation and responsibility for the RTA is congruent with empirical evidence outlined in the introduction chapter. This result provides further support for the argument that the subjective psychological experience of minor RTA is of greater importance in terms of psychological morbidity than objective environmental factors.

#### 4.4 Maintenance of PTSD and the Mediation of Significant Predictive Variables

Ehlers and Clark (2000) propose that PTSD symptoms will become persistent, or maintained, when an individual interprets these symptoms negatively, ruminates about their trauma and engages in cognitive avoidance of trauma related thoughts and memories. The extensive multivariate analysis reported above have enabled us to directly test this proposition. First, the result that these three maintenance factors are significantly related to one another supports the hypothesis that they are functioning post-trauma in a similar direction; the maintenance of a sense of current threat according to Ehlers and Steil (1995). The finding that maintenance factors heavily mediate the predictive role between anxiety sensitivity and the severity of PTSD symptoms is of great importance. This lends support to both empirical

evidence (Ehlers et al., 1998) and recent conceptualisations of PTSD (Ehlers and Clark, 2000), that maintenance factors play an important role in the development and persistence of PTSD. The finding that maintenance factors independently predict the severity of PTSD symptoms and that anxiety sensitivity does not, indicates again that this relationship is mediated. Also, the independent predictive role of maintenance factors over and above anxiety sensitivity shows that non-anxiety sensitive individuals are prone to persistent PTSD post minor-RTA, if they engage in poor coping strategies. The consequences for treatment of persistent PTSD, as suggested by Ehlers and Clark (2000), are that individuals must be helped to understand their maladaptive coping and to adopt less avoidant and catastrophic cognitive styles. Cognitive therapy may be utilised to address problematic attributions that give rise to a current sense of threat. Appraisals can be modified and examined in the light of alternative available evidence. For instance, an individual who thinks 'I must be going mad' in response to intrusive recollections post-RTA, may be helped to understand the importance of emotional processing, and to eventually come to the conclusion 'These are normal intrusive memories which I need to process in order to get over the RTA'. Through education and collaborative cognitive formulation individuals can be encouraged and helped to drop ruminative and thought suppression coping strategies. These strategies are suggested to prevent memory elaboration, hinder re-assessment of problematic appraisals and exacerbate symptoms. The finding that in our population of minor-RTA victims, maintenance factors heavily mediate and independently predict the severity of PTSD symptoms over and above anxiety sensitivity, is therefore of considerable clinical relevance.

As described in the results chapter, it was also found that maintenance factors heavily mediated the relationship between baseline severity of PTSD symptoms, and the severity of PTSD symptoms at follow-up. This again provides support for a non-linear relationship between initial post-traumatic distress and subsequent persistence of symptoms. One interpretation of this result is that the strong relationship between baseline and follow-up symptoms exists, for the most part, due to the maintenance factors. This would again fit with the conceptualisation proposed by Ehlers and Clark (2000). The finding that maintenance factors predict follow-up PTSD symptoms independent, or over and above, baseline symptoms provides further evidence of the importance of the maintenance factors. The implications for treatment outlined above would apply equally to a group of individuals whose initial symptoms quickly lead to maladaptive coping strategies.

It has now been illustrated that maintenance factors mediate the predictive relationships between both anxiety sensitivity and baseline symptoms, and follow-up symptoms. It was also found that baseline symptoms heavily mediated the predictive role of anxiety sensitivity in relation to follow-up symptoms. The implications of this finding are firstly that anxiety sensitivity has now been shown to be mediated by both maintenance factors and baseline symptoms on its path to predicting follow-up symptoms. Secondly, anxiety sensitivity measures may be contaminated by baseline distress. It is likely that, although anxiety sensitivity is a dispositional variable, it can be contaminated by state factors. The results for anxiety sensitivity are both

statistically weak (although borderline significant) and heavily mediated by other factors. Caution must therefore be taken in generalising these results.

There is a concern that the strong mediating role of maintenance factors found between baseline PTSD symptoms (IES) and follow-up PTSD symptoms (IES), could be due to strong associations between the IES and rumination (one third of maintenance factors). IES follow-up scores and rumination are strongly correlated ( $r=.553$ ,  $p=.000$ ). The SRS-PTSD measure of PTSD diagnostic status does not have such an overlap of ruminative questions as the IES and may therefore be seen as a more robust measure. The finding that maintenance factors also heavily mediate the relationship between baseline diagnostic status, and follow-up diagnosis dispels concerns raised by the IES. The role of maintenance factors in predicting PTSD diagnosis at follow-up is almost independent of baseline diagnosis. This finding provides further support for the importance of maladaptive cognitive coping strategies in the persistence of PTSD. The implications for treatment, as outlined above, may be more significant for those individuals with a diagnosis of PTSD, as they are more likely to receive psychological input.

The final predictive factor to be investigated for mediation was peri-traumatic dissociation. The findings that maintenance factors significantly mediate the relationship between peri-traumatic dissociation and both PTSD diagnosis and severity of symptoms, is congruent with other findings and with empirical evidence for the importance of maintenance factors (e.g. Ehlers et al, 1998). The relationship between maintenance factors and IES follow-up was also seen to be independent of

peri-traumatic dissociation. This would imply that individuals engaging in maladaptive cognitive coping may develop PTSD in the absence of peri-traumatic dissociation. This has also been shown to be the case with baseline symptoms and anxiety sensitivity. The overwhelming theme to be drawn from the multivariate analysis is that of the powerful independent and mediating impact of maintenance factors. This emphasis on cognitive attribution and coping strategies is complimentary to the conceptualisation and treatment implications outlined by Ehlers and Clark (2000). None of the hypothesised predictive variables acted on follow-up PTSD diagnosis or severity, independent of maintenance factors.

#### 4.5 Theoretical Implications

The results from the current study have a number of theoretical implications. The impact upon recent conceptualisations of PTSD shall be considered. In their theoretical models of PTSD Brewin et al. (1996), Ehlers and Clark (2000) and Dalgleish (1999) all place emphasis on the manner in which traumatic memories are laid down. Common to all these theories is the notion of an alteration in normal memory processing induced by intense emotion, confusion or dissociation. Brewin et al. (1996) understand this disruption in autobiographical memory to give rise to two distinct forms of memory, SAMS and VAMS. Ehlers and Clark (2000) suggest that a tendency to process sensation based, rather than meaning based, information occurs during trauma, leading to strong associative memories that are poorly elaborated in terms of time and context. Both theories propose that it is as a result of these alterations in autobiographical memory, that individuals experience involuntary intrusive trauma related memories, a major symptom of PTSD. The

finding in the current study that dissociation significantly predicts PTSD provides further empirical support for the proposition that disruptions in information processing during trauma, lead to PTSD.

Ehlers and Clark (2000) extend their theory to capture possible functions of the maintenance, or persistence, of PTSD. It is suggested that the manner in which an individual appraises, or interprets, their initial post-traumatic symptoms (e.g. intrusive memories generated as described above), will impact upon the maintenance of those symptoms. Negative interpretations such as 'I am going mad' function both to create a sense of current threat and to make avoidance strategies more likely. Such interpretations, it is suggested, may also directly give rise to negative emotions. The finding in the current study that negative interpretations of symptoms strongly predicts PTSD and heavily mediates the predictive role of pre-disposing, peri-traumatic and immediate post-traumatic variables, provides further empirical support for the proposal of Ehlers and Clark (2000) that attribution is integral to maintenance.

Both Brewin et al. (1996) and Ehlers and Clark (2000) propose that behavioural and cognitive avoidance of trauma related stimuli, will function to maintain PTSD symptoms. Ehlers and Clark (2000) suggest specifically that negative interpretations of symptoms will drive avoidance strategies. The finding that rumination and thought suppression both significantly predict PTSD and heavily mediate the prediction of PTSD by dispositional, peri-traumatic and immediate post-traumatic variables, is also supportive of the theoretical conceptualisation of Ehlers and Clark

(2000). The evidence from the current study in support of these models is qualitatively different from other published research, in so far as the population had suffered minor-trauma only. The implication is that the theoretical conceptualisations of PTSD described above apply to minor traumatic events. If this is the case, the proposed underlying mechanisms of memory and cognitive style may not be specific to individuals involved in particularly adverse traumatic events. It may be that the manner in which it is suggested information is processed post trauma can inform our understanding of the normal processes of memory, cognition and emotion. As suggested by Dalgleish (1999) it may be that PTSD can be seen as a logical and adaptive way of coping with aversive stimuli and that individuals embroiled in the symptoms of PTSD require education and understanding to help them adopt alternative ways of coping with salient, incongruent, trauma information.

#### 4.6 Clinical Implications

It has been shown that individuals who go on to exhibit PTSD at 4 months post-trauma, can be identified in the first few weeks following RTA. The variables of anxiety sensitivity, peri-traumatic dissociation, baseline symptoms, negative interpretations of symptoms, rumination and thought suppression are able to identify those individuals. It was discussed in the introduction chapter that currently few RTA victims have their emotional or psychological needs attended to in hospital. For many RTA victims, a brief visit to A&E is the only contact they have with health services, despite current evidence that up to 18% of the population studied experienced 'high' levels of post-traumatic symptomatology 4 months later.

Currently, little or nothing is done to utilise this predictive information in trying to prospectively identify those RTA victims at risk of long term PTSD.

It is now widely recognised that immediate post-traumatic debriefing is potentially damaging to the natural process of emotional adjustment (e.g. Mayou et al., 2000). Mayou et al. suggest that for many individuals immediate psychological de-briefing can be harmful. The authors note that de-briefing may interfere with the natural process of intrusion and avoidance, and may encourage rumination. Therefore, being able to identify individuals immediately post-trauma who are particularly distressed and offering them debriefing, is not recommended. Indeed, our results indicate that there is a non-linear relationship between initial distress and PTSD; even if debriefing were effective, concentrating on those individuals highly distressed post-RTA would result in a number of subsequent PTSD sufferers being missed. What may be of clinical utility is taking the significant predictive and maintaining factors and screening RTA victims in A&E. Those with high risk profiles could then be followed up at approximately 1 month post-accident and offered appropriate psychological therapy if indeed their high risk profile had acted as expected. The advantage of screening for all predictors would be that treatments could be tailored to individuals' needs (e.g. dissociation would require an emphasis on elaborating and contextualising traumatic memory; negative interpretations suggests cognitive therapy; thought suppression suggests education and cognitive-behavioural interventions). Also, individuals could be followed up and seen prior to maladaptive cognitive and behavioural strategies become entrenched and more difficult to shift. The stimulus generalisation view of PTSD proposed by Keane et



al. (1985) may be pre-empted by the introduction of more adaptive coping strategies over the first few months post-RTA.

The empirical evidence reported in this study in support of the role of maintenance factors in persistent PTSD, as proposed by Ehlers and Clark (2000), provides further support for psychological therapies that address such factors. An understanding of what maintains PTSD is arguably of more clinical utility in the therapy room, than knowledge of aetiology. Therapies that attempt to challenge catastrophic misinterpretations of initial symptoms, through education and Socratic questioning and dialogue should be seriously considered. Also, formulations and assessments that cover cognitive-behavioural coping strategies such as thought suppression, are implicated in the successful resolution of the maintenance of PTSD. Therapists must collaborate with and educate clients if maladaptive strategies that bring short-term relief are to be discarded in favour of long term non-avoidant coping methods.

Clinically significant levels of anxiety and depression identified in this population require careful interpretation. It may be that some individuals develop specific panic-like, or phobic reactions post-RTA, where others become depressed, or develop PTSD. It is highly unlikely that three distinct post-RTA groups occur, given the overlap with both anxiety and depression, of PTSD symptoms. It was unfortunately beyond the remit of this study to further examine the complex interrelationships between the dependent variables, although such an analysis would be fruitful. Clinically, it is important to understand that individuals will react in

unique ways to what objectively appear to be very similar situations. The result that it is attribution and not severity of injury, that predicts long term distress is important. Any interventions aimed at the psychological needs of RTA victims must be individually tailored, whether that means addressing anxiety, depression or PTSD, or a specific element of one of these disorders. Protocol treatments or interventions are in danger of missing the subtle individualised meaning based attributions, that appear so integral in the persistence of distress. It seems logical that individual attribution will play a role in post RTA anxiety and depression, as well as PTSD. This would fit with the cognitive conceptualisations of anxiety (Clark, 1986) and depression (Beck, 1979).

#### 4.7 Methodological Limitations

Empirical research on a population of RTA victims is not without problems. As noted in the introduction chapter, the adoption of a longitudinal prospective research design solves methodological issues arising from retrospective analyses. Similarly, recruiting a sample of consecutive attenders to hospital allows for as naturalistic as possible a population, avoiding difficulties inherent in samples of RTA victims presenting as litigants, or to request psychological help. However, a number of methodological weaknesses remain. Individuals involved in an RTA that does not lead to hospital attendance have not been sampled. In some circumstances ambulance crews called to the scene of an RTA recommend that victims attend their GP surgery in the days following an accident, or perform roadside treatments. Given the lack of predictive power in this and other studies, of accident severity in leading to PTSD, it may be that a number of individuals who are not badly hurt

subsequently develop PTSD. As a result of the design of this study, such individuals have been excluded from examination.

A further sampling difficulty is that of response rate. As noted above, 60 individuals of 352 contacted (17%) responded to a letter inviting them to take part in the research study. Therefore, a very large proportion of RTA victims have elected not to take part, meaning that conclusions from the current study cannot be generalised to all RTA victims. Based on prevalence figures found in this study, it would be expected that approximately 13% of the 292 individuals who failed to respond (38) would be diagnosable with PTSD at 4 months post-RTA. However, if the non-respondents were characterised by individuals with very low levels of psychological symptoms post-RTA, it would be expected that 13% would be an over-estimate of PTSD. Baseline symptoms have been shown to be highly predictive of PTSD at follow-up. Alternatively, it may be that a number of individuals who are experiencing high levels of psychological distress and intrusive phenomena when they are contacted regarding the research, choose to avoid it. Both behavioural and cognitive avoidance are characteristics of PTSD which will make sufferers less likely to take part in research. In such circumstances a prevalence rate of 13% would be an underestimate of PTSD in non-respondents.

The rate of attrition in the current study was 20%. In any prospective longitudinal research attrition is to be expected, but of more relevance is the nature of the group who dropped out. Descriptive analysis of the 11 respondents at baseline who failed to respond at follow-up indicated that their mean IES-B score was higher than the

baseline group as a whole. Also, 42.8% of individuals diagnosed with PTSD at baseline dropped out of the study. This indicates, in line with the predictive power of baseline symptoms for follow-up PTSD, that follow-up prevalence in this study may be an underestimation. Cognitive and behavioural avoidance may have played a key role in those participants who chose to drop out.

The predisposing, or characterological measures of ASI and AEE may have been contaminated by baseline distress levels. Multivariate analysis indicated that indeed ASI was significantly related to IES-B and that IES-B heavily mediated the predictive relationship between ASI and IES-F. Similar analysis of the AEE may have found this contamination. Methodologically this is important as the true effects of ASI and AEE may not have been found. In future research it would be prudent to measure ASI or AEE both at baseline and follow-up in order to assess for state/trait effects.

The non-predictive value of the AEE measure has already been discussed above. Another theory driven independent maintaining variable not to behave statistically as expected was anger. Anger has been proposed by Ehlers and Clark (2000) to act alongside other maintenance factors in both leading to a current sense of threat and in directly generating anxiety and depression. Ehlers and Clark (2000) suggest that anger cognitions will inhibit alterations in traumatic memory and lead to ruminative processes, also associated with PTSD persistence. In the current study although all three other hypothesised maintenance factors predicted PTSD and mediated the predictive roles of other variables, anger did not. This may be because anger in this

minor-RTA population was particularly low, meaning that it failed to be significantly predictive of PTSD. Alternatively, it may be that anger fails to function as suggested by Ehlers and Clark (2000) in this population. Further analysis of anger using well-standardised measurement tools would enable elaboration of the issues raised above. The non-standardised measures of anger used in this study may have failed to accurately measure this potentially important emotion.

The failure to find a predictive relationship between behavioural avoidance, as measured by an unstandardised question and subsequent PTSD may be due to methodological issues. In light of the predictive power of both cognitive avoidance and baseline symptoms (including behavioural avoidance) it is surprising not to have found a separate relationship for behavioural avoidance alone. The confounded nature of the question used may be responsible; individuals whose vehicle had been written off were technically behaviourally avoiding. In future research it is recommended that this issue be corrected.

Although many of the dependent and independent variables measured were done so using standardised, psychometrically robust (see research methodology) assessment tools, a number of key variables were not. Of particular importance in the context of both clinical and theoretical implications are the measures of rumination, thought suppression and negative interpretation of symptoms. These measures have been seen to act significantly and importantly in this sample, making clinical generalisations tempting, especially given the theoretical underpinnings (see Ehlers and Clark, 2000). The unstandardised assessment of these variables was conducted

in line with Ehlers et al. (1998), although other than the internal consistency reported here (Cronbach's alpha = .686) for the collapsed variable of all three measures, no psychometric analysis has been conducted. Ehlers and Clark have recently developed more comprehensive measurement tools (as yet unpublished) for these key variables and both psychometric analysis and adoption of these measures would be recommended in any future research. These newer measures were unfortunately unavailable for the current study.

The SRS-PTSD: DSM-IV measure has been seen to perform well psychometrically when compared to structured clinical interview (see research methodology). However, in order to maximise sensitivity and specificity of measurement of PTSD and related symptoms, formal clinician administered assessments could be incorporated into future research designs. Similarly, with greater resources and time it could be arranged to see respondents at more frequent and numerous time intervals, nearer to the RTA in the first instance and to extend follow-up to 6 months or a year. A larger sample size could be recruited without the constraints placed on the current study, maximising statistical power and minimising sampling error. Future research which is able to improve on these elements of research design would lend itself to more confident generalisation of results.

#### 4.8 Methodological Strengths

Despite the limitations outlined above the current study incorporates a number of robust methodological features, lending support to both results and subsequent conclusions. The adoption of a prospective longitudinal research

design, a sample of consecutive attenders to hospital and psychometrically standardised measures increase the confidence with which results can be interpreted. Furthermore, the use of theoretically driven hypotheses allow for results to be incorporated directly into current clinical and theoretical conceptualisations of PTSD.

#### 4.9 Implications for Future Research

In terms of theoretical direction for future research, it would be interesting to further explore the following issues. First, the relationship between disruption in autobiographical memory and the role of emotional expression in adaptive trauma recovery. The failure to find a role for negative AEE raises questions over how 'traumatic' a minor RTA is in terms of memory disruption. Second, further examination of anxiety sensitivity and investigation of how high AS may lead to catastrophic misinterpretations of symptoms and subsequent thought suppression. An in depth analysis of this hypothesised pathway to avoidance, although beyond the remit of the current study, would be of great theoretical interest and potential clinical utility. Finally, exploration of the nature of cognitive style implicated in the maintenance of PTSD, particularly the stability of catastrophic misinterpretation and the impact that stressful life events have on such attributional bias. This is related to the anxiety sensitivity issue and warrants further analysis. Is it that individuals who negatively interpret intrusive traumatic memories have a catastrophic attributional style predisposing them to such interpretation, or is this something particular to trauma?

#### 4.10 Conclusion

The current study found that in a sample of minor-RTA victims, assessed up to 4 months post-RTA, clinically significant levels of anxiety, depression and PTSD are present. Further examination of these dependent variables revealed that PTSD could be significantly predicted by a number of independent variables. Anxiety sensitivity, immediate post-traumatic reaction and peri-traumatic dissociation were all found to predict PTSD. Negative interpretation of symptoms, rumination and thought suppression (taken together) were found to heavily mediate the relationships of all these predictive factors with follow-up PTSD. These maintenance factors were the only variables to independently and significantly predict follow-up PTSD.

These results have a number of clinical and theoretical implications. It has been shown that even minor-RTA can lead to clinically significant psychological distress and that a number of predictive and maintaining factors can identify those individuals at risk in the few weeks post-RTA. Therapeutically, addressing maladaptive cognitive and behavioural strategies collaboratively with RTA victims should aid in the remittance of PTSD. Theoretical conceptualisations of PTSD that place emphasis on a disruption in autobiographical memory and on cognitive-behavioural maintenance of symptoms (e.g. Ehlers and Clark, 2000; Brewin et al., 1996) have been supported empirically. Finally, it has been shown that psychological factors are more important than environmental factors (e.g. accident/injury severity) in the development and persistence of PTSD. The implications for the manner in



which health services perceive, assess and treat RTA victims are significant.

There is clearly a need for a focus on psychological and emotional sequelae as

well as the more obvious physical impact.

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## APPENDICES

### 6.1 Cognitive Models of PTSD

One of the first authors to develop a cognitive model of PTSD was Horowitz (1976). Horowitz argues that the main impetus within the cognitive system for the processing of new information comes from a 'completion tendency'. Horowitz proposed that the completion tendency was the psychological need to match new information with existing mental models of older information and the revision of both until they agree. The theory posits that the completion tendency allows the mind to remain in accord with current reality, allowing for effective decision making and action.

Horowitz (1976) proposed that following trauma there is an initial 'crying out' or stunned reaction, followed by a period of information overload in which the thoughts, memories and images of the trauma cannot be integrated into existing models. As a result of this initial failure to complete, defensive mechanisms begin to operate in order to keep highly salient, incongruent trauma information in the unconscious; this is experienced by the individual as numbing and denial. Concurrently, Horowitz proposes, the completion tendency keeps trauma information in active memory, causing it to break through the defences and intrude into consciousness (e.g. flashbacks, nightmares and unwanted thoughts). It is suggested that this intrusion marks an attempt to integrate new trauma information into existing memory. During this processing stage an individual will experience an oscillation between intrusion and avoidance. A failure to complete this integration

may lead to trauma memory being 'stuck' in active memory, leading to chronic post-traumatic reactions.

The model proposed by Horowitz accounts well for the constellation of symptoms characteristic of PTSD and for the success of exposure based treatments. Similarly, it provides a good account of how normal reactions to trauma can become chronic. The model does not, however, explain why some individuals fail to complete trauma information integration, or how existing mental representations fail to accommodate new information. Furthermore, it is not clear that all individuals experience an initial period of denial (Dagleish, 1999). Creamer et al. (1992) propose that individuals experience an initial period of intrusions.

Janoff-Bulman (1985, 1992) proposed a cognitive appraisal theory of PTSD. Janoff-Bulman proposed that PTSD is the result of a 'shattering' of certain basic assumptions about the world, others and the self following a traumatic event. It is suggested that assumptions such as 'I am invulnerable' or 'The world is meaningful and comprehensible', provide structure and meaning to an individual, but that they cannot be maintained in the face of new traumatic experiences. Experiences that show the world to be cruel, unfair, uncontrollable or unpredictable may shatter these assumptions plunging the individual into a confusion of intrusions, avoidance and hyperarousal. Dagleish (1999) suggests that Janoff-Bulman's model is of importance, as it emphasises how traumatic information may be incongruent with usual beliefs and assumptions. However, it has been criticised. Dagleish (1999) notes that the model fails to explain how assumptions and beliefs are usually

represented and the process by which they are shattered. Furthermore, it is unclear how exposure-based treatments could relieve PTSD symptoms according to this conceptualisation. Finally, for those individuals whose pre-existing beliefs and assumptions are negative e.g. 'I attract trouble', Janoff-Bulman's idea of shattering may be inappropriate. For such individuals assumptions and beliefs are likely to be confirmed by traumatic experience.

Applying Lang's (1977; 1985) theory of fear structures, Foa (Foa and Kozak, 1986; Foa and Riggs, 1993; Foa et al., 1989, 1992), have developed an information processing theory of PTSD. The theory centres around a proposed fear network in long term memory, containing stimulus, physiological, cognitive and behavioural information relating to a traumatic event. The fear network is capable of being activated by trauma related cues or triggers, causing trauma information to intrude into conscious awareness (re-experiencing symptoms of PTSD). In response to such intrusion, individuals attempt to avoid and suppress trauma information, leading to the avoidance symptoms of PTSD. Foa et al. suggest that successful resolution of the traumatic event can only occur by integrating trauma information into existing memory structures. In order for integration to occur, it is proposed that activation of the fear network is necessary, in order that it is available for modification and that incompatible information from a safe non-traumatic environment may contaminate the fear network, altering the overall memory structure. Foa et al. posit that in severe trauma the memory traces that are laid down may be fragmented and disjointed, making integration into existing memory structures difficult. Dalgleish (1999) notes that this fear network model accounts



well for the constellation of PTSD symptoms, provides an explanation of the cognitive architecture involved, explains the role of pre-existing mental representations and accounts for the efficacy of exposure-based treatments. However, it remains unclear why fear networks develop in some individuals and not others.

The SPAARS (Schematic, Propositional, Associative and Analogical Representational Systems) approach to emotions is proposed by Dalglish and Power (1995) and Power and Dalglish (1996). SPAARS is a multi-representational model, in which two routes to the generation of emotions are specified. SPAARS is a functional theory of emotions. Within SPAARS emotions are seen as functional tools which the cognitive system employs to resolve problems with active, valued goals. For instance if a goal such as the maintenance of personal safety is threatened this will be appraised within SPAARS and a fear module will be activated, enabling the individual to deal with the current threat. Such a reconfiguration in this example would involve preparation for fight or flight, attentional bias for threat related information and activation of threat related concepts in memory. Similarly, if a goal is lost this will be appraised within SPAARS and a sadness module will be invoked. Thus, within SPAARS, emotions are seen as adaptive processes which reorganise the cognitive system in different ways to deal with changes in the internal or external environment. The suggestion is that the adaptiveness of emotions can sometimes go array and this can lead to the development and maintenance of emotional disorders, (Dalglish, 1999).

Dalgleish and Power (1995) describe four levels of representation. The analogical level stores visual, auditory, olfactory, gustatory and proprioceptive information, memories of specific events or representations of elements of events. The propositional level stores visual information such as beliefs, ideas, objects and concepts, meanings and semantic facts about the world, self and others. The sequence of events will be stored at the propositional level; what I was thinking, what I was doing etc. Dalgleish and Power (1995) note the similarities in the analogical and propositional levels with SAMs and VAMs respectively, as described by Brewin et al. (1996). The schematic level within SPAARS is analogous to what other authors have described as schemas (e.g. Bartlett, 1982), scripts (Schank and Abelson, 1977), frames (Minsky, 1975), mental models (Johnson-Laird, 1983) and the Implicational level of Interacting Cognitive Subsystems (Barnard, 1985, and Teasdale and Barnard, 1993). The schematic level acts as a guiding construct for the way information is processed and organised and is a higher order representation of knowledge, providing an individual with a sense of self, reality and meaning. This level of representation is also hypothesised to be concerned with an individual's hierarchy of goals, the highest level of which is the maintenance of the current configuration of dominant schematic models, to maintain the sense of self and a grasp on reality. As noted above, it is proposed that one route to the production of emotions is via the schematic level. Events are appraised at the schematic level with respect to the active goal structure. If a traumatic event occurs, threatening goals with disintegration of the self, fear will be generated in order to protect goals. The authors propose that the emotion takes over the system and reorganises it to deal with the goal discrepancies that have arisen.

The fourth level of representation in SPAARS is the associative level and also acts as the second route to the generation of emotions. SPAARS asserts that a useful distinction can be made between controlled and automatic cognitive processes and that automatic processing occurs via an associative level of representation, requiring no concurrent access to schemas. It is hypothesised that associative emotion generation is unrelated to schematic appraisal and is a function of an individual's emotional responses in the past; the result of emotion-event pairing. Dalglish (1999) also suggests that some events are biologically prepared to generate automatic emotion e.g. animal phobia (Seligman, 1971).

Utilising the SPAARS conceptualisation of emotion generation and cognitive architecture, Dalglish (1999) proposes a model of PTSD. At the time of trauma, information is appraised at the schematic level in a threat related way, generating fear. Concurrently, trauma information is encoded and represented at the analogical and propositional levels of meaning. If the traumatic event provides the individual with highly salient, incompatible information, that threatens existing schematic representations of the self, world and others as safe, predictable and controllable, the new trauma information will be poorly integrated with existing models at the stage of encoding.

Dalglish (1999), proposes that, in an attempt to assimilate and integrate new trauma information into existing models. The SPAARS system will allocate processing resources accordingly. As a consequence of this need for integration the

system will continue to appraise trauma information as a threat and as incompatible, until such time as compatibility can be achieved. Thus, an individual experiences constant activation and reactivation of the fear module. As a virtue of its 'activated' state, trauma information will intrude into consciousness e.g. intrusive thoughts and nightmares. Dagleish proposes that as a result of this chronic activation of the fear module, individuals will exhibit a number of cognitive-processing biases for trauma related information. Both internal and external cues will activate trauma information in memory therefore, and increase the chance of intrusions. Also, according to the theory, links between different aspects of trauma memory will be stronger than links between trauma memory and existing representations. Trauma memory as a whole, prior to assimilation, is seen as cohesive, and Dagleish (1999) proposes that this state will leave an individual vulnerable to the triggering of the whole memory e.g. flashback. The longer the state of disintegration lasts, the more the individual will experience automatic activation of fear via the associative level of meaning.

Dagleish (1999) accounts for the avoidance symptoms of PTSD. It is proposed that an individual experiencing intrusions of traumatic memory will recruit a number of protective mechanisms and processes. These may include a conscious attempt to avoid reminders of the trauma (behavioural) and strategies of thought suppression (cognitive). As regards hyperarousal symptoms, Dagleish suggests that the cognitive processing resources being utilised by the activation/reactivation of the schematic level fear module, leave the more sophisticated levels of appraisal less likely to operate. It is proposed that an individual 'pre-occupied' with trauma

processing will be more likely to be irritable and angry at actions, even when these actions were not deliberate or could not be helped; the more sophisticated mediating emotional processes are unavailable.

The SPAARS conceptualisation of PTSD attempts to explain why some individuals exposed to the same trauma develop PTSD, where others do not. Dalgleish (1999) suggests that the schematic models of self, other and world will differ across individuals due to different developmental experiences. Also, as schemas differ so will methods of dealing with information (such as traumatic experience), that is incompatible with existing models. Some individuals may have highly practised repression and denial such that incompatible information has rarely been integrated into schematic models. Others will emotionally process incompatible information (Rachman, 1980), such that assimilation occurs. Dalgleish suggests that most individuals possess schematic level representations in which the world is reasonably safe, the individual is reasonably safe and in which there is a high concordance between individual's actions and their consequences, (most goals are achieved). These 'reasonably' models are flexible and adaptive to be able to incorporate disparate information. Such individuals following a traumatic event will experience an initial period of post-traumatic reactions, or none at all, and will be able to integrate trauma information over the following few weeks or months. They will not develop PTSD (Dalgleish, 1999).

In contrast, individuals with overvalued models and assumptions will find it much more difficult to integrate disparate information. Such individuals may cope with

life using avoidance rather than emotional processing or integration, or may have led very safe and uneventful lives. Dagleish (1999) proposes that such individuals are highly vulnerable to extreme emotional distress following trauma and will have no way of defending against its impact. These individuals may be unable to resolve this tension and may abandon their old models of the world, others and self. Janoff-Bulman would describe this as 'shattered assumptions'. For those individuals who attempt to employ repression and avoidance strategies in the face of trauma, it may be that they are most at risk from delayed-onset PTSD. Dagleish suggests that these individuals will still experience some automatically generated emotions via the associative level, that their cognitive processing will be biased towards trauma information and that future stress or threat may lead to a breakdown in their defended coping. Finally, Dagleish notes that individuals with primarily damaged models of themselves may have their schematic models confirmed by the new trauma and they may experience increases in associated anxiety or depression as a result. It is hypothesised that the compatible nature of the trauma information will not lead to the constellation of PTSD symptoms characteristic of the emotional processing of incompatible information. Dagleish hypothesises that the problems of PTSD are one way in which individuals react with the world. Intense trauma, until it is resolved, is represented as a continuing threat, and intrusion, avoidance and hyperarousal are seen as highly adaptive ways of dealing with such circumstances.

## 6.2 Biological models.

Van der Kolk et al. (1984) propose a biological model based on the observation that PTSD shares many similarities with the animal model of inescapable shock. They

postulate that PTSD symptoms result from changes in neurotransmitter activity following trauma; initial overactivity of noradrenalin is thought to account for hyperarousal, exaggerated startle response and aggression and its subsequent depletion to account for anhedonia, social withdrawal and affective numbing. Van der Kolk et al. suggest that endogenous opiates released during re-exposure to trauma related stimuli result in stress induced analgesia. Subsequent depletion of the endogenous opiates is then experienced as aversive, setting up a cycle of behaviour in which an individual may seek exposure to stress repeatedly in an attempt to regain the analgesic effects. In their comprehensive review, Calhoun and Resick (1993) suggest that this biological model accounts poorly for the characteristic avoidance symptoms of PTSD and lacks empirical support.

Yehuda et al. (1991) present the Hypothalamic Pituitary Adrenal Dysfunction theory of PTSD. The authors suggest that PTSD is characterised by an altered Hypothalamic Pituitary Adrenal (HPA) system, in which HPA is deemed to be one of the major hormonal systems mediating stress response. The theory asserts that in PTSD the HPA system is underactive, where stress is usually associated with an overactivation of the HPA. Yehuda et al. account for this phenomenon by explaining how under circumstances of chronic stress the HPA may become underactive, perhaps as a compensatory mechanism to prevent harmful sequelae of chronically elevated glucocorticoid levels. The authors report that cortisol secretion was found to be lower than normal in soldiers exposed to prolonged threat of imminent attack. Yehuda et al. propose that HPA underactivity in PTSD reflects a unique psychopathology of the disorder and that both early contact with

environmental stress, or the post-traumatic reaction itself, may alter the response of the HPA system to subsequent stress. The authors posit that underactivation of the HPA leads to reduced basal cortisol levels, as seen in soldiers exposed to chronic stress.

In support of Yehuda et al. (1991), McFarlane et al. (1999) reports evidence of reduced cortisol levels in 26 individuals diagnosed with PTSD assessed soon after trauma. McFarlane et al. emphasise the role of cortisol in the consolidation of memory and refer to psychological conceptualisations of PTSD that advocate difficulties in initial memory encoding in the development of PTSD (e.g. Brewin et al., 1996). They propose that the reduced cortisol activity reflects underactivity of the HPA and call for future research to tease out the relative importance of prior experience, trauma and post-traumatic reactions in the alteration to HPA.

Siegel (1995), puts forward an alternative neuro-biological explanation of PTSD that has many, as yet untested, parallels with cognitive conceptualisations (e.g. Brewin et al., 1996; Dalgleish, 1999; Ehlers and Clark, 2000). Siegel suggests that the explicit processing of traumatic experiences may be specifically inhibited by at least two mechanisms; i) extreme stress may inhibit hippocampal functioning via the release of adrenocorticosteroids, and ii) divided attention, which is associated with impaired explicit yet intact implicit memory retrieval, may lead to impaired processing of certain features of the traumatic event. Siegel hypothesises that inhibition of the hippocampus during trauma, leads to an impairment in the memory consolidation process, where explicit memory cannot be properly embedded in the



associational cortex. If this was the case, traumatic memory would not be properly encoded, processed and consolidated and may therefore intrude into consciousness with an unresolved quality (e.g. reliving). This neurobiological explanation has many similarities to what other authors describe as poorly discriminated, contextualised and elaborated memory (Ehlers and Clark, 2000); situationally accessible memories (Brewin et al. 1996), and poorly integrated trauma memory, incompatible with existing models (Dalglish, 1999).

The conceptualisation put forward by Siegel (1995) also suggests that, although fear can be conditioned via the amygdala without any hippocampal functioning, the hippocampus plays a role in determining the specificity and context dependency of conditioned fear. The hippocampus provides context and meaning. Therefore, if hippocampal functioning is inhibited during trauma, conditioned fear will lack the temporal and contextual information the hippocampus would ordinarily provide. Siegel uses this explanation to account for the 'here and now' quality of fear reactions in PTSD and the tenacious, meaningless and inexplicable intrusions experienced. Again, there are parallels between Siegel's explanation and the associative level fear conditioning described in SPAARS (Dalglish, 1999). Siegel suggests that improvements in PTSD symptoms following therapy result from cognitive override mechanisms rather than the elimination of basic learning. This may rely on hippocampal and cortical processing to override established amygdala circuits, (Siegel, 1995). The therapeutic benefits of elaborating on raw traumatic memories, giving them meaning and context (Ehlers and Clark, 2000), or developing detailed VAMs capable of inhibiting SAMs (Brewin et al., 1996), could

be seen as similar versions (explanations) of the same process. It remains to be seen how much explanatory power biological models of PTSD contain and how cognitive and biological explanations may compliment each other.



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**Ref: 99-13**

Mr B. J. Smith  
Clinical Psychologist in Training  
Clinical Health Psychology, UCL  
3 Waterloo Terrace  
Islington  
LONDON N1 1TQ

22nd April 1999

Dear Mr Smith,

**99-13 Predictors of Poor Psychological Morbidity after minor road traffic accidents**

I am pleased to inform you that the above named project has been approved.

Approval is for two years from the date of this letter. Extension of this period will be dependent on the submission of a brief synopsis of the project together with an estimation of the time required for its ultimate completion.

The Ethics Committee will require an annual report on the progress of the study, and a copy of the completed study together with any consequent publication. In addition, the Committee must be informed, by the completion of the relevant form, of any untoward or adverse events which occur during the course of the study. The person who provided independent review of the original protocol should also be sent information regarding adverse events.

The Ethics Committee must be informed of, and approve, any proposed amendment to your initial application which has a bearing on the treatment or investigation of patients or volunteers.

A copy of the patient consent form and information sheet must be lodged in the clinical notes.



Mr Smith  
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Furthermore, whilst I am sure that every effort is already made to preserve the confidentiality of any patient information used in this study, could you please ensure that the team of investigators are aware that everyone who has access to patient information appreciates the importance of maintaining confidentiality particularly in respect of the use of computers and the statutory regulations laid down in the Data Protection Act 1998.

In terms of the managerial and financial implications associated with the study, where these relate to additional costs for the Trust, Mr Rob Hurd (Management Accountant, Finance Department, Whittington Hospital), will be in contact with you to discuss the Trust's schedule of charges for research projects. **Approval of these issues must be obtained from your general manager before the study commences.**

**In any correspondence regarding the study please quote the above Ethics Committee reference number.**

Yours sincerely

A handwritten signature in black ink, appearing to read 'John Farrell', written over a horizontal line.

Mr John Farrell  
**Chairman - Local Research and Ethics Committee**

## Information Sheet.

I would like to invite you to participate in a research project looking at how people react to being in road accidents. Unfortunately, road accidents are common and research is needed to help us understand how best to treat those people who have been involved. As someone who has recently been involved in an accident your opinions and experiences will be of great benefit, whether or not you feel particularly affected by the accident.

If you do choose to participate you will be asked to provide brief information about your recent accident, and will go through a few questionnaires concerning your responses to the accident. You will be asked to fill in these questionnaires on two occasions, 3 months apart. I will be available at all times to discuss any issues raised. The questionnaires can easily be completed at home.

All the information you provide will be completely confidential, and I will be the only person who has access to it.

It should be emphasised that you do not have to take part in this study if you do not want to. If you decide to take part you may withdraw at any time without having to give a reason. Your decision to take part will not affect your care and management in any way.

Yours Sincerely,



Ben Smith.  
Clinical Psychologist in Training .

**CONSENT FORM.**

**ROAD ACCIDENT RESEARCH 1999.**

Have you read the information sheet about this study ? Y / N.

Have you had an opportunity to ask questions and discuss this study ? Y / N.

Have you received satisfactory answers to your questions ? Y / N.

Have you received enough information about this study ? Y / N.

Which doctor have you spoken to about this study ? .....

Do you understand that you are free to withdraw from this study,

- at any time ? Y / N.
- without giving a reason for withdrawing ? Y / N.
- without affecting your future medical care ? Y / N.

**Do you agree to take part in this study ? Y / N.**

**Participant's Signature .....**

**Investigator's Signature .....**

**Personal Information.**

**Please answer the following questions as accurately as possible:**

Name : .....

Age : .....

Gender : M / F

Occupation : .....

Ethnicity : .....

Marital Status : .....

How long have you been a qualified driver ? .....

Have you had any previous accidents, and if so how many ? .....

Have you ever seen a therapist or counsellor in the past ? .....

Do you think you are the sort of person who usually worries a lot ? .....

**Accident Information.**

**Please answer the following questions about your accident as accurately as you can.**

Did you sustain any injuries during the accident, and if so what were they ? .....

Who was responsible in your opinion ? .....

What was the damage to property ? .....

Was anyone else injured ? .....

Were you alone in the car ? .....

Did the thought go through your mind, " This is it, I'm going to die!" ?.....

Did you think others lives were threatened ? .....

Do you plan to undertake legal action regarding the accident ? .....

**For the following questions please circle the answer that is most appropriate for you.**

How frightening was the accident ?

1. Not frightening    2. Quite frightening    3. Very frightening    4. Extremely frightening.

Were you suffering from emotional problems prior to the accident ?

1. Not at all    2. A little    3. A lot.

After the accident how angry were you ?

1. Not at all    2. A little    3. More than a little    3. A lot    4. Extremely.



To what extent are you physically recovered from the accident ?

1. Fully recovered
2. Still minor problems
3. Still major problems.

What has been the financial impact of the accident ?

1. None
2. Minor impact
3. Major impact.

When you have recollections of the accident how often do you think, "I must be going mad" or "I'll never get over this" ?

1. Never
2. Sometimes
3. Often
4. Always.

How often do you dwell on memories of the accident ?

1. Never
2. Occasionally
3. Daily
4. More than once a day.

When you remember the accident do you think "Why did this happen to me?" ?

1. No, never
2. Sometimes
3. Often
4. Always.

Do you try to push memories of the accident out of your mind when they occur ?

1. No, never
2. Sometimes
3. Often
4. Always.

When you remember the accident do you feel angry ?

1. No
2. A little
3. A lot
4. Extremely.

When you remember the accident do you think "Others have harmed me" ?

1. No, never
2. Sometimes
3. Often
4. Always.

### Driving Questionnaire.

Please answer either yes or no to the following questions by circling either Y or N.

- Are you anxious about travelling in cars ? Y / N
- Have you driven since the accident ? Y / N
- When you are driving do you experience any physical signs of anxiety, such as muscular tension, quick-shallow breathing, palpitations or sweating ? Y / N
- When you are driving are you worried another accident will happen ? Y / N
- Do you think the probability of another accident occurring is high ? Y / N
- Do you avoid driving when it is not essential ? Y / N
- Do you avoid driving under certain conditions, such as at night, on certain roads, in the wet or in heavy traffic ? Y / N

### Passenger Questionnaire.

- Have you been a passenger in a car since the accident ? Y / N
- When you are a passenger do you experience any physical signs of anxiety, such as muscular tension, quick-shallow breathing, palpitations or sweating ? Y / N
- When you are a passenger are you worried another accident will happen ? Y / N
- Do you think the probability of another accident occurring is high ? Y / N
- Do you avoid being a passenger when it is not essential ? Y / N
- Do you avoid being a passenger under certain conditions, such as at night, on certain roads, in the wet or in heavy traffic ? Y / N
- Do you distract yourself when travelling as a passenger ? Y / N
- Do you find yourself "back seat driving" ? Y / N
- Do you find you feel anxious at the thought of travelling in a car ? Y / N

### Impact Of Event Scale-Revised

**INSTRUCTIONS:** Below is a list of comments made by people after stressful life events. Please check each item, indicating how frequently these comments were true for you **DURING THE PAST SEVEN DAYS** with respect to the event. If they did not occur during that time, please mark the "not at all" column.

	Not at all	Rarely	Some- times	Often
1. Any reminder brought back feelings about it.	0	1	3	5
2. I had trouble staying asleep.	0	1	3	5
3. Other things kept making me think about it.	0	1	3	5
4. I felt irritable and angry.	0	1	3	5
5. I avoided letting myself get upset when I thought about it or was reminded of it.	0	1	3	5
6. I thought about it when I didn't mean to.	0	1	3	5
7. I felt as if it hadn't happened or wasn't real.	0	1	3	5
8. I stayed away from reminders about it.	0	1	3	5
9. Pictures about it popped into my mind.	0	1	3	5
10. I was jumpy and easily startled.	0	1	3	5
11. I tried not to think about it.	0	1	3	5
12. I was aware that I still had a lot of feelings about it, but I didn't deal with them.	0	1	3	5
13. My feelings about it were kind of numb.	0	1	3	5
14. I found myself acting or feeling like I was back at that time.	0	1	3	5
15. I had trouble falling asleep.	0	1	3	5
16. I had waves of strong feelings about it.	0	1	3	5
17. I tried to remove it from my memory.	0	1	3	5
18. I had trouble concentrating.	0	1	3	5
19. Reminders of it caused me to have physical reactions such as sweating, trouble breathing, nausea, or a pounding heart.	0	1	3	5
20. I had dreams about it.	0	1	3	5
21. I felt watchful and on-guard.	0	1	3	5
22. I tried not to talk about it.	0	1	3	5

### Impact of Event Scale—Revised

#### Scoring Information

Intrusion Subscale=sum of items 1, 2, 3, 6, 9, 16, 20

Avoidance Subscale=sum of items 5, 7, 8, 11, 12, 13, 17, 22

Hyperarousal Subscale=sum of items 4, 10, 14, 15, 18, 19, 21

Item response levels are:

0=Not at All 1=Rarely 3=Sometimes 5=Often

This instrument, by Daniel S. Weiss, may be reproduced without charge and freely distributed, as long as no funds are exchanged.

## APPENDIX

## Self-Rating Scale for PTSD (SRS-PTSD; DSM-IV)

*Questions About Effects of Traumatic Event*

Below are several statements that might be applicable to you *ever since* you experienced the traumatic event. Please fill in the  before the response that best describes your situation. Please bear in mind that we are asking about *the past 4 weeks*.

- 1a. I thought about the event regularly, even if I didn't want to.  
 not at all  
 less than four times a week  
 four or more times a week
- 1b. Sometimes images of the event shot through my mind.  
 not at all  
 less than four times a week  
 four or more times a week
- 2a. I repeatedly dreamed about the event.  
 not at all  
 once a week  
 twice a week or more
- 2b. Sometimes I woke up in a pool of sweat or screaming.  
 not at all  
 once a week  
 twice a week or more
3. I had the feeling I was reliving the event (or certain moments of it).  
 not at all  
 once  
 more than once
4. I felt very bad (sad, angry, scared, etc.) or got upset whenever I was reminded of the event, for example, by the radio, television, newspaper, people, or situations.  
 not at all  
 a little bit  
 very much
5. If I think about the event, it makes me feel bad physically. For instance, my chest aches, I shiver or perspire, I get nauseous or I get a headache.  
 not at all  
 a little bit  
 very much
- 6a. I did my best or forced myself not to think about the event.  
 not at all  
 a little bit  
 very much
- 6b. Which of the following have you done since the event? (You can fill in more than one response.)  
 drink more alcohol  
 use more drugs  
 gamble  
 take more medicine  
 escape by working a lot  
 stop working  
 not want to watch television any more  
 not want to read a newspaper any more  
 want to see fewer people  
 wander the streets
7. Ever since the disaster I have been avoiding people or things (such as shops, restaurants, movies, airports, parties) that remind me of the event.  
 not at all  
 a little bit  
 very much
- 8a. As regards the memory of the event:  
 I can remember everything very well  
 I can remember only a few details  
 I have no memory at all of a large part of it
- 8b. I had the feeling that the event was a bad dream, as if it did not really happen.  
 not at all  
 a little bit  
 very much
9. Ever since the event, I have not enjoyed or been interested in things I used to like such as hobbies or recreational activities.  
 not at all  
 a little bit  
 very much
10. Ever since the event, I have not been spending as much time with other people.  
 not at all  
 a little bit  
 very much
11. Ever since the event, I have felt less involved with other people; it is as if my feelings are not there any more.  
 not at all  
 a little bit  
 very much
12. Ever since the event, I have been pessimistic about my future. For example, I do not expect much from life, my job, or relationships with other people.  
 not at all  
 a little bit  
 very much
13. Ever since the event, I have had trouble sleeping. I have trouble falling asleep, or I wake up in the middle of the night and can't get back to sleep.  
 not at all  
 once or twice a week  
 three or more times a week
14. Ever since the event, I have been more apt to be impatient or lose my temper.  
 not at all  
 once every 2 weeks  
 more than once a week
- 15a. Ever since the event, I have been having trouble concentrating, for example, on reading a book or the newspaper or on my work.  
 not at all  
 a little bit  
 very much
- 15b. Ever since the event, I have been more apt to forget things.  
 not at all  
 a little bit  
 very much
16. Ever since the event, I have felt less at ease or less safe.  
 not at all  
 a little bit  
 very much
17. Ever since the event, I have been more nervous and more jumpy, for instance if I hear an unexpected sound.  
 not at all  
 once every 2 weeks  
 more than once a week

Name:

Date:

Doctors are aware that emotions play an important part in most illnesses. If your doctor knows about these feelings he will be able to help you more.

This questionnaire is designed to help your doctor to know how you feel. Read each item and place a firm tick in the box opposite the reply which comes closest to how you have been feeling in the past week.

Don't take too long over your replies: your immediate reaction to each item will probably be more accurate than a long thought-out response.

Tick only one box in each section

I feel tense or 'wound up':

- Most of the time .....
- A lot of the time .....
- Time to time. Occasionally .....
- Not at all .....

<input type="checkbox"/>	<input checked="" type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I feel as if I am slowed down:

- Nearly all the time .....
- Very often .....
- Sometimes .....
- Not at all .....

<input checked="" type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I still enjoy the things I used to enjoy:

- Definitely as much .....
- Not quite so much .....
- Only a little .....
- Hardly at all .....

<input checked="" type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I get a sort of frightened feeling like 'butterflies' in the stomach:

- Not at all .....
- Occasionally .....
- Quite often .....
- Very often .....

<input type="checkbox"/>	<input checked="" type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I get a sort of frightened feeling as if something awful is about to happen:

- Very definitely and quite badly .....
- Yes, but not too badly .....
- A little, but it doesn't worry me .....
- Not at all .....

<input type="checkbox"/>	<input checked="" type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I have lost interest in my appearance:

- Definitely .....
- I don't take so much care as I should....
- I may not take quite as much care .....
- I take just as much care as ever .....

<input checked="" type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I can laugh and see the funny side of things:

- As much as I always could .....
- Not quite so much now .....
- Definitely not so much now .....
- Not at all .....

<input checked="" type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I feel restless as if I have to be on the move:

- Very much indeed .....
- Quite a lot .....
- Not very much .....
- Not at all .....

<input type="checkbox"/>	<input checked="" type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

Worrying thoughts go through my mind:

- A great deal of the time .....
- A lot of the time .....
- From time to time but not too often .....
- Only occasionally .....

<input type="checkbox"/>	<input checked="" type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I look forward with enjoyment to things:

- As much as ever I did .....
- Rather less than I used to .....
- Definitely less than I used to .....
- Hardly at all .....

<input checked="" type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I feel cheerful:

- Not at all .....
- Not often .....
- Sometimes .....
- Most of the time .....

<input checked="" type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I get sudden feelings of panic:

- Very often indeed .....
- Quite often .....
- Not very often .....
- Not at all .....

<input type="checkbox"/>	<input checked="" type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I can sit at ease and feel relaxed:

- Definitely .....
- Usually .....
- Not often .....
- Not at all .....

<input type="checkbox"/>	<input checked="" type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I can enjoy a good book or radio or TV programme:

- Often .....
- Sometimes .....
- Not often .....
- Very seldom .....

<input checked="" type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

Do not write below this line



DATE \_\_\_\_\_

is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY, by placing an X in the corresponding space in the column next to each symptom.

	NOT AT ALL	MILDLY It did not bother me much.	MODERATELY It was very unpleasant, but I could stand it.	SEVERELY I could barely stand it.
1. Numbness or tingling.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
2. Feeling hot.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3. Wobbliness in legs.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
4. Unable to relax.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
5. Fear of the worst happening.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
6. Dizzy or lightheaded.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
7. Heart pounding or racing.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
8. Unsteady.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9. Terrified.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
10. Nervous.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
11. Feelings of choking.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
12. Hands trembling.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
13. Shaky.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
14. Fear of losing control.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
15. Difficulty breathing.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
16. Fear of dying.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
17. Scared.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
18. Indigestion or discomfort in abdomen.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
19. Faint.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
20. Face flushed.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
21. Sweating (not due to heat).	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

THE PSYCHOLOGICAL CORPORATION  
HARCOURT BRACE JOVANOVICH, INC.

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7 8 9 10 11 12 A B C D E

09018425

A S I . . .

Below is a list of reactions that might occur when you are anxious or nervous. Please mark to what extent these reactions worry or affect you.

1. Very little
2. A little
3. Some
4. Much
5. Very much

- \_\_\_ 1. It is important to me not to appear nervous
- \_\_\_ 2. When I cannot keep my mind on a task I worry that I might be going crazy
- \_\_\_ 3. It scares me when I feel 'shaky'(trembling)
- \_\_\_ 4. It scares me when I feel faint
- \_\_\_ 5. It is important to me to stay in control of my emotions
- \_\_\_ 6. It scares me when my heart beats rapidly
- \_\_\_ 7. It embarrasses me when my stomach growls
- \_\_\_ 8. It scares me when I am nauseous
- \_\_\_ 9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack
- \_\_\_ 10. It scares me when I become short of breath
- \_\_\_ 11. When my stomach is upset I worry that I might be seriously ill
- \_\_\_ 12. It scares me when I am unable to keep my mind on a task
- \_\_\_ 13. Other people notice when I feel 'shaky'
- \_\_\_ 14. Unusual body sensations scare me
- \_\_\_ 15. When I am nervous, I worry that I might be mentally ill
- \_\_\_ 16. It scares me when I am nervous





## PDEQ

*Instructions:* please complete the items below by circling the choice that best describes your experience and reactions *during the \_\_\_\_\_ and immediately afterwards.*  
If an item does not apply to your experience, please circle "Not at all true".

1. 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.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

2. I found that I was on "automatic pilot" - I ended up doing things that I later realised I hadn't actively decided to do.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

3. My sense of time changed - things seemed to be happening in slow motion.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

4. What was happening seemed unreal to me, like I was in a dream or watching a movie or play.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

5. 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 outsider.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

6. There were moments when my sense of my body seemed distorted or changed. I felt disconnected from my body, or that it was unusually large or small.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

7. I felt as though things that were actually happening to others were happening to me - like I was being trapped when I really wasn't.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

8. I was surprised to find out afterwards that a lot of things had happened at the time that I was not aware of, especially things I ordinarily would have noticed.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

9. I felt confused; that is, there were moments when I had difficulty making sense of what was happening.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true

10. I felt disorientated; that is, there were moments when I felt uncertain about where I was or what time it was.

1	2	3	4	5
Not at all true	Slightly true	Somewhat true	Very true	Extremely true



Sub-Department of Clinical Health Psychology

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**Dear**

**Re : Road traffic accident research.**

I would like to invite you to participate in a research project looking at how people react to being in road accidents. Unfortunately, road accidents are common and research is needed to help us understand how best to treat those people who have been involved. As someone who has recently been involved in an accident your opinions and experiences will be of great benefit, whether or not you feel particularly affected by the accident.

If you do choose to participate you will be asked to provide brief information about your recent accident and will go through a few questionnaires concerning your responses. You will be asked to fill in these questionnaires on two occasions, 3 months apart. I will be available at all times to discuss any issues raised. The questionnaires can easily be completed at home.

All the information you provide will be completely confidential and I will be the only person who has access to it.

It should be emphasised that you do not have to take part in this study if you do not want to. If you decide to take part you may withdraw at any time without having to give a reason. Your decision to take part will not affect your care and management in any way.

If you do decide to participate in this research into how people react to being involved in road accidents, or would like more information, then please contact me as soon as possible. You can do this easily by returning the enclosed reply slip in the pre-paid envelope.

**Thankyou in advance for your consideration.**

**Yours sincerely,**

**Ben Smith.  
Clinical Psychologist in Training .**

.....  
I ..... would like to take part in the above research / would like some more information and am happy for Ben Smith to contact me regarding this. Please note below the most convenient point of contact.

**Home telephone / fax :**

**Work telephone / fax :**

**e mail :**