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Potential Predictors of Post Traumatic Stress Disorder Symptoms in Spinal Cord Injury

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General Abstract

Traumatic spinal cord injury (SCI), resulting from tearing or severing of the spinal cord, has far reaching consequences both psychologically and physiologically that are often catastrophic to the individual. The individual, and his or her family, have to learn to adapt and adjust to circumstances that have changed drastically. The initial traumatic event that caused the injury can result in the onset of post traumatic stress disorder (PTSD), adding a further complicating factor to the adjustment process. The development of this disorder poses a significant problem for the SCI population, particularly in terms of rehabilitation, adjustment and long-term management of the injury. The literature review explores the history of theories of psychological adjustment to SCI and the relationship between SCI and PTSD.

The literature review explores what makes some people with SCI more psychologically vulnerable to PTSD than others. Knowledge of such vulnerability factors would help establish criteria by which to aid the identification of those at risk of developing PTSD and the development of treatment protocols.

The empirical paper investigates the presence of PTSD symptoms and potential predictors of PTSD within this population. High levels of PTSD symptoms were found. The study also found negative cognitive appraisals of self and neuroticism to be associated with the symptoms of PTSD for those with SCI.

List of Tables

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CONTENTS

Literature Review Paper

Psychological Implications of Spinal Cord Injury

Mal Bebbington Hatcher

Psychological Bulletin was used as a guide in determining the preparation of this paper (see Appendix 1 for notes to contributors).

Abstract

Spinal Cord Injury (SCI) results in a high cost disability that has enormous impact on life plans and expectations (Trieschmann, 1980). In terms of physical and psychological impact this complex form of trauma disrupts almost every system of the body.

Sustaining a SCI affects every aspect of the individual and his, or her, family's life. A number of theories have been put forward to explain psychological adjustment to SCI and this literature review highlights the empirical progress that has been made over the years.

The initial trauma that causes the injury, and the injury itself, can lead to the development of post traumatic stress disorder (PTSD), adding a further complicating factor to the adjustment process. The ability to identify risk factors that increase vulnerability to PTSD is important for facilitating the prevention, referral and treatment of the disorder. This review explores the relationship between SCI and PTSD by looking at the history of theories of psychological adjustment to SCI, exploring risk factors implicated in the development of PTSD and discussing psychological models of the disorder.

Key words: Post Traumatic Stress Disorder, PTSD, Spinal Cord Injury, SCI, Pre-, Peri- and Posttraumatic Predictors

Introduction

In terms of physical and psychological impact a spinal cord injury (SCI) is a complex form of serious trauma that causes loss of motor, sensory and autonomic function (Grundy & Swain, 1996). This form of trauma leads to disruption of almost every system of the body and is potentially life threatening (Whalley Hammell, 1995).

The spinal cord, a delicate bundle of nerve fibres that transport nerve messages, connects the brain to the rest of the body. Two pairs of nerve roots, consisting of a sensory and a motor root, connect to form mixed spinal nerves, which carry sensory and motor information to the arms, legs and trunk through defined pathways. Damage to these pathways results in sensory and motor loss, with loss of bodily sensations and movement.

Tetraplegia describes paralysis resulting from a SCI situated below the neck or top of the back, while paraplegia describes paralysis from an injury sustained below the waist. The neurological extent of the impairment, whether the cord is completely or partially damaged, determines the degree of weakness or paralysis experienced and impacts on functional expectations. Motor and sensory function is absent from below the level of injury with a complete lesion but preserved with an incomplete or partial lesion. The amount of preservation varies dependant upon the degree of impairment sustained. Damage to the spinal cord therefore affects many areas of the body (Appendix 2).

The number of SCIs sustained from wars and mining accidents have diminished in recent years while those from road traffic accidents have increased (Whalley Hammell, 1995). Tetraplegia has become far more common as a result of increasing sport and road traffic accidents while incidents of paraplegia have decreased, leading to both types of injury occurring on an almost equal basis (Oliver, Zarb, Silver, Moore, & Salisbury, 1998). It is estimated that between 900 and 1,000 SCIs occur in the UK each year (Whalley Hammell, 1995) with a far greater incidence among younger males (Partridge, 1994; Trieschmann, 1980), estimated at 82% compared to 18% of females (Whalley Hammell, 1995).

This is an overwhelming injury with distressing consequences. The goal for patients is not of complete medical recovery, which is presently an impossibility, but of accepting circumstances that have drastically altered (Whalley Hammell, 1995). For some patients experiencing the traumatic event that caused their SCI results in the onset of a specific anxiety condition, termed post traumatic stress disorder (PTSD), adding a further complicating factor to their adaptive adjustment to SCI.

The overall aim of this literature review is to explore the relationship between SCI and the development of PTSD by examining: the history of theories of psychological adjustment to SCI; PTSD in SCI; risk factors in the development of PTSD and discussion of psychological models of PTSD.

Adjustment to SCI

A number of theories have been put forward to explain psychological adjustment to SCI. The following section will discuss the main theoretical proposals. However, before beginning this section it is necessary to define the terms 'adjustment' and 'adaptation', which are used frequently throughout the review. Adjustment is defined here as "psychological balance or freedom from abnormality in face of pathological circumstances" while adaptation is defined as "the extent to which an individual can accommodate the demands of a stressful situation" (Pless & Pinkerton, 1975, as cited in Pit-ten Cate, 2003).

Stage theories of adjustment to SCI

Historically, psychological adjustment to SCI was based on a process of sequential stages. The view of early, descriptive models was that to achieve an effective outcome in psychological adjustment, the stages of adjustment had to be worked through sequentially (Trieschmann, 1980). The stage theories tended to view the adjustment process as being predictable, as well as sequential (Richards, 1986). The idea of a progressive chain of emotions unfolding to accommodate the temporal nature of recovery was appealing. The initial development of these stage models was largely informed by clinical observation and by bereavement (Engel, 1972; Parkes, 1971) and psychodynamic literature looking at issues of loss and mourning (Freud, 1957). Typically such models listed a series of changes that people go through when faced with loss and adjustment.

Tucker (1980) produced a five-stage model citing severe depression, anger, confusion, withdrawal and acceptance as the linear process patients with SCI had to proceed through to ensure optimum adjustment. Patients judged as 'stuck' at any stage were considered not to be proceeding adequately.

Most stage models considered depression to be a necessary and functional process in adjusting to SCI (Siller, 1969; Tucker, 1980), viewing depression as an inevitable post-injury reaction associated with mourning the loss of physical capacity (Gunther, 1971). Tucker found those patients with SCI who were depressed in the early stages of injury were more likely to demonstrate better long-term adjustment, although patients were most likely to get 'stuck' in the depression or anger stages of the model. Those patients who did not display depression were considered to be in denial (Siller, 1969) and it was even suggested that, for such patients, depression should be induced in order to facilitate the onset of appropriate grieving (Nemiah, 1957).

Because depression was considered by the stage models to be a critical element of the adjustment process it has been empirically studied in an attempt to examine these claims (Elliott & Kennedy, 2004).

Contrary to the early theories of depression as a beneficial and necessary process (Nemiah, 1957; Siller, 1969) Frank, Elliott, Corcoran and Wonderlich (1987) argued that depression might be a maladaptive reaction. Empirical studies have found depressive behaviour in those with SCI to be associated with psychological and medical complications (Elliott & Frank, 1996) including pressure sores, urinary

tract infections and contractures (Elliott & Frank, 1996; Herrick, Elliott, & Crow, 1994). An association has also been found between depression and self-neglect (Macleod, 1988) with depressed patients with SCI displaying poorer levels of self care and requiring more rehabilitation (Malec & Niemeyer, 1983).

Dinardo (1972) questioned whether depression was a necessary process in adjusting to SCI. His hypothesis, that the absence of a depressive reaction to SCI would correlate with adaptive psychological improvement, was supported in his study of 53 male patients with SCI. Those who had experienced depressive episodes were less adjusted throughout rehabilitation. Howell, Fullerton, Harvey and Klein (1981) evaluated 22 patients with recent onset SCI and found only 5 had diagnosable depression. Although higher than the incidence of depression in the general population this was less than had been anticipated. Hancock, Craig, Dickson, Chang and Martin (1993) compared 41 patients with SCI to 41 able bodied controls, matched on age, sex and education, and also found higher rates of depression amongst those in the SCI group (25%) compared to the control group (5%).

The above studies provide evidence that, contrary to the stage model theories, even though depression rates are higher than in the general population (Howell et al, 1981; Hancock et al, 1993) depression is not an inevitable reaction following SCI (Frank, Kashani, Wonderlich, Lising, & Viscot, 1985; Fullerton, Harvey, Klein, & Howell, 1981; Howell et al, 1981), and that, rather than being a necessary, functional process, depression in SCI is maladaptive (Frank, Elliott et al, 1987).

Rates of depression are also higher in those with SCI compared to levels found in

the rest of the medical population (Elliott & Frank, 1996). It is unclear if this difference in prevalence is due to the use of dissimilar diagnostic and interview techniques or differences between the populations (Elliott & Frank, 1996). Even within the SCI population there have been variable rates of depression noted. Studies using conservative diagnostic measures, such as clinical interviews based on DSM III criteria, have found the rate of major depressive episodes to range from 22.7% to over 30% (Frank et al., 1985; Fullerton et al., 1981) while lower rates have been observed in studies using less stringent methods, such as standardised self-report measures (11%; Frank et al, 1992). It has been mooted that higher rates of depression could be due to medical, social and functional challenges faced by those with SCI or to pre-injury factors including having a previous history of psychological disorders, alcohol or substance abuse (Elliott & Frank, 1996), highlighting the need for research to identify vulnerability and risk factors in this group (Kennedy & Rogers, 2000).

In addition to the above mentioned problems with diagnostic criteria, research into depression in the SCI population has been plagued by a number of methodological problems (Elliott & Kennedy, 2004) including the use of the Beck Depression Inventory (BDI; Beck & Steer, 1987). Many of the somatic symptoms experienced by individuals with a SCI, such as disruptions in appetite and sleep patterns, are the same as those listed in the BDI as symptomatic of depression (Frank, Elliott et al., 1987). However, the employment of measures of depression that emphasise somatic symptoms can inflate the scores of those who are physically ill or disabled (Elliott & Frank, 1996; Kennedy & Rogers, 2000), highlighting the need for the development and use of specific measures standardised for use with medical populations

(Overholser, Schubert, Foliart & Frost, 1993).

If patients go through a series of sequential stages in their adjustment to the SCI, one would expect greater levels of distress in those more recently injured, with less distress in those injured longer. Medical advances have resulted in people with SCI living longer (Krause & Crewe, 1987). Two aspects of aging, chronological age and time since injury, have been extensively studied to detect changes in adaptation to SCI but have produced conflicting results. Although studies have shown psychological and functional outcomes to be inversely correlated with age (Schulz & Decker, 1985; Eisenberg & Salz, 1991) and positively correlated with time since injury (Pentland & Twomey, 1994; McColl & Rosenthal, 1994) other studies have found no significant differences (Buckelew, Frank, Elliott, Chaney & Hewett, 1991; Crisp, 1992). Buckelew et al's (1991) study consisted of two separate groups of patients with SCI admitted to a rehabilitation centre. The first group was admitted during 1981 and 1982, the second during 1984 to 1986. Both groups were compared across age and time since injury. Contrary to the stage model theories neither age nor time since injury were related to psychological distress measures.

Studies employing a longitudinal design to investigate symptom change (Bracken & Shepard, 1980; Craig, Hancock, & Dickson, 1994; Hancock et al, 1993; Kennedy, Lowe, Grey & Short, 1995) have also found either no significant differences or only moderate changes over time. A more recent longitudinal study (Kennedy & Rogers, 2000) found a specific pattern in mood during the first 2 years following a traumatic SCI. There was a moderate decrease in depression and anxiety during the acute phase of hospital care, from the initial week of contact to week 18. From week 18

to week 48 there was a steady rise in scores resulting in scores at week 48 being above the clinical cut-off in the measures used for both depression and anxiety. Following discharge the scores on both decreased to a similar, although lower, range comparable to those identified in the acute phase. Although this result would appear to support the stage models theory that those recently injured would experience greater distress, the authors hypothesised that this reflected that patients become more depressed and anxious the longer they remained in the hospital environment.

This hypothesis would support Buckelew et al's study (1991), discussed above. When looking at their two separate groups over two distinct time periods Buckelew et al noted that higher levels of hostility and anxiety were reported in their later group. They hypothesised that this was due to the changes in acute medical care that occurred between 1982 and 1984, resulting in the patients in the later group spending less time in acute care before starting inpatient rehabilitation. The above studies highlight the need for future longitudinal studies to investigate the role of the hospital environment as a potential contributing factor in depression and the timing of treatment phases on adjustment to SCI.

A criticism of studies looking at age and time since injury in SCI concerns the limited range focussed on (Krause, 1992). Buckelew et al's (1991) study only compared patients who were between 1.7 and 3.6 years post injury. Kennedy and Rogers (2000) longitudinal study looked at patients from immediate contact in the acute setting to 2 years post discharge into the community. Although these studies increase knowledge they do not address issues of ageing within this population. Restricting the range of observation, to shortly after injury or a decade or more

since injury, results in difficulty in identifying any significant relationships over time. Such relationships need to be established to clarify how increasing life expectancy and broad societal changes in this group has influenced adjustment.

Although empirical studies reveal the stage model theories to be flawed, a number of methodological criticisms have also been levelled at the empirical studies, including small sample sizes, failure to separate out participants according to either time since injury or level of injury, or to operationally define what constituted depression in participants, so that future researchers could reliably replicate the studies (Frank, Elliott et al., 1987; Frank, Van Valin & Elliott, 1987).

Coping theories of adjustment to SCI

Because SCI usually results in a disabling condition, major changes tend to occur in the roles and activities undertaken by the individual. These changes are not always immediately apparent and therefore the individual is often involved in a process of adjusting to the changing circumstances brought about by their disability, rather than experiencing an immediate and permanent state of change (Trieschmann, 1980).

Before continuing with this section it is necessary to define the terms 'moderator' and 'mediator'. A moderator is defined here as a variable that plays a role in the association between two other variables by increasing or decreasing the structure of their relationship. For example, the presence of negative attributions (moderator variable) could lead to a strengthening of the association between the variables

'distress' and 'depression'. A mediator is defined as a variable that effects a transition between one variable and another. For example, negative appraisals (mediator) may generate distress thus leading to increased depression (Baron & Kenny, 1986; Backenstrass et al., 2006).

The challenges made to the validity of stage models proposing a sequential process of adjustment after SCI have led to empirical studies focussing on the psychological factors that influence effective coping.[1]

Factors found to influence recovery from depression following a SCI include the use of active coping strategies and social support (Sherbourne, Hays, & Wells, 1995). Certainly, factors other than simply time since injury have been shown to influence psychological adjustment to SCI, including subject characteristics such as locus of control (Shadish, Hickman, & Arrick, 1981) and environmental factors such as social support (Schulz & Decker, 1985).

Buckelew et al (1991) suggested that patients' adjustment to their SCI may have more to do with their individual characteristics than with the amount of time that had elapsed since their injury. They recommended time should be spent identifying and developing an understanding of the characteristics most likely to aid adjustment, believing that identifying the characteristics of patients who coped well with the

[1] Although there is a wide literature on coping it is beyond the scope of this review to present a comprehensive discussion of coping theories. Instead this review will focus only on those areas considered relevant.

adjustment to their SCI would enable clinicians to enlighten those patients who struggle.

Folkman and Lazarus (1980) identified two general coping strategies used by people to reduce stress and moderate its emotional impact. Emotion-focussed coping (such as the use of humour, blame or anger) regulates stressful emotions while problem-focussed coping (such as seeking therapy or devising a plan of action) changes the person-environment relation causing the distress (Folkman, Lazarus, Dunkel-Schetter, DeLongis, & Gruen, 1986). Their research indicates that people use both strategies, sometimes simultaneously, to deal with most types of stressful events. The strategy used is determined not only by individual preference but also by the type of stressful event encountered. Problem-focussed coping tends to be used more with stressors that are perceived as being controllable (Mikulincer & Solomon, 1989), such as work or family related problems, while emotion focussed coping tends to be used more with stressors perceived as uncontrollable (Ben-Zur & Zeidner, 1995; Folkman & Lazarus, 1980; Mikulincer & Solomon, 1989) for example physical health problems such as SCI. Generally speaking, problem focussed coping strategies are considered more effective in dealing with stressful events, with the perceived amount of control an individual has over a stressful situation being more important that the actual control they have (Heaney, House, Israel, & Mero, 1995). More passive, avoidant coping strategies appear to be a psychological risk factor for adverse responses to stressful life events (Holahan & Moos, 1987). Reidy, Caplan and Shawaryn (as cited in Duff & Kennedy, 2003) found a strong positive correlation between depression and the use of escape and

avoidance coping strategies in those with SCI.

The decision regarding which coping strategy to use is influenced by the cognitive appraisal processes mooted by the transactional model of stress (Folkman, 1984) in which primary appraisal is the initial perception of threat to self and secondary appraisal is when the threat is brought to mind. Wineman, Durand and Steiner (1994) looked at coping behaviours in participants with either multiple sclerosis or SCI. They found a significant difference in coping strategies employed depending on illness uncertainty and the appraisal of life with a disability. The use of emotion-focused coping was associated with high uncertainty and with situations that were appraised as dangerous. In other situations problem-focused coping was used more frequently. Research has found that adopting emotion-focussed strategies that rely on avoidance are associated with emotional difficulties, including depression and anxiety (Duff & Kennedy, 2003).

A series of studies (Krause, 1992; Krause & Crewe, 1987) noted that those who died early after SCI tended to be more poorly adjusted to the social, psychological and vocational implications of their injury. Those who adjusted well to SCI during the rehabilitation phase continued to improve, irrespective of physical limitations, supporting Trieschmann's view of rehabilitation being a lifelong process of adjusting and adapting within one's own environment (Trieschmann, 1988).

Stage theories of adjustment held the primary predictor of psychological response to SCI to be acquiescence to loss rather than individual characteristics. Frank,

Umlauf, et al., (1987) disagreed. They found individuals vary in their coping style and personal resources prior to injury and that these factors, more than any other, moderate an individual's psychological response to SCI while Coyne and Holroyd (1982) found that response to catastrophic injury and illness tended to be mediated, in part, by cognitive processes.

This section has so far outlined empirical studies that have provided evidence that it is individual coping styles and personal resources that moderate psychological responses to SCI, which contrasts with the lack of evidence provided by the descriptive stage models. The next section will look at locus of control following SCI.

Locus of control

Studies have found that those with an internal attribution of control have lower levels of distress and depression and display more adaptive behaviours during rehabilitation (Rosenbaum & Raz, 1977; Shadish et al., 1981), while those at risk for developing psychological difficulties have a greater external locus of control (Chan, Lee, & Lieh-Mak, 1992, 2000).

A study using cluster analysis found two subgroups of people with SCI that used different coping styles and displayed different degrees of psychological distress (Frank, Umlauf et al., 1987). Participants in Cluster 1 had less effective coping strategies, evidenced in their increased psychological distress and prevalence of

depressive symptomatology compared to Cluster 2. Those in Cluster 2 had a stronger reliance on internal attributions of control. This has strong implications for treatment, helping those with SCI to develop internal attributions of control, and runs counter to clinical interventions derived from stage models, in which persons with SCI were encouraged to confront the reality and implications of their injury, learning to accept this with passive support from their clinician thereby placing emphasis on external attributions of control and of reliance on others (Frank, Umlauf et al., 1987).

Social Support

In addition to locus of control, social support has been found to moderate the effects of trauma in diverse populations (Andrykowski & Cordova, 1998; Carlier, Lamberts, & Gersons, 1997).

According to the buffering hypothesis under stressful circumstances social support might buffer the impact of events, possibly by enhancing perceived control (Cohen & Wills, 1985), so that those who perceive themselves as having high levels of quality support will have lower levels of distress (Lazarus & Folkman, 1984). Studies looking at individuals with SCI living in the community found that those who had greater satisfaction with social support were better adjusted to their injury, had lower levels of depression and fewer health problems (Anson, Stanwyck & Krause, 1993; Chase & King, 1990; Rintala, Young, Hart, Clearman & Fuhrer, 1992).

In contrast, lack of social support has been found to be integral to the maintenance

of depressive disorders, with those individuals with SCI who displayed depressed behaviour in the first six months post trauma failing to recover if their support network was minimal (Kishi, Robinson, and Forrester, 1994). Suicidal ideation and plans have also been associated with low levels of social support (Kishi & Robinson, 1996).

Coyne, Aldwyn and Lazarus (1981) reported that depressed persons elicit social support initially but then experience rejection and avoidance from others because of their antagonistic behaviour. This results in a downward spiral of depressive behaviour, social isolation and rejection.

This could compromise the process of hospital rehabilitation if staff-patient interaction is affected by the patient's behaviour impacting on the reciprocal behaviour of staff (Folkman & Lazarus, 1986). Frank et al (1986) investigating the reaction of staff listening to a tape-recorded male actor, imitating a depressed person with a spinal cord injury, revealed negative and hostile responses by staff and the induction of dysphoric mood among them. The researchers hypothesised that this negative attitude by staff may reflect a history of failure in being able to cure patients of their depression and might, therefore, leave staff vulnerable to decreased feelings of self-esteem.

Kennedy et al (1995) found that decreased social support after discharge from hospital corresponded to a reduction in the use of coping strategies concerned with social support. Richards (1986), however, found that although the process of adjustment continued after leaving hospital, the level of emotional distress

experienced by patients was far less than expected. This led Richards to surmise that it would have been those who experienced the most difficulty in navigating the adjustment process, post-discharge, who would come to the attention of researchers observing the process. Richards believes that such observations may have led to the belief that the actual process of post-discharge adjustment was a difficult one for patients to make, leading to the overestimation of those who experienced post-discharge distress.

A criticism however is that social support is often viewed as a unidimensional concept resulting in potential underlying relationships being concealed (Herrick et al., 1994). Studies looking at type of support, and who provided it, have found that not all social support is beneficial. Emotion-focussed support in those with SCI has been found to be associated with greater life satisfaction when provided by partners and children but not when provided by friends (Post, Ros, and Schrijvers, 1999). Problem-focussed support has been posited to be more relevant in the acute stages of SCI recovery (Post et al, 1999). Because social support is a multifaceted concept problems exist in the SCI population regarding its measurement, with the use of differing measures resulting in large methodological variations across studies (Alloway & Bebbington, 1987).

In summary, advances in medical treatment and procedures in SCI led to increased life expectancy (Whalley Hammell, 1995) along with a concomitant interest in the psychological adjustment to SCI (Krause, 1992). The early, descriptive models tended to focus on the direct relationship between SCI and psychological adjustment, with the injury itself being considered the primary factor influencing the

subsequent behaviour and experience displayed by the individual.

Implicit in the early stage theory models was the assumption that in order to adjust well individuals must go through a series of stages viewed as involving a number of naturally occurring phases. Depression and prolonged psychological distress were considered natural responses to the injury, which had to be experienced to ensure optimum adjustment to their disability. Time since injury was considered a critical factor (Mueller, 1962; Stewart, 1977). The final stage mooted in almost all of the stage theories was of acceptance (Siller, 1969; Tucker, 1980). From this it would be expected that individuals with SCI could expect less distress the longer the time since injury.

A number of researchers challenged the assumptions of the stage models, believing that individuals differ in their coping style and personal resources, and suggesting that these differences moderate a person's psychological response to SCI (Elliott & Frank, 1996; Frank, Elliott et al., 1987).

A main tenet of the stage theories was that SCI must lead to a period of depression and yet depression was never adequately defined in stage models (Howell et al., 1981). Contrary to the view that a depressive episode is adaptive, empirical studies have found that not only is it not an inevitable reaction to injury (Fullerton et al., 1981; Howell et al., 1981), it has been associated with increased mortality (Zimmerman et al., 1994), increased length of stay in hospital and decrease in functional improvement in rehabilitation (Malec & Neimeyer, 1983).

Empirical studies have found other variables, including social support (Kennedy et al., 1995), locus of control and active coping strategies (Frank, Umlauf et al., 1987), to influence adjustment to SCI.

SCI is clearly a traumatic event that impacts on all areas of the individual's life. Rather than viewing depression as an independent event it should be viewed as part of an interaction of processes experienced by the individual at risk of loss, or reduction of coping skills, social support systems, and interpersonal environments (Frank, Elliott et al., 1987).

Frank & Elliott (1987) proposed that SCI can deplete coping resources, including social and financial support, thus increasing vulnerability to distress. For persons with SCI the process of adapting to the number of changes needed, including learning new ways of eating, sleeping, socialising and performing self-care routines, requires great effort.

The demise of stage models as a way of understanding adjustment to SCI has led Frank and Elliott (1987) to suggest that Folkman and Lazarus's (1986) life stress model replace the stage models as a way of understanding adjustment.

An alternative to the Folkman and Lazarus model may be to regard the accident and the SCI as a severe traumatic event. As a result of the initial traumatic event that caused the SCI a complicating factor to the adaptive adjustment of SCI may be the development of PTSD, necessitating the need to consider models of PTSD when looking at the impact of SCI. The next section will explore the relationship

between SCI and PTSD.

SCI and PTSD

The psychiatric definition of PTSD requires exposure to a traumatic stressor in which 'the person experienced, witnessed or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others (to which) the person's response involved intense fear, helplessness, or horror' (American Psychiatric Association, 1994) (Appendix 3).

Medical advances have resulted in increasing numbers surviving life threatening traumas and serious physical injury. Such patients have been identified as being at greater risk of developing PTSD (Bennett & Brooke, 1999; Feinstein, 1993; Malt, 1988; Spindler & Pedersen, 2005; Van Loey & Van Son, 2003).

Given that the majority of SCI's in the UK are caused by traumatic accidents (Grundy & Swain, 1996), life threatening events resulting in serious physical injury and impacting on functional ability, it could be assumed that PTSD might be a significant problem for this population. In addition, having a SCI can lead to life-threatening secondary medical problems (Brown, 1992), thus potentially increasing the risk of PTSD for this group.

Development of PTSD as a psychological concept

One of the first articles in the medical literature on PTSD, published in 1866, discussed the psychological abnormalities displayed by people who had experienced microtraumas of the spinal cord following railway accidents (Lamprecht & Sack, 2002). The description of symptoms included amnesia for the trauma, displays of emotional distress, an inability to sleep and experiencing a vague sense of alarm. The explanation of concussion to the spine causing chronic inflammation of the spinal cord and membranes, thereby producing the symptoms, led to the concept of "railroad spine syndrome" (Lamprecht & Sack, 2002). Interest in the effects of trauma continued throughout the twentieth century. The inclusion of PTSD as a separate diagnostic category in DSM-III (American Psychiatric Association, 1980) was due largely to descriptive and empirical studies of the experiences of veterans of war (Robbins, 1997; Shalev, Peri, Canetti, & Schreiber, 1996; van der Kolk et al., 1996). The central feature in this diagnosis was the traumatic event (March, 1993; O'Brien, 1998), expected to evoke 'significant symptoms of distress in almost anyone'.

However, this concept of the stressor assuming aetiological importance was not supported by a number of studies that found it was the individual's emotional response to the traumatic event that ultimately had the greatest influence in determining outcome (Feinstein & Dolan, 1991; Horowitz, Weiss & Marmar, 1987; Wheaton, 1983). This led to a revised, two-part definition of PTSD in DSM-IV (American Psychiatric Association, 1994). Part A1 expanded the range of qualifying stressors while A2 required the emotional responses to this trauma to include

'feelings of intense anxiety, helplessness or horror'. The specific symptoms experienced after the trauma were categorised as 'persistent re-experiencing of the event, avoidance of traumatic and associated stimuli, and symptoms of hyperarousal'.

Prevalence rates of PTSD have been reported from a number of studies, the most common of which are those looking at the effects of exposure to trauma on military personnel. The National Vietnam Veterans Readjustment Study (Weiss et al., 1992) reported a prevalence rate for current PTSD of 15.2% for males and 8.5% for women and a prevalence rate for lifetime PTSD of 30.9% for men and 26% for women. Epidemiological research with civilians identified PTSD in approximately 10-20% of all those in a community sample exposed to a traumatic event (Breslau, Kessler, Chilcoat, Schultz, & Davis, 1998).

Although scant research attention has been given to PTSD following SCI (Kennedy & Duff, 2001) the prevalence of PTSD in this group ranges from 7.1% to 40% (Kennedy & Duff, 2001; Martz and Cook, 2001; Nielsen, 2003b; Radnitz et al., 1995). The differences reported can be explained by the different methodologies used, making comparisons across studies difficult.

It is important to note that most epidemiological studies of PTSD tend to have been undertaken in the United States and therefore may not be generalisable to European populations (Spindler & Pedersen, 2005). Regardless of the limitations the prevalence rates would appear to show that the occurrence of PTSD following a trauma is the exception rather than the rule.

Breslau & Kessler (2001) found that almost every one of the 2,181 people they interviewed had experienced a traumatic event of the type described in DSM-IV (American Psychiatric Association, 1994). This concurs with an earlier study that found an estimate of exposure approaching 90% (Breslau et al., 1998), raising the question of why so few go on to develop PTSD? Is PTSD a distinct disorder resulting from exposure to trauma or it is due to a combination of factors for example the experience of trauma combined with vulnerability factors?

Risk factors for the development of PTSD

The ability to identify risk factors that increase vulnerability to the development of PTSD is essential for facilitating the prevention, referral and treatment of the disorder. It is outside the scope of this review to explore all of these risk factors, therefore only those that research has identified as being important will be outlined. The research between PTSD and SCI is scant (Kennedy & Duff, 2001) but where research exists it will be acknowledged. Such factors can be classified into three areas: pre-traumatic, peri-traumatic and posttraumatic.

Pre-traumatic factors

Pre-traumatic factors are viewed as predisposing vulnerability factors that exist prior to the traumatic event. Research has highlighted important pre-traumatic risk factors include: demographic factors; personality traits; previous history of psychiatric disorder and prior exposure.

Demographic factors

Epidemiological studies have revealed higher rates of PTSD in females compared to males (Brewin, Andrews, & Valentine, 2000; Bryant & Harvey, 2003; Holbrook, Hoyt, Stein, & Sieber, 2002; Van Loey & Van Son, 2003). This difference was explained in one study by males reporting serious accidents as their traumatic event while females reported rape, assault or ongoing physical or sexual abuse (Breslau, Davis, Andreski, Peterson, & Schulz, 1997).

Lower levels of education and social economic status have also been identified as associated with PTSD development (Armenian et al., 2000; Brewin et al., 2000; Cohen & Roth, 1987; Holbrook, Hoyt, Stein, & Sieber, 2001; Ozer, Best, Lipsey, & Weiss, 2003) although not all studies found this (Breslau et al., 1997; Weaver & Clum, 1995).

Age is also related to PTSD development, with younger age at trauma associated with higher rates of PTSD among disaster victims (Finnsdottir & Elklit, 2002) and psychiatric patients (Neria, Bromet, Sievers, Lavelle, & Fochtmann, 2002). However, Brewin et al's (2000) meta-analysis of 85 data sets found younger age at trauma was only a risk factor in military, compared to civilian, populations.

Personality traits

Neuroticism has been found to be significantly correlated with symptoms of

PTSD (Holeva & Tarrier, 2001; Joseph et al., 1996; Lauterbach & Vrana, 2001; McFarlane, Clayer, & Bookless, 1997) and has been found to be a better predictor of posttraumatic morbidity than exposure to the trauma (McFarlane, 1989), although not all of these studies measured neuroticism before the trauma. Of those that did measure it some have found it to be significantly correlated with PTSD symptoms (van den Hout & Engelhard, 2004) while others have not (Lee, Vaillant, Torrey and Elder, 1995).

Alexithymia is the inability to label and express emotions due to a lack of emotional awareness (Taylor, 1994). A relationship between PTSD and alexithymia has been established (Krystal, 1982; Shipko, Alvarez, & Noviello, 1983) with one study finding a significant association with alexithymia and the severity of PTSD symptoms (Yehuda et al., 1997) suggesting this may be a risk factor for the development of PTSD. It has been hypothesised that the inability to express emotions may lead to the harbouring of emotional distress without the verbal outlet important in the psychological recovery from trauma (Brewin, Dalgleish, & Joseph, 1996; Dalgleish, 2004; Ehlers & Clark, 2000; Foa & Kozak, 1986).

Dissociation is a known peri-traumatic risk factor for PTSD (Ozer et al., 2003) and a number of studies have found a relationship between dissociation and alexithymia (Elzinga, Bermond, & Van Dyck, 2002; Grabe, Rainermann, Spitzer, Gänsicke, & Freyberger, 2000; Irwin & Melbin-Helberg, 1997; Modestin, Lötscher, & Erni, 2002). However it has been argued that this may be an artefact and actually due to depressed mood (Wise, Mann, & Sheridan, 2000) particularly as alexithymic features have been related to depressive symptoms (Honkalampi et al., 2001).

Role of psychiatric disorder

Having a personal or family history of psychiatric disorder is another factor that significantly predicts PTSD development (Breslau, 2002; Brewin et al., 2000; Ozer et al., 2003), particularly if the traumatic event involves interpersonal violence (Ozer et al., 2003). It is not clear whether the aetiology of psychiatric disorder is genetic/biological or environmental.

Prior exposure to traumatic events

Prior exposure to traumatic events is also associated with the development of PTSD (Brewin et al., 2000; Ozer et al., 2003) with exposure to different stressors potentially increasing the likelihood of a pathological response by sensitising the victim to the new stressor and overwhelming their coping resources (Dougall, Herberman, Delahanty, Inslicht, & Baum, 2000; Peretz, Baider, Ever-Hadani, & De-Nour, 1994; van der Kolk & Greenberg, 1987). Conversely, exposure to similar stressors may provide immunity to victims by habituating them to repeated traumas, with victims becoming more resilient and therefore reducing the incidence of posttraumatic stress (Anderson, 1968; Bolin, 1985; Norris & Murrell, 1988). However, the experience of interpersonal violence, either in childhood or as an adult, greatly increases the risk of chronic PTSD developing when the individual is exposed to future trauma of any kind (Breslau, 2002; Brewin et al., 2000; Ozer et al., 2003).

With a specific focus on prior exposure and PTSD in the SCI population, Radnitz and Schlein (2000) looked at previous combat exposure in American veterans. They found that the additive trauma led to the veterans experiencing greater difficulty in recovering from their SCI-related PTSD but no indication that prior trauma, in the form of combat exposure, predicted PTSD development.

Having outlined the pre-traumatic risk factors involved in the development of PTSD the same will now be done with peri-traumatic risk factors. These are the factors related to the experiences of the trauma itself, and include type and severity of trauma, threat to life and dissociation.

Peri-traumatic factors

Type and severity of trauma

Trauma intensity has been identified as a significant predictor of PTSD severity (Armenian et al., 2000; Brewin et al., 2000; Lauterbach & Vrana, 2001; Lee et al., 1995), and chronicity (Foy, Sipprelle, Rueger, & Carroll, 1984; March, 1993). With regard to those with SCI, trauma severity has been significantly related to self-reported PTSD symptoms (Radnitz, Hsu, Willard et al., 1998).

Interpersonal traumas such as rape, assault or torture have been shown to result in higher risk of PTSD (Breslau et al., 1998; Norris, 1992; Rosenman, 2002). Those whose SCI was caused by deliberate violence are at greater risk of PTSD than

those who acquire it accidentally (Radnitz, Hsu, Tirch et al., 1998).

Threat to life

Individuals perception of life threat during the traumatic event is associated with higher levels of PTSD (Kangas, Henry, & Bryant, 2005; Ozer et al., 2003). Perception of threat has been found to have a greater influence on distress levels than more objective indicators of violence such as weapons and injuries (Bernat, Ronfeldt, Calhoun, & Arias, 1998).

Dissociation

Dissociation involves psychological detachment from the traumatic event. Peri-traumatic dissociative responses emerged as the strongest predictor of PTSD symptoms (Ozer et al., 2003) particularly at 6 months post trauma when it explained 30% of the variance in PTSD symptoms (Shalev et al., 1996). It is mooted that dissociation indicates an inability by the trauma victim to fully process the traumatic event and accompanying implications thus promoting symptoms of PTSD (Brewin et al., 1996; Ehlers & Clark, 2000; van der Kolk & Fisler, 1995).

To conclude this section post- traumatic risk factors will now be outlined.

Posttraumatic factors

Posttraumatic factors include ongoing life stresses and major life events and are associated with the long-term course of the trauma response. Research has identified salient post-traumatic risk factors as loss, social support, injury and time since injury.

Loss

The loss of property and possessions due to trauma can lead to the disruption of personal relationships and normal routines, which amplify stress (La Greca, Silverman, Vernberg, & Prinstein, 1996). A study looking at loss as a determinant of PTSD in earthquake survivors (Armenian et al., 2000) found a dose-response relationship between amount of loss and risk of developing PTSD. The authors speculate that their finding of a higher risk of PTSD also being associated with low levels of education may reflect the difficulty those participants had of accessing the social support and human resources provided by the community.

The loss, by death, of a family member or friend during a traumatic event is believed to be a particular risk factor for PTSD, especially if the death was witnessed (Pynoos & Nader, 1988).

Social support

Strong social support has been shown to help people cope with life stresses more effectively (Cohen & Wills, 1985) and to lessen the impact of trauma (La Greca et al., 1996). Social support may be lost after a traumatic event for a number of reasons including death or injury of family members and friends (Vernberg, La Greca, Silverman, & Prinstein, 1996).

Although lack of social support is significantly associated with PTSD development (Brewin et al., 2000; Holbrook et al., 2001; Ozer et al., 2003), level of support and functioning prior to a trauma is not generally investigated (Ozer & Weiss, 2004). Lack of social support is a known associate of PTSD symptomatology in those with SCI (Danner & Radnitz, 2000; Nielsen, 2003a).

Injury

Injury and PTSD have been studied in both civilian and military populations with results indicating that moderate to high rates of PTSD have been found among the injured population (Koren, Arnon, & Klein, 1999; Mayou, Bryant & Duthie, 1993; Pitman, Altman, & Macklin, 1989) suggesting that traumatic injury may increase the risk of PTSD.

A recent study that directly compared injured and non-injured survivors of the same trauma found evidence that physical injury is a major risk factor for PTSD

(Koren, Norman, Cohen, Berman, & Klein, 2005). The same study also proposed that the probability of developing PTSD is eight times higher if injury is a consequence of the traumatic event.

Among veterans with SCI, type of SCI was found to be the most consistent predictor of PTSD diagnosis and severity (Radnitz, Hsu, Willard et al., 1998). Those with tetraplegia reported less symptoms of PTSD than those with paraplegia, hypothesised as possibly due to damage to the higher levels of the spinal cord impairing the action of nerve fibres and thus blocking the symptoms of pyschophysiological arousal associated with PTSD.

Time since injury

Time has long been researched as a factor associated with whether, or how, individuals adapt to injury. As noted in an earlier section of this literature review, adaptation to SCI was initially seen as a process of sequential, temporal adjustment to recovery (Richards, 1986; Trieschmann, 1980). Research into time since injury has continued, yielding conflicting results. Studies have found time since injury to reduce symptoms of anxiety and depression while increasing adaptive reactions such as acceptance and adjustment (Dijkers, 1999; Woodrich & Patterson, 1983) while others have not (Craig et al, 1994).

A more recent study (Livneh & Martz, 2003) looking at those with SCI, divided into two groups by time since injury (4 years and 20 years), found that those in the

more recently injured group had elevated levels of both anxiety and shock. This led the authors to hypothesise that intrusive, repetitive thoughts might be hindering the adjustment process.

Although all of the studies reported in this section provide some evidence of an association between potential predictors and development, or chronicity, of PTSD they do not clearly state the nature of that association and must therefore be interpreted with caution. Most traumatic events tend to be uncontrollable and unpredictable explaining the lack of prospective longitudinal studies that would present more conclusive evidence.

It has been proposed that cognitive factors are involved in mediating and maintaining PTSD, with the severity of PTSD symptoms being linked to dysfunctional cognitive appraisals about the self, the world and the nature of the traumatic memory (Brewin et al., 1996; Foa & Riggs, 1993; Horowitz, 1997; Janoff-Bulman, 1985). The following section will go on to look at three recent, influential cognitive models of PTSD which are underpinned by theories regarding the risk factors hypothesised to mediate and maintain PTSD. Among the SCI population, where the injury is likely to be a traumatic accidental event, any understanding of PTSD development will be important.

Psychological Models of PTSD

Emotion Processing Model

Information processing theories believe that how information about the traumatic event is integrated into memory determines whether psychopathology will result or not (Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988; Creamer, Burgess, & Pattison, 1992; Foa, Steketee, & Rothbaum, 1989; Litz & Keane, 1989). Lang (1979) proposed a fear network. The frightening event is encoded into memory as a network of associated concepts, tendencies or informational nodes. The different nodes hold three types of prepositional information structured into i) stimulus information about the event; ii) information about cognitive, emotional, physiological and behavioural responses to the event; and iii) information that links the stimulus and response elements together. Activation of one node will activate others. Lang hypothesised that those with anxiety disorders have fear memories that are easily activated by stimuli, even stimuli only vaguely associated with their fear memory.

Lang (1979) believed that prepositional information in the fear structure therefore integrates cognition and affect within a program designed to provide the individual with sufficient survival information to employ escape or avoidance behaviour. Foa and Kozak (1986) argued that the fear structure must contain information that cues the individual to recognise stimulus situations and/or responses as dangerous. They developed Lang's (1977, 1979) work further by attempting to distinguish

the difference between the structures of normal fears and the pathological fears encountered in anxiety disorders. They hypothesised that pathological fear structures include response elements such as flawed appraisals of threat, high fearfulness and extreme avoidance behaviours. They also hypothesised that the fear structures were resistant to modification, due partially to their rigid structure (Lang, 1977) but also possibly to impairments in the mechanisms that process fear-relevant information (Foa & Kozak, 1986).

To modify the overly strong associations in the fear network and reduce the fear experienced Foa and Kozak (1986) proposed that the fear network had to be activated, by exposure to fear-relevant stimuli, and then modified by integrating new elements, incompatible with fear, to change its fundamental structure and form a new memory. The two mechanisms believed to modify the fear network are the extinction of the fear reaction, by repeated exposure to the fear stimulus, and disconfirmation of pathological cognitions, particularly the belief that to experience the symptoms of anxiety would be dangerous and must therefore be avoided.

Foa and Kozak's network theory was developed further, into the emotional processing theory (Foa & Riggs, 1993; Foa & Rothbaum, 1998), to account for knowledge accumulated from empirical studies. They explained disorganised and amnesic memory of the trauma as resulting from disrupted and biased information processing during the trauma. Foa and her colleagues further hypothesised that two dysfunctional cognitions ('the world is extremely dangerous' and 'one's self is totally incompetent') mediated the development of PTSD. Foa and Rothbaum (1998) hypothesised that these dysfunctional cognitions are formed when

individuals who hold rigid views, positive or negative, about the safety of the world and the competence of self, encounter a traumatic situation. Rigid positive views would be disconfirmed by the event while rigid negative views would be confirmed, thus leaving the individual vulnerable to developing PTSD. Individuals with more flexible cognitions are able to interpret the trauma as a unique experience with little or no ongoing threat to self.

Foa and colleagues (1993, 1998) postulated that the emotional processing of negative appraisals of post trauma reactions to self, such as interpreting posttraumatic symptoms as a weakness, could inflate views of personal incompetence and maintain the posttraumatic state. Unsympathetic responses from others reinforce the view that the world is a dangerous place.

Levels of Representation Model

The Levels of Representation Model is one of the few neuro-science models of PTSD. Brewin et al (1996) proposed a model based on two memory systems; verbally accessible memory (VAM) and situationally accessible memory (SAM). The VAM system contains representations of the person's conscious experience of events pre, peri and post trauma, which are processed, stored and integrated with other non-trauma related memories. These fully contextualised, temporal representations can be voluntarily retrieved and modified when required. VAMS only contain information that has been consciously attended to. Under conditions of extreme stress or arousal information encoded is liable to contain significant gaps

(van der Kolk & Fisler, 1995).

The SAM system contains physiological responses to the trauma and representations of the nonconscious processing of the traumatic event. Although the representations are not usually consciously accessible, they are involuntarily recalled when the person is exposed to the context stimulus features, or meaning of an event similar to that of the traumatic situation. The context they are recalled in can be either internal (conscious thought processes) or external. These perceptual, affect laden memories break into consciousness in the form of flashbacks following exposure to trauma related stimuli and activation of the SAMs (Grey, Holmes, & Brewin, 2001).

Although both memory representations share a number of similarities, there are important differences. VAMs can be deliberately and progressively edited to create a more detailed representation of the trauma. They contain primary emotions experienced during the trauma and secondary emotions formed, post trauma, from cognitive appraisals of the event. SAMs are not subject to the same manipulation and processing capacity as VAMs and therefore remain unchanged. They do not have a verbal code and therefore cannot communicate with, or be updated by, other autobiographical knowledge. SAMs are difficult to control as they are activated by a wide range of cues that act as reminders of the event, leaving the individual feeling out of control.

Several studies have proposed that the relative functioning of the amygdala and the hippocampus, areas of the brain involved in the body's stress response, mediates the response to trauma (Metcalfe & Jacobs, 1998; Pitman, Shalev, & Orr, 2000; van

der Kolk, 1996; van der Kolk et al., 1996).

Talking the trauma through enables the hippocampus to make a full, rich, verbal account of the trauma. The hippocampus can then exert an inhibitory response on the amygdala. The hippocampus also provides the information that the trauma is in the past because the hippocampus processed VAM is updated and has a temporal context. Treatment comprises of activating the SAM through exposure, and integrating it with verbal, contextual and other relevant information, thereby creating a new VAM. Difficulties can arise if prior beliefs held by the individual are inconsistent with the information about the trauma contained in the SAMS. Treatment should then aim to cultivate new beliefs derived from both the prior beliefs and the trauma information.

This model proposes that original trauma memories remain extant and can still be reactivated, regardless of how well represented the new VAM trauma memory is. If the SAM memory contains information that does not correspond to information held in the VAM memory, by unexpectedly accessing detailed, specific reminders of the trauma, the individual can experience flashbacks of the event.

The same principal of retrieval competition is used to explain how negative cognitions about the self, the world and the future can be subjugated after therapy. By increasing the likelihood that positive identities are retrieved after activation of negative cognitions, the individual can feel profoundly changed following therapy. However, old negative cognitions remain unchanged and can still be reactivated in the future if the retrieval bias is weighted towards them.

The cognitive model proposed by Ehlers and Clark (2000) incorporates many of the ideas presented by the previous two models (Brewin et al., 1996; Foa & Kozak, 1986). The model provides an explanation of why people feel anxious about the future after experiencing a trauma. It proposes that those with PTSD process the traumatic event and/or its sequelae in a way that can lead to a persistent sense of current threat. How the trauma is appraised will determine whether it is viewed as a time-limited event or one that poses a continuous threat. The perceived threat can be external ('Nowhere is safe') or internal ('It was my fault'). These negative cognitive interpretations are thought to maintain PTSD by producing a sense of current threat accompanied by intrusions, arousal, and strong emotional responses. They also motivate the person to engage in dysfunctional behaviour and cognitive strategies such as thought suppression, rumination and safety seeking behaviour intended to control or reduce the perceived threat. These control strategies may exacerbate PTSD symptoms and maintain the problem by preventing change in the appraisal and trauma memory.

The nature of the trauma memory also contributes to the sense of current threat. Ehlers and Clark (2000) propose that the poor elaboration of trauma memory, and its weak integration into its context in time, place and other existing autobiographical memories, accounts for difficulties in intentionally recalling aspects of the trauma memory. It also explains the occurrence of re-experiencing symptoms (having no contextual information gives a 'here and now' quality to the memory) and emotional responses to trauma-related cues. Poorly elaborated memories make it difficult

for information that might disconfirm negative appraisals to be integrated, thus further maintaining the problem.

Ehlers and Clark (2000) posit a relationship between trauma memory and trauma appraisals. When people with PTSD recall the traumatic event, the appraisal they hold about the event influences retrieval so that only information consistent with that appraisal is recalled. This prevents information that contradicts the appraisal being retrieved thereby disconfirming, or changing, the appraisal. Conversely, if information cannot be retrieved, and this is appraised as indicating a serious problem, the sense of current threat is maintained.

Evaluation of the models

Reviewers have acknowledged that although all three of the cognitive models recognise the beneficial effects of exposure to the trauma memory and the cognitive restructuring of negative appraisals in aiding symptom reduction, each model provides a different explanation as to why this is beneficial, based on their specific theoretical predictions (Brewin & Holmes, 2003; Dalgleish, 2004).

Some of the risk factors can be explained by the models. All three models agree that disrupted or faulty processing of the traumatic event leads to disorganised and incomplete recall of the trauma memory. A risk factor that may contribute to this is dissociation during the trauma (Brewin et al., 1996; Ehlers & Clark, 2000; van der Kolk & Fisler, 1995).

Dysfunctional cognitive appraisals about the self and the world are involved in mediating and maintaining PTSD (Brewin et al., 1996; Foa & Riggs, 1993; Horowitz, 1997; Janoff-Bulman, 1985) and may be present in a number of risk factors such as perception of threat of life (I am no longer safe), prior exposure to interpersonal violence or the act of interpersonal violence during a traumatic event (This is happening because of me, who I am) or being female, which may lead to more traumatic events involving interpersonal violence such as rape, assault or ongoing physical or sexual abuse (Breslau et al., 1997).

Lack of social support or unsympathetic responses from others can reinforce appraisals that the world is a dangerous place or that the individual was to blame for the trauma, while a physical injury can be a constant reminder of the traumatic event. Those whose personalities tend to be more negative are more likely to access negative cognitive appraisals of the event while those with alexithymia may struggle with the emotional processing of the trauma.

Coping and PTSD

As PTSD is a chronic disorder it is important to identify coping behaviours that influence PTSD symptomatology (Lazarus, 1966). Coping, defined as the thoughts and acts that people use to manage the internal and/or external demands posed by a stressful encounter (Folkman & Lazarus, 1986, p108), is critically related to adjustment following a wide range of severe life stressors (Benotsch et al., 2000; Lazarus & Folkman, 1984; Moos & Schaefer, 1993; Solomon, Mikulincer, &

Arad, 1991; Wolfe, Keane, Kaloupek, Mora & Wine, 1993).

As presented in the SCI literature earlier, adopting emotion-focussed strategies which rely on avoidance can lead to emotional difficulties, including depression and anxiety (Duff & Kennedy, 2003). Similar results have been found within the PTSD literature, with avoidance coping being associated with more PTSD symptoms (Benotsch et al, 2000; Solomon, Mikulincer and Flum, 1988) and greater PTSD severity (Moos & Schaefer, 1993; Bryant & Harvey, 1995; Sutker, Davis, Uddo, & Ditta, 1995). A criticism of the studies, however, is that although they looked at the orientation of coping (avoidance versus approach), they did not differentiate between the method of coping used (cognitive or behavioural). A recent study (Tiet et al, 2006), looking at method of coping, found that using cognitive avoidance coping predicted more PTSD symptoms. They hypothesised that this result might be due to attempts to deny or suppress the severity of the problem leading to greater post-trauma intrusions, and that therefore interventions based on reducing cognitive avoidance coping may lessen future PTSD symptoms.

These findings concur with cognitive models of PTSD, which hypothesise that attempts to control increasing anxiety leads to the use of avoidant coping strategies, paradoxically increasing symptoms of reexperiencing and hyperarousal (Ehlers & Clark, 2000; Brewin & Holmes, 2003).

Avoidant coping strategies have been linked to poorer physical health status in those with PTSD (Beckham et al, 1998; Kimerling, Clum & Wolfe, 2000), even after controlling for the effects of age, health behaviour and gender (Lawler, Ouimette,

Dahlstedt, 2005), and more problems with adherence to treatment (Aldwin & Yancurra, 2004). Avoidance coping has been proposed to partially mediate the association between PTSD and poorer physical health (Schnurr & Green, 2004). This has implications for those with SCI, particularly as having a SCI can lead to life-threatening secondary medical problems (Brown, 1992).

Although higher levels of depression have been found in SCI patients with PTSD compared to SCI patients without PTSD (Kennedy & Evans, 2001; Nielsen, 2003b) the fact that both experience depression raises the question of whether PTSD and depression are two distinct reactions following SCI (Nielsen, 2003b). The higher levels of depression noted in those with PTSD may be explained by symptom overlap between the two diagnoses (Nielsen, 2003b).

Kennedy and Evans (2001), evaluating PTSD in the first 6 months after SCI, found depression to be significantly related to symptoms of intrusion and avoidance. Future research should investigate whether PTSD and depression are both reactions to the initial trauma or if depression is a risk factor for developing PTSD following SCI (Nielsen, 2003b). The prevalence of PTSD among those with SCI is comparable to the prevalence of depression in this population, making the identification of both a prime necessity for ensuring essential intervention.

To summarise, PTSD occurs in response to a traumatic event. As the majority of SCI's in the UK are the result of a traumatic accident (Grundy & Swain, 2002) causing serious physical injury that impacts on functional ability there is the potential for PTSD to be a significant problem for this population (Blanchard et al., 1995;

Holbrook, Anderson, Sieber, Browner & Hoyt, 1999).

Although prevalence rates of the disorder differ between studies due to the different methodologies used it is clear that, although the diagnosis of PTSD is by no means uncommon, the occurrences of PTSD following a trauma would appear to be the exception rather than the rule.

Although the re-classification of PTSD in DSM-IV helped explain why not everyone experiencing the same trauma developed PTSD it still did not provide an explanation for why only a minority went on to develop the disorder. This led to a search for pre-disposing vulnerability factors for the development of PTSD. Those believed to be implicated in SCI include: trauma severity (Radnitz, Hsu, Tirch et al., 1998); interpersonal traumas (Radnitz, Hsu, Tirch et al., 1998); lack of social support (Danner & Radnitz, 2000; Nielsen, 2003a) and type of SCI (Radnitz, Hsu, Tirch et al., 1998).

Cognitive factors are believed to mediate and maintain PTSD (Brewin et al., 1996; Ehlers & Clark, 2000; Foa & Riggs, 1993). Cognitive models of PTSD that attempt to explain how information about the traumatic event is integrated into memory include the emotion processing model (Foa & Riggs, 1993; Foa & Rothbaum, 1998), the levels of representation model (Brewin et al., 1996) and the cognitive model of PTSD (Ehlers & Clark, 2000). Although all three models recognise how symptom reduction is aided by exposure to trauma memory and cognitive restructuring of negative appraisals each provides a different rationale as to how this is achieved based on each model's theoretical hypothesis. However, some of the assumptions

made by the models have been questioned (Brewin & Holmes, 2003; Dalgleish, 2004).

Conclusion

The aim of this literature review was to explore the psychological implications of spinal cord injury. The impact of living with this devastating injury clearly affects all areas of the individual's life and necessitates such a high degree of adjustment that the individual is at risk of further negative events, increasing their vulnerability to stress.

The early focus on stage models, which predicted a sequential process of recovery, dictated a passive role in intervention, with the expectation that time would eventually heal all. The more recent focus on depression as being maladaptive has provided greater insight into this condition from empirical studies.

Although depressive symptoms and major depressive disorder is highly prevalent among those with SCI (Elliott & Frank, 1996; Frank & Elliott, 1987) there is little evidence that detection or treatment of this condition has improved (Bombardier, Scott Richards, Krause, & Tulsky, 2004), even though it has been associated with medical and psychosocial complications (Elliott & Frank, 1996). More effective screening and diagnosis of this condition is needed, using clear and unambiguous definitions of depression and accurate measurements devised to take into account the anomaly that somatic symptoms of SCI are the same as those of depression and can therefore inflate depression scores.

A dynamic model to explain the adjustment process is needed. Future studies that disclose coping characteristics and strategies that aid adjustment to SCI will enable clinicians to detect those individuals who, without those characteristics or strategies, will struggle. As cognitive processes play such an important role in individuals choosing which coping strategy to use, research should focus on identifying how this is mediated.

That those with SCI are at risk of developing PTSD is clear from reported prevalence rates (Kennedy & Duff, 2001; Martz and Cook, 2001; Nielsen, 2003b; Radnitz et al., 1995). Research into PTSD related to SCI has been remarkably scant (Kennedy & Duff, 2001) particularly as prevalence rates are similar to rates of depression in SCI (Boekamp, Overholser, & Schubert, 1996; Howell et al., 1981). Therefore more needs to be done to identify those with SCI that experience this disorder.

Such research can offer potential clinical value by gaining a greater understanding of how vulnerability, mediating and maintaining factors impact on the development and maintenance of the disorder and so guide progress in developing treatment protocols. This can best be achieved by identifying those factors that predict PTSD.

By identifying which potential predictors of PTSD symptoms in this population are important, screening tools and treatment protocols can then be designed. SCI is potentially life threatening and affects every aspect of the individual and his or her family. As physical injury has been identified as a major risk factor for PTSD (Koren et al., 2005) and the chances of developing the disorder has been identified as eight times higher if the injury is a consequence of a traumatic event (Koren et al.,

2005) it is important that injury as a predictor of PTSD is investigated in this group.

It is known that social support can mediate symptoms of PTSD (Buckley, Blanchard, & Hickling, 1996; King, King, Fairbank, Keane, & Adams, 1998). Those with less access to a social support network after a trauma have been shown to manifest delayed onset PTSD (Buckley et al., 1996). Many with SCI report decreased social support from friends (Taricco et al., 1992). It is not known if this is due to how others view those with SCI or because those with SCI change in the way they relate to others. Does lack of social support predict whether this population experience traumatic symptoms or not?

A number of other questions remain unanswered such as whether certain personality traits or cognitive appraisals of the trauma, the self or the world predict whether PTSD develops. Do those living with the consequences of such a traumatic injury blame themselves and does this impact on whether they develop PTSD?

These questions all reflect how little is known about PTSD in this population. Attempts to address such questions will increase the knowledge base of PTSD related to SCI. Understanding fully the long term impact and process of adjustment to this disability will go a long way towards ensuring optimal rehabilitation and quality of life for all individuals living with SCI.

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Empirical Paper

Potential Predictors of Post Traumatic Stress Disorder Symptoms in Spinal Cord Injury

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The British Journal of Health Psychology has been used as a guide in determining the preparation of this paper (see Appendix 4 for notes to contributors).

Abstract

Objectives. Although spinal cord injury (SCI) is a severe, traumatic event little research on the role of PTSD subsequent to the injury has been published to date. This study has been conducted in order to investigate potential risk factors for the development of post traumatic stress disorder symptoms in those with spinal cord injury.

Design. This cross sectional study used multiple regression analysis to look for associations between PTSD symptom severity and potential risk factors such as age and gender.

Method. A total of 102 participants with SCI completed measures of neuroticism, alexithymia, acceptance of injury, social support, post traumatic cognitions and post traumatic stress.

Results. High levels of PTSD symptoms were found. Potential risk factors for the development of PTSD were negative cognitions of self and neuroticism. Variables that added to the variance explained by the models included alexithymia, age at injury, time since injury, lack of social support, and negative cognitions of the world.

Conclusions. The study highlights the need for services to be aware of the psychological difficulties experienced by this client group. It revealed important associations between independent variables and symptoms of PTSD that would benefit from further research and that have implications for treatment of PTSD symptoms in this population.

Introduction

In terms of physical and psychological impact Spinal cord injury (SCI) is a complex form of trauma that disrupts almost every system of the body, causing loss of motor, sensory and autonomic function (Grundy & Swain, 1996) leading to potentially life-threatening secondary medical problems (Brown, 1992).

Depending upon the site of the injury, SCI can result in paralysis from below the waist (paraplegia) or below the neck (tetraplegia). The degree of motor or sensory function maintained depends on whether the cord is completely or partially damaged.

An estimated 900 to 1,000 SCIs occur in the UK each year (Whalley Hammell, 1995) with a greater incidence among younger males (Partridge, 1994; Trieschmann, 1980). SCI results in a high–cost disability that has an enormous impact on life plans and expectations (Trieschmann, 1980). Advances in medical treatment and SCI management have resulted in significant numbers surviving this injury (Whalley Hammell, 1995) however psychological and social adjustment processes have not received as much attention as physical recovery (Frank, Van Valin, & Elliott, 1987).

The majority of SCI's in the UK are caused by life-threatening traumatic events, primarily road traffic accidents (RTAs) (Grundy & Swain, 1996). Victims of RTAs have been found to have experienced greater psychiatric morbidity (Malt, 1988; Mayou, Bryant, & Duthie, 1993), particularly post traumatic stress disorder (PTSD) (Blanchard et al., 1996; Green, McFarlane, Hunter, & Griggs, 1993; Mayou et al., 1993). PTSD is initiated by exposure to a traumatic event and characterised by

the persistent re-experiencing of the event, avoidance of traumatic and associated stimuli, and symptoms of hyperarousal (American Psychiatric Association, 1994).

The development of PTSD poses a significant problem for the SCI population in terms of rehabilitation, adjustment and long-term management of the injury (Williams, 1997) yet scant research attention has been given to PTSD following SCI (Kennedy & Duff, 2001).

Although high numbers of people have experienced a traumatic event only a minority go on to develop PTSD (Breslau & Kessler, 2001; Breslau, Kessler, Chilcoat, Schultz, & Davis, 1998). Of those exposed to extreme and prolonged trauma, known to be associated with high rates of PTSD (Goldstein, van Kammen, Shelly, Miller, & van Kammen, 1987; Kluznick, Speed, Van Valkenhurg, & Magraw, 1986; Yehuda et al., 1995) many do not develop it (McFarlane, 1990). A recent study concluded that the prevalence of PTSD after SCI, is similar to that found in other epidemiological research (Nielsen, 2003a) So few who experience a trauma go on to develop PTSD leading to the conclusion that, rather than being a normal response to an extreme environmental event, PTSD is a disorder resulting from a amalgamation of factors such as the experience of trauma combined with vulnerability factors (Yehuda & McFarlane, 1995).

The ability to identify risk factors that increase vulnerability to the development of PTSD is important for facilitating the prevention, referral and treatment of the disorder. Such factors can be classified into three distinct areas: pre-traumatic (predisposing vulnerability factors that exist prior to the trauma); peri-traumatic

(risk factors related to the experiences of the trauma itself, such as type and severity of trauma); and post-traumatic (factors that include ongoing life stresses and major life events associated with the long-term course of the trauma response).

Cognitive factors are also proposed to mediate and maintain PTSD, with the severity of PTSD symptoms being linked to dysfunctional cognitive appraisals about the self, the world and the nature of the traumatic memory (Brewin, Dalgleish, & Joseph, 1996; Foa & Riggs, 1993; Horowitz, 1997; Janoff-Bulman, 1985).

This exploratory study is interested in what makes some people with SCI more psychologically vulnerable to PTSD than others. Knowledge of such factors would help establish criteria to aid the identification of those at risk of developing PTSD and the development of treatment protocols, hopefully ensuring optimal rehabilitation and quality of life.

A number of predictors for PTSD in other trauma populations, such as cancer (Kangas, Henry & Bryant, 2005), are known but there may be others pertinent to this population. Those who sustain a SCI differ from many who experience a traumatic event because, as a consequence of their trauma, they are left with a life threatening and life-changing injury that impacts dramatically on their existence.

This study will look at some of the known risk factors of PTSD in other trauma populations to see whether they impact similarly on the SCI population. The risk factors that will be looked at are outlined below.

Neuroticism, or negative affect, has been found to be significantly correlated with symptoms of PTSD (Holeva & Tarrier, 2001; Joseph et al., 1996; Lauterbach & Vrana, 2001; McFarlane, Clayer, & Bookless, 1997; van den Hout & Engelhard, 2004) and has been found to be a better predictor of posttraumatic morbidity than exposure to the trauma (McFarlane, 1989).

Alexithymia is the inability to label and express emotions due to a lack of emotional awareness (Taylor, 1994). A relationship between PTSD and alexithymia has been suggested (Krystal, 1982; Shipko, Alvarez, & Noviello, 1983) with one study finding a significant association with alexithymia and the severity of PTSD symptoms (Yehuda et al., 1997) suggesting this may be a risk factor for the development of PTSD.

Social support has been known to mediate symptoms of PTSD (Buckley, Blanchard, & Hickling, 1996; King, King, Fairbank, Keane, & Adams, 1998) with lack of social support after a traumatic event associated with delayed onset PTSD (Buckley et al., 1996). This may be particularly problematic in the SCI population as many report decreased social support after sustaining their injury (Taricco et al., 1992). Social support is known to be important in aiding adjustment to SCI (Piazza et al., 1991; Trieschmann, 1988) with higher levels of social support associated with better health outcomes (Anson, Stanwyck, & Krause, 1993; Herrick, Elliott, & Crow, 1994) lower depression rates (Schulz & Decker, 1985) higher quality of life (Holicky & Charlifue, 1999) and greater life satisfaction (Schulz & Decker, 1985).

Physical injury is a major risk factor for PTSD, with the probability of developing

PTSD reported as eight times higher if the injury is a consequence of the traumatic event (Koren, Norman, Cohen, Berman, & Klein, 2005). Acceptance of injury has been inversely associated with psychological distress (Kennedy, Lowe, Grey, & Short, 1995; Wade et al., 2001) and used as an adaptive coping strategy in uncontrollable situations (Carver, Scheier, & Weintraub, 1989; Kennedy et al., 1995) such as SCI (Kennedy et al., 1995). Acceptance of injury and time since injury will be explored in this study to see if they have any effect on levels of PTSD symptomology. A recent study looking at psychosocial adaptation to SCI as a function of time since injury (Livneh & Martz, 2003) found reactions of psychological distress suggesting a non-acceptance of injury among those most recently injured (four years post SCI). The authors posited that the reactions of shock and anxiety experienced by those most recently injured may be indicative of intrusive, repetitive and unresolved issues similar to the trauma-related intrusive thoughts and physiological symptoms experienced by those with PTSD. It is certainly known that injuries sustained from RTAs tend to serve as unavoidable, frequent reminders that trigger symptoms of arousal (Blanchard & Hickling, 1997).

As cognitive factors are known to mediate and maintain symptoms of PTSD the existence of trauma related thoughts within the SCI population will also be investigated. The cognitive model of Ehlers and Clark (2000) proposes that those with PTSD process the traumatic event and/or its sequelae in a way that can lead to a persistent sense of current threat. How the trauma is appraised determines whether it is viewed as a time-limited event or one that poses a continuous threat. The perceived threat can be external ('Nowhere is safe') or internal ('It was my fault'). These negative cognitive interpretations are thought to maintain PTSD by

producing a sense of current threat that is accompanied by intrusions, arousal, and strong emotional responses. They also motivate the person to engage in dysfunctional behaviour and cognitive strategies such as thought suppression, rumination and safety seeking behaviour intended to control or reduce the perceived threat (Ehlers & Clark, 2000). These control strategies may exacerbate PTSD symptoms and maintain the problem by preventing change in the appraisal and trauma memory. It is possible that in some individuals an accident resulting in SCI might result in a variety of cognitive sequelae, including repetitive intrusive thoughts.

Aim: The aim of this exploratory study was to investigate potential risk factors for the development of symptoms of PTSD in adults who have experienced a SCI. Specifically the study asked what is the degree of association between the post traumatic stress symptoms of intrusion and avoidance and the character traits of alexithymia and neuroticism, injury related variables such as time since injury and individual differences in acceptance of injury, type of disability sustained and trauma related thoughts and demographic variables including social support, age, gender and education.

Method

Design

This cross-sectional study employed multiple regression analysis to establish the degree of association between potential risk factors for the development of symptoms of posttraumatic stress, such as age, social support and acceptance of illness, and the post traumatic stress symptoms of intrusion and avoidance. The dependent variables were therefore the posttraumatic stress symptoms reported by participants. The independent variables were: alexithymia; negative affect; perceived social support from both family and friends, acceptance of injury; post traumatic cognitions; age at injury; gender; type, level and cause of injury; length of time since injury; and education status. Non-parametric tests were employed to look at group differences between demographic variables.

Participants

Participants were recruited from Salisbury District Hospital and either seen as inpatients or outpatients, or sent postal questionnaires. All participants that met the inclusion criteria were identified and approached by staff at the spinal injuries treatment centre and invited to participate. Those who consented comprised a sample of self-selected participants.

Inclusion criteria for recruitment were that participants:

i) were aged between 18 years and 65 years of age;

ii) were English speaking, to maintain the validity of the questionnaires,

iii) had sustained a spinal cord injury through an abrupt traumatic event rather than a gradual process such as disease progression,

iv) had consented verbally, or in writing, to participate in the study,

v) were at least twelve weeks post-injury.[2]

Of 400 questionnaire packs posted to participants, 80 were returned completed. Of the 21 inpatients and 38 patients attending outpatient clinics eligible to participate only 22 did so.

102 participants completed the questionnaires (19 women, 83 men). Of these, 80 responded to posted study packs, 10 attended outpatient clinics and 12 were inpatients. A table of demographic information is shown overleaf (Table 1).

[2] During the initial twelve weeks of sustaining a spinal cord injury medical proceedings tend to be concentrated on stabilising the injury. This often results in patients being immobilised and sedated while medical and surgical treatment is performed. It was felt that any responses to questionnaires given by participants during this time could be influenced by such invasive procedures.

Table 1 Demographic features of the study population

		MALE (n=83)	FEMALE (n=19)	TOTAL (n=102)
AGE		45.76 yrs (Range 19.18-65.00)	45.18 yrs (Range 23.95-64.83)	45.66 yrs (Range 19.18-65.00)
MEAN AGE AT INJURY		29.64 yrs (Range 15.31-61.74)	33.66 yrs (Range 16.61-64.55)	30.39 yrs (Range 15.00-65.00)
MEAN TIME SINCE INJURY		16.12 yrs (SD 11.82)	11.51 yrs (SD 11.01)	15.26 yrs (SD 11.76)
TETRAPLEGIA	COMPLETE	21 (53.8%)	3 (42.9%)	24 (52.2%)
	INCOMPLETE	18 (46.2%)	4 (57.1%)	22 (47.8%)
	TOTAL	39	7	46
PARAPLEGIA	COMPLETE	28 (63.6%)	3 (25%)	31 (55.4%)
	INCOMPLETE	16 (36.4%)	9 (75%)	25 (44.6%)
	TOTAL	44	12	56
TRAUMATIC EVENT THAT CAUSED SCI	RTA*	40 (48.2%)	9 (47.4%)	49 (48.9%)
	SPORTS	24 (28.9%)	5 (26.3%)	29 (28.4%)
	INDUSTRIAL	9 (10.8%)	1 (5.3%)	10 (9.8%)
	DOMESTIC	7 (8.4%)	2 (10.5%)	9 (8.8%)
	MEDICAL	1 (1.2%)	2 (10.5%)	3 (2.7%)
	CRIME	2 (2.4%)	0	2 (2.9%)
	TOTAL	83	19	102

*RTA = Road Traffic Accident

Measures

Participants were asked to complete all questionnaires at just one time point and to provide demographic information on: age; gender; education; time since injury; disability (Appendix 5).

Dependent measure:

The Impact of Events Scale (IES) (Appendix 6)

The IES (Horowitz, Wilner, & Alvarez, 1979) was used to provide an overall measure of PTS symptom severity and measure the posttraumatic stress symptoms of intrusion and avoidance. The fifteen items in this self-report scale were derived from interviews with individuals who described the symptoms they experienced after a major life event. These descriptive statements were assigned to one of two subscales, intrusion and avoidance. The intrusion subscale comprises seven items and measures the frequency of intrusive memories of the traumatic event. The avoidance subscale comprises the remaining eight items and measures the extent to which the individual tries to avoid unpleasant memories, or reminders, of the event and feelings of distress. Combined, all 15 scores produce a Total Impact of Event score which provides a useful indication of the extent to which the traumatic event is still impacting on the individual.

Although not a diagnostic tool, the IES has been designed to measure current

symptom severity regardless of when the traumatic event happened. It asks participants to assess how frequently they experienced the symptoms in the scale during the past seven days. As the scale can be anchored to any life event participants in this study were prompted to complete it with reference to the event that caused their SCI by having "an accident resulting in an SCI" entered as the life event at the top of the scale. The IES has the advantage of being a brief measure, employed extensively for both assessment of, and research into, PTSD (Joseph, Yule, Williams, & Hodgkinson, 1993) and used to study PTSD in the SCI population (Radnitz et al., 1998).

Horowitz et al (1979) reported that the IES has good empirical validity, sensitivity and internal reliability. Cronbach's alpha scores demonstrate high internal consistency for both subscales (intrusion = 0.78; avoidance = 0.82) with a split half reliability for the total score of r = 0.86. Continuous scores are obtained for both subscales (maximum intrusion score = 35, maximum avoidance score = 40). Although there is not strict cut-off for the IES, Horowitz (1982) provides a classification system for assigning scores to correspond to one of three levels of distress (low $\leq$ 8, medium 9-19, high $\geq$ 20).

Independent measures:

The Positive and Negative Affect Schedule (PANAS) (Appendix 7)

The PANAS (Watson, Clark, & Tellegen, 1988) was developed as a brief measure

of positive and negative dimensions. The schedule is constructed of 10 adjectives describing negative moods and 10 adjectives describing positive moods. It was used to measure the trait dimensions of negative affect (neuroticism). Both subscales show satisfactory internal consistency (≥ 0.84) and test-retest reliability (0.68 = positive subscale; 0.71 negative subscale). Watson et al (1988) demonstrated validity in the independence of the two subscales (r = -0.09) and further confirmed this using factor analysis.

Acceptance of Injury (AIS) (Appendix 8)

The AIS (Felton, Revenson, & Hinrichsen, 1984) focuses directly on the degree respondents accept their illness without experiencing negative feelings or responses. This brief scale asks respondents to rate the extent to which they agree or disagree with statements about their illness. The composite responses provide a range of scores from 8 (extremely low acceptance/adjustment) to 40 (extremely high acceptance/adjustment). The scale has good psychometric properties with high internal consistency (Cronbach's alpha = 0.81 to 0.83) and reliability (Felton et al., 1984; Revenson & Felton, 1989). For the purposes of this study the scale was adapted to refer to 'injury' rather than 'illness' (Tegg, 1999).

Perceived Social Support Scale (PSS) (Appendices 9 & 9a)

The PSS (Procidano & Heller, 1983) is a forty item self-report questionnaire containing two subscales measuring perceived social support from friends (PSSfr)

and perceived social support from family (PSSfam). Scores for both subscales range from 0 (no perceived social support) to 20 (maximum perceived social support). The scale has good internal consistency with Cronbach's alpha of 0.88 and 0.92 for the friends and family subscales, respectively. The psychometric status of the scale was confirmed in a validation study by Lyons, Perotta, & Hancher-Kvam (1988) across chronic psychiatric, college and diabetic samples.

Toronto Alexithymia Scale (TAS) (Appendix 10)

The TAS (Bagby, Parker, & Taylor, 1994a; Bagby, Taylor, & Parker, 1994b) is divided into three subscales: difficulty identifying feelings (TASdif); difficulty describing feelings (TASddf) and external-concrete thinking (TASeot). Responses to all 20 items are scored on a 5-point Likert Scale. This measure has good internal consistency (Cronbach's alpha = 0.81), test-retest reliability (0.77, $p<0.01$), construct, convergent, discriminant and criterion validity (Bagby et al., 1994a; Bagby et al., 1994b; Taylor, 1994).

Posttraumatic Cognitions Inventory (PTCI) (Appendix 11)

The PTCI (Foa, Ehlers, Clark, Tolin, & Orsillo, 1999) is a thirty-seven item self-report questionnaire that measures trauma-related thoughts and beliefs. Scores from 33 of the items comprise three factors (negative thoughts about the self; negative thoughts about the world, and self-blame) which all have excellent internal consistency and good test-retest reliability (Beck et al., 2004; Foa et al., 1999). Tests of convergent validity between the PTCI and other scales measuring

dysfunctional self and world related thoughts and beliefs have shown the questionnaire to correlate moderately to strongly with those measures (Foa et al., 1999; Emmerik, Schoorl, Emmelkamp & Kamphuis, 2006). It also discriminates well between traumatised individuals with and without PTSD (Foa et al., 1999). Although there is a high correlation between the PTCI and PTSD severity (for example as measured by the PDS) they have different underlying constructs (Foa et al, 1999).

Procedure

The study was granted ethical approval by the Department of Psychology, Southampton University, and the Research Ethics Committee, Salisbury Health Care NHS Trust (Appendices 12 & 13). Participants were recruited from the Duke of Cornwall Spinal Treatment Centre, Salisbury District Hospital with the permission of the medical consultants involved (Appendix 14). All participants gave written, informed consent prior to participation.

A condition of Research Ethics Committee approval required that patient confidentiality was maintained within the unit, without the researcher having access to patient information, and that patients who did not respond, or declined to participate at initial point of contact, were not contacted again. The unit consultants agreed that the database manager would select all potential participants from the database, based on the specified inclusion criteria, while secretaries and other unit staff would distribute the information to them. The researcher provided the

secretaries with the study paperwork to be distributed and her contact details.

Patients resident on the ward were given an envelope by their consultant, or a member of the ward staff, containing an introductory letter from the consultant (Appendix 15), an invitation letter (Appendix 16) asking if they would like to be contacted by the researcher to have any questions they had answered and to discuss whether they would be willing to take part in the study, an information sheet (Appendix 17), and a reply slip. This allowed patients the time to reflect on whether they wished to participate or to gain more information about the study.

Those patients who requested a visit were seen by the researcher in a quiet area of the spinal unit to ensure privacy. They were given the opportunity to ask questions about the study and then, having agreed to participate, were asked to sign a consent form (Appendix 18), provide demographic information (Appendix 5) and to complete the six questionnaires (Appendices 6 to 11).

Patients attending outpatient clinics had a letter from their consultant introducing the study, an invitation to participate letter (Appendix 16b), the study information sheet, a reply slip and a pre-paid envelope all enclosed with their next outpatient appointment date. Upon receipt of the reply slip agreeing to participate in the study the researcher phoned participants to arrange a convenient time for them to be seen, either before or after their outpatient appointment. They were given the opportunity to ask questions about the study both during this phone call and at the meeting. The meeting took place in a quiet area of the centre, where patients could not be overheard. The participants were informed that any carer attending the interview

with them could be present during this meeting, should the participant wish them to be. The researcher offered to complete the questionnaires for participants with tetraplegia, if they needed this assistance. The majority of participants attending outpatient clinics preferred to take the questionnaire packs home with them to complete in their own time and posted them back to the researcher in the pre-paid envelope provided.

For participants sent questionnaires by post the researcher provided study packs in unsealed, stamped envelopes to the Consultant's secretaries. The secretaries then included the introductory letter from the consultant, addressed the envelope to the participant and posted the pack out. Packs contained an invitation letter from the researcher (Appendix 16c) providing a date by which the pack should be posted back, the study information sheet, consent form, demographic information form, questionnaires and a reply paid envelope. For those participants who might have difficulty completing the questionnaires by hand, particularly those with tetraplegia, a disk was enclosed containing the demographic information sheet and questionnaires. Participants were also provided with a web address where they could provide this information online. Consent forms were designed so that they could be signed by participants or a representative.

All potential participants were offered the opportunity to receive a copy of the summary findings of the research, whether they consented to participate or not.

Results

Statistical analysis

Analysis was conducted using the Statistical Package for Social Sciences (SPSS, 2003) for Windows, (version 12). As there were 10 independent variables, 100 participants were considered adequate for using linear regression analysis (Whitaker, C., personal communication, May 2004; Tabachnick & Fidell, 2001).

As this is an exploratory analysis backwards, forwards and stepwise regressions were run to examine the models derived by all three methods. The different regression methods all led to the same model. Such consistency in model choice provides potential evidence that the data presents good evidence for the model generated. Only stepwise regressions are presented here as they include variables significant at the 5% level thus tending to exclude those variables involved in suppressor effects. This increases the risk of making a Type II error (eliminating a variable that predicts the outcome) but leads to a more powerful model.

In contrast, the backward model includes variables significant at the 10% level and so is more likely to include variables that are not associated with the outcome (thus increasing the risk of making a Type I error; wrongly rejecting a true null hypothesis).

The regressions were examined for potential problems. Analysis of the residuals revealed no departures from the assumptions of normality or equal variability

(Howell, 2007). To ensure that there were no problems with multicollinearity the variance inflation factor (VIF), which measures the impact of collinearity among the variables in a regression model, was examined. Typically a VIF value greater than 10 is of concern (Marquandt, 1980). The majority of VIFs in this study were less than 2 (with only one being greater than 2 but less than 3), suggesting no multicollinearity problems.

However, when the regression model was run it was noted that, although the relationship between the dependent measure of intrusion and the 'difficulty describing feelings' factor of the Toronto Alexithymia Scale (TASddf) (0.29) was positive when looked at in isolation, TASddf changed to a negative partial correlation (-.26), contrary to expectation, when other variables were included in the regression model.

When the regressions were run with TASddf and the other two dependent variables (avoidance and total IES score) again negative relationships were found. Partial plots did not reveal any obvious outliers that could have explained this. In summary the TASddf variable correlated positively with intrusion when considered in isolation but negatively when other variables were taken into account which may be indicative of multicollinearity problems (Dr Whitaker, personal communication Sept 2007). For this reason the TASddf variable was excluded from the analysis.

As the data was not normally distributed non parametric correlations were used to identify variables that appeared to be correlated with the response variable. These were entered in the regression at step one, with the remaining variables entered on

succeeding steps.

Regression analyses were performed on the total IES scale scores and also on the two subscales of Avoidance and Intrusion. The construct 'negative affect' subsumes a broad range of aversive mood states, including distress and depression (Watson & Pennebaker, 1989). As it was considered possible that scores on the PANAS scale could reflect low mood, those regression analyses that included negative affect in the model were performed again with negative affect removed (Bennett & Brooke, 1999).

Group differences between demographic variables were explored using Mann Whitney tests on the categorical variables of gender, type and level of injury and Kruskal Wallis tests on the ordinal variables of levels of education, causes of injury and professional status.

Descriptive statistics

Means and standard deviations of the dependent and independent measures are shown in Appendix 19. Both dependent and independent variables were examined for differences between gender, level and type of injury. Results are discussed below.

Dependent variables

Initially the IES scores were categorised into low (≤8), medium (9-19) and high (≥20) levels of distress (Horowitz, 1982). This revealed that of the 102 participants 39 (38.2%) reported medium levels of distress while 63 (61.8%) of participants reported experiencing high levels of distress.

Gender

The Mann-Whitney U test found that women had higher scores on the total IES score than men ($U = 499$, $n_1 = 83$, $n_2 = 19$, $Z = -2.50$, $p<.05$) and reported significantly more intrusions than men ($U = 474$, $n_1 = 83$, $n_2 = 19$, $Z = -2.75$, $p<.01$). There was no difference between gender on the avoidance subscale.

Level of injury

Results showed that total IES scores ($U = 986$, $n_1 = 55$, $n_2 = 47$, $Z = -2.07$, $p<.05$) and intrusion subscale scores ($U = 999$, $n_1 = 55$, $n_2 = 47$, $Z = -2.01$, $p<.05$) were significantly higher for partial compared to complete level of injury. There was no difference between levels of injury on the avoidance subscale.

Type of injury

No differences were found between types of injury.

Independent Variables

Gender differences

Scores on the independent measures were also examined for differences between gender. The Mann-Whitney U test found that more men had higher scores on the acceptance of injury scale (AIS) ($U = 393$, $n_1 = 83$, $n_2 = 19$, $Z = -3.40$, $p<.05$), that women had greater difficulty identifying feelings (TASdif) ($U = 528.5$, $n_1 = 83$, $n_2 = 19$, $Z = -2.23$, $p<.05$) had more negative cognitions of self (PTCIself) ($U = 532$, $n_1 = 83$, $n_2 = 19$, $Z = -2.21$, $p<.05$) and reported greater negative affect (NA) ($U= 485.5$, $n_1 = 83$, $n_2 = 19$, $Z = -2.61$, $p<.05$).

Spearman's correlations, looking at female scores for the independent variables, revealed that PTCIself was significantly correlated with TASdif ($r_s=.672$, $df = 17$, $p<.01$), AIS ($r_s=-.507$, $df = 17$, $p<.05$) and NA ($r_s = .549$, $df = 17$, $p<.05$).

AIS and NA were also significantly negatively correlated ($r_s = -.736$, $df = 17$, $p<.01$) with higher scores on NA associated with lower scores on AIS, a finding that was expected.

It is possible that the correlations for females between AIS and PTCIself and TASdif and PTCIself may have been influenced by NA. Partial correlations were therefore performed to examine this.

The Spearman's correlation between PTCIself and AIS was $r_s= .507$ ($df= 17$,

$p<.05$). However, the correlation between PTCIself and AIS controlling for negative affect declined to $r_s = .065$, which was no longer significant ($df = 14, p=.799$). In other words there was no significant relationship between PTCIself and AIS indicating that negative affect was influencing the initial results.

The correlation between PTCIself and TASdif was $r_s = .672$ ($df = 17, p<.01$). When negative affect was controlled for the correlation between PTCIself and TASdif was $r_s = .655$, which remained significant ($df = 14, p<.01$). In other words negative affect was not influencing the strong correlation between PTCIself and TASdif.

Gender and injury type and level

Chi^2 tests showed no difference between gender in the type of injury (paraplegia or tetraplegia) they sustained (two-tailed Fisher exact p.091) but significantly more males had a complete level of SCI than females (χ^2 (2, N = 102) = 4.69, $p<.05$).

Demographic variables and injury type and level

Kruskal-Wallis tests looked at differences within levels of education, causes of injury and professional status. No significant differences were found.

Regression Analysis.

These were computed separately, initially for the total IES score and subsequently for the IES subscales of avoidance and intrusion.

Impact of Event Scale (Total Score).

The significant correlation coefficients between IES total score and the independent variables were considered important variables to look at and therefore gave a starting point for the exploratory regressions (see Appendix 20).

The stepwise regression revealed that PTCIself explained 44% of the variance in symptoms of PTSD ($F_{1,100} = 78.24$, $p<.001$) while NA explained a further 6% ($F_{1,99} = 12.9$, $p<.001$). Greater symptoms of PTSD were associated with greater levels of post trauma cognitions of self and negative affect (see Appendix 21).

A hierarchical regression was then performed, entering the variables from the model above, followed by the non-significant variables from the Spearman's correlation to explore whether they might explain any unexplained variability within the model. Social support from family (FamSppt) explained a further 5% of the variance ($F_{1,98} = 10.42$, $p<.01$) and age at injury accounted for a final 2% ($F_{1,97} = 5.45$, $p<.05$) (see Appendix 22).

A General Linear Model of IES total on the factors gender and level of injury and the covariates PTCIself, FamSppt, TASdif, NA and age at injury was then performed. This showed that neither gender ($F_{1,95} = .001$, $p.972$) nor level of injury ($F_{1,95} = .856$, $p.357$) had a significant effect when added to a model already containing the covariates.

Total scores analysed without negative affect

To ensure the IES was not simply measuring low mood and distress the analysis was performed again, without negative affect.

The stepwise regression revealed that 44% of the variance in symptoms of PTSD was explained by PTCIself ($F_{1,100} = 78.24$, $p<.001$) with TASdif explaining a further 4% ($F_{1,99} = 7.04$, $p<.001$). Greater symptoms of PTSD were associated with negative cognitions of self and difficulty identifying feelings (see Appendix 23).

Adding the non-significant variables from the Spearman's correlation revealed that social support from the family explained a further 4% of the variance ($F_{1,98} = 9.27$, $p<.01$) and age at injury a final 2% ($F_{1,97} = 4.13$, $p<.05$) (see Appendix 24).

A General Linear Model of IES total on the factors gender and level of injury and the covariates PTCIself, TASdif, time since injury and FamSppt was performed. This showed that neither gender ($F_{1,95} = .092$, $p.763$) nor level of injury ($F_{1,95} = .110$, $p.741$) had a significant effect when added to a model already containing the covariates.

Avoidance subscale

The significant correlation coefficients between IES Avoidance score and the potential risk factors gave a starting point for the exploratory regressions (see Appendix 20).

In the stepwise regression NA explained 43% of the variance in symptoms of PTSD ($F_{1,100} = 76.57, p<.001$) and PTCIself explained a further 8% of the variance ($F_{1,99} = 14.31, p<.001$) showing that greater symptoms of avoidance were associated with greater levels of both negative affect and post trauma cognitions of self (see Appendix 25).

A hierarchical regression was then performed, entering in the variables from the stepwise model above followed by the non-significant variables from the Spearman's correlation.

The initial variables of NA and PTCIself explained a significant 51% of the variance in symptoms of avoidance ($F_{2.99} = 50.54, p<.001$). When FamSppt score was entered a further 3% of the variance was explained ($F_{1,98} = 6.01, p<.05$) (see Appendix 26).

Avoidance scores analysed without negative affect

The analysis was then run again, without negative affect. In the stepwise regression PTCIself explained 40% of the variance in symptoms of PTSD ($F_{1,100} = 65.86, p<.001$) and PTCIworld explained a further 2% of the variance ($F_{1,99} = 4.11, p<.05$) showing that greater symptoms of avoidance were associated with greater levels of both post trauma cognitions of self and of the world (see Appendix 27).

A hierarchical regression was then performed, first entering the variables from the stepwise model above then entering the non-significant variables from the Spearman's correlation to see if these accounted for any further, unexplained

variability.

The initial variables of PTCIself and PTCIworld explained a significant 42% of the variance in symptoms of avoidance ($F_{2,99} = 36.01, p<.001$). The addition of FamSppt explained a further 4% of the variance ($F_{1,98} = 7.06, p <.01$) (see Appendix 28).

Intrusion subscale

The significant correlation coefficients between IES Intrusion score and the independent variables gave a starting point for the exploratory regressions (see Appendix 20).

In the stepwise regression PTCIself explained 37% of the variance in symptoms of intrusion ($F_{1,100} = 58.64, p<.001$), TASdif explained a further 5% of the variance ($F_{1,99} = 8.49, p<.005$) and time since injury explained a final 3% of the variance ($F_{1,98} = 4.62, p<.05$) showing that greater symptoms of intrusion were associated with post trauma cognitions of self, difficulty identifying feelings and with longer time since injury (see Appendix 29).

A hierarchical regression was then performed, first entering the variables from the stepwise model above, followed by the non-significant variables from the Spearman's correlation.

The initial variables of PTCIself, TASdif and time since injury explained a

significant 45% percent of the variance in symptoms of intrusion ($F_{3,98} = 26.26$, $p<.001$). When FamSppt was entered a further 3% of the variance was explained ($F_{1,97} = 7.16$, $p<.01$) (see Appendix 30).

A General Linear Model of IES intrusion on the factors gender and level of injury and the covariates PTCIself, TASdif, timesince and FamSppt was performed. This showed that neither gender ($F_{1,96} = 0.11$, $p.29$) nor level of injury ($F_{1,96} = 0.21$, $p.65$) had a significant effect when added to a model already containing the covariates.

Discussion

The primary aim of this research was to identify potential risk factors of symptoms of PTSD in people with SCI.

This study found negative cognitions of self and neuroticism to be strongly associated with greater symptoms of PTSD following a SCI. Other variables that contributed to the amount of variance explained by the regression models included: difficulty identifying feelings, time since injury, negative cognitions of the world, social support from family, and age at injury.

Characteristics of the participants

Medium levels of distress were reported by 38.2% of participants, while an astonishing 61.8% scored in the high range indicating severe symptomatology as

measured by the IES distress scoring system (Horowitz, 1982). Not one participant reported low distress. However, this may reflect that it was primarily those with greater levels of distress who participated.

Without establishing a diagnosis of PTSD according to DSM-IV criteria it is not known how many participants in this study fulfilled the criteria for this diagnosis (the version of the IES utilised in this study did not assess hyperarousal symptoms). It is known that many traumatised people can experience high levels of distress but only fulfil the criteria for partial or subsyndromal PTSD (Blanchard, Hickling, Vollmer et al., 1995; Carlier & Gersons, 1995; Schützwohl & Maercher, 1999), highlighting how an underestimation of the need for services can be caused by concentrating only on cases that meet the full diagnostic criteria for PTSD (Carlier & Gersons, 1995).

Women had significantly higher scores on the total IES scale and the intrusion subscale, reporting greater levels of distress and significantly more intrusions than men. This finding was not unexpected as higher rates of PTSD have been found in females compared to males (Brewin, Andrews, & Valentine, 2000; Bryant & Harvey, 2003; Holbrook, Hoyt, Stein, & Sieber, 2002; Van Loey & Van Son, 2003). This gender difference has been explained in one study by type of traumatic event experienced, with females reporting more acts of interpersonal violence (Breslau, Davis, Andreski, Peterson, & Schulz, 1997). In this study all causes of SCI for women were accidental. It is possible that the women in this study had experienced prior trauma, which may have influenced the findings, but this was not investigated. The differences in scores on independent measures between gender revealed women to have fared less well than men, having greater difficulty identifying feelings and

more negative cognitions of self. Women also reported greater neuroticism than men and were less accepting of their injury. It is not known if this was a pre-existing personality trait or if the women developed neuroticism as a result of their traumatic experience.

Looking at the findings for the female participants it appeared that having negative cognitions of self was significantly associated with difficulty identifying feelings, acceptance of injury and neuroticism. It was felt that neuroticism may have been influencing these relationships and closer examination, using partial correlations, confirmed this assumption. Higher neuroticism in females may have accounted for the association between negative cognitions of self and acceptance of injury but not for negative cognitions of self and difficulty identifying feelings, where a significant association remained after controlling for neuroticism.

The association found for female participants between negative cognitions of self and difficulty identifying feelings may have been due to suppression. Suppressing negative feelings is a key avoidance symptom of PTSD and thus the results are in accordance with recent cognitive models (Ehlers & Clark, 2000). Perhaps the only way the women in the study could cope with their injury, or the consequences of their injury, was to deny their feelings about it, leading to increased negative cognitions of self. If so, this finding would have more to do with acceptance of SCI than PTSD. Although there is an association between these two variables, causality cannot be established. Do negative cognitions of self lead to difficulty identifying feelings or vice versa?

Acceptance of injury and neuroticism were also significantly negatively correlated. Extent of injury has been found to interact with personality factors in predicting psychological health in the months following accidents (Frommberger et al., 1999).

Looking at injury differences revealed that the total IES and intrusion subscale scores were significantly greater for those with a partial level of injury compared to those with a complete level of injury. This is the converse of a study that found individuals with a complete SCI to have a higher risk of developing PTSD (Nielsen, 2003b). Severity of physical injury has been found to be one of the strongest and most reliable predictors of PTSD in victims of accidents (Blanchard, Hickling, Mitnick et al., 1995; March, 1993). It is possible that it was not the extent of the injury that led to inflated distress and intrusion scores but some other factor. Blanchard and Hickling (1997) noted that injuries sustained from motor vehicle accidents, which was the biggest cause of SCI in the present study, tended to serve as unavoidable, frequent reminders that triggered symptoms of arousal in those with PTSD. As the intrusion subscale scores were increased in those with a partial level of SCI, it is possible that this level of SCI injury was causing greater intrusive thoughts. Those with paraplegia experience greater symptoms of PTSD, hypothesized as their experiencing greater pyschophysiological arousal, than those with tetraplegia (Radnitz et al., 1998). It is possible that a similar mechanism is at work with those with a partial level of injury. It is also possible that this was due to expectation. If

an individual is getting some biological feedback from their site of injury they may expect their injury to improve.[3]

Regressions

Multiple regression analyses demonstrated that the greatest amount of variance in PTSD scores for both the total IES scores and the intrusion subscale scores was explained by negative cognitions of self.

For the avoidance subscale the greatest amount of variance in PTSD scores was explained by neuroticism. However, when neuroticism was removed from the analysis negative cognitions of self again explained the greatest amount of variance.

Main associations

Negative cognitions of self

That negative cognitions of self accounted for the greatest amount of variance in all outcome measures, except the avoidance subscale, supports research that states that dysfunctional cognitive appraisals about self are involved in mediating and maintaining PTSD (Brewin et al., 1996; Foa & Riggs, 1993; Horowitz, 1997; Janoff-Bulman, 1985). A SCI is a devastating injury that affects all areas of the individual's

[3] This constant reminder of what they have lost, and how the injury was sustained, may be the cause of their intrusions. However, this interpretation is purely speculative and requires further investigation.

life. This in itself may well influence the type of negative cognitions held. A recent paper, purporting to be the first to look at negative cognitive appraisals in PTSD symptoms in those with SCI (Agar, Kennedy, & King, 2006), also found negative cognitions of self to be a main predictor of PTSD symptoms in this population.

A key theme identified in such maladaptive appraisals is of current threat, even when the traumatic event is in the past (Ehlers & Clark, 2000). Those holding negative cognitions of self experience the traumatic event as continuing to have damaging implications, precipitating anxiety. Threatening beliefs (I am weak; I cannot cope) may cause individuals to doubt their own ability, threatening their view of themselves (Dunmore, Clark, & Ehlers, 1999). In an attempt to control this sense of ongoing threat individuals may engage in strategies such as suppressing intrusive thoughts. Unfortunately the use of such strategies can paradoxically increase the frequency of intrusions, which consequently amplifies their PTSD symptoms (Davis & Clark, 1998).

Neuroticism

Significant amounts of variance in the avoidance subscale scores were explained by neuroticism. This supports the finding of other studies that have found neuroticism to be significantly associated with symptoms of PTSD (Holeva & Tarrier, 2001; Joseph et al., 1996; Lauterbach & Vrana, 2001; McFarlane et al., 1997; van den Hout & Engelhard, 2004) and a better predictor of posttraumatic morbidity than exposure to the trauma (McFarlane, 1989).

A prospective study of personality traits and exposure to trauma in Dutch

peacekeepers (Bramsen, Dirkzwager, & van der Ploeg, 2000) found the individuals with the most severe symptoms of PTSD were those exposed to the highest number of stressful events. However, a high level of PTSD symptoms was also found in those peacekeepers who had rated highest on negative personality traits, suggesting that the chances of developing PTSD can depend as much on an individual's personality as on the traumatic event experienced.

Neuroticism has been identified as a risk factor for the development of anxiety (Andrews, Stewart, Morris-Yates, Holt, & Henderson, 1990; Clark, Watson, & Mineka, 1994), by increasing sensitivity to negative stimuli (Goldberg, 1992). This may explain why neuroticism is so strongly associated with avoidance subscale scores in this study. Individuals with a traumatic SCI have not only experienced a traumatic event but continue to experience ongoing difficulties due to their injury. If neuroticism increases sensitivity to negative stimuli, leading to the development of anxiety, then the significant association between high levels of neuroticism and symptoms of avoidance found here should not be surprising. This potentially life threatening injury affects every aspect of the individual's life necessitating such a high degree of adjustment that the individual is at risk of further negative events.

That negative cognitions of self and neuroticism both explained significant amounts of variance in the PTSD symptom scores supports the hypothesis that specific individual differences can compliment broad personality domains (Paunonen, 1998). The findings presented here also support Cox, MacPherson, Enns and Williams (2004) study, which found that the broad personality trait of neuroticism and the more specific individual difference trait of self-criticism were both significantly

associated with PTSD, suggesting that both may represent robust psychological dimensions associated with the presence of PTSD. However an alternative explanation is that both neuroticism and negative cognitions of self are simply consequences of elevated symptoms of PTSD.

There was always the concern in this study that the PANAS scale might be tapping into constructs of depression, rather than neuroticism (Watson & Penebaker, 1989), raising the question of whether the study findings were reflecting the difficulty of depressed patients in adjusting to their SCI (Frank & Elliott, 1987), rather than of neurotic patients experiencing symptoms of PTSD.

That the combined effect of other variables with those of neuroticism and negative cognitions of self increased the amount of variance explained by the regression models by as much as 11% may be understood in terms of a model of interactions (Agar et al., 2006, Brewin et al., 2000). Brewin et al., (2000) asserted that the data they analysed in their meta-analysis may be 'regarded as consistent with a model in which the impact of pretrauma factors on later PTSD is mediated by responses to the trauma or, alternatively, with a model in which pretrauma factors interact with trauma severity or trauma responses to increase the risk of PTSD' (p756). Those variables that added to the amount of variance explained are discussed below:

Alexithymia

A significant association has been found between alexithymia and the severity of PTSD symptoms (Yehuda et al., 1997) suggesting this personality trait may be a

risk factor for the development of PTSD. Difficulty identifying feelings accounted for some variance in the total IES scale (when neuroticism was excluded from the regression equation) and in the intrusion subscale. This inability to process emotions has been thought to disrupt the integration of memories and their emotional associations into more general memory systems (Brewin et al., 1996).

That women in the study had greater difficulty identifying their feelings was unexpected as research has shown that males tend to have higher levels of alexithymia (Bagby et al., 1994a; Carpenter & Addis, 2000; Honkalampi et al., 2004) although other studies have found no gender differences (Parker, Taylor, & Bagby, 1989). It is possible that the women in this study were at higher risk for psychopathology because they had difficulty identifying their feelings, which may have inflated the scores. This issue also raises a question regarding gender specific risk factors which could not be explored in this study.

However, patients with higher psychological acceptance of SCI have been found to have greater alexithymia, a finding hypothesised as being due to patients suppressing or denying their feelings and thus developing alexithymic features (Fukunishi, Koyama, & Tobimatsu, 1995), rather than it being an innate personality characteristic influencing the onset of PTSD symptoms.

Age at injury

Age at injury also explained a small amount of variance when neuroticism was removed from the total IES score, with those of younger age experiencing greater

symptoms of PTSD, a finding supported by other studies (Finnsdottir & Elklit, 2002; Neria, Bromet, Sievers, Lavelle, & Fochtmann, 2002).

It is possible that both the above pretrauma factors were interacting with the trauma response to increase the risk of PTSD (Brewin et al., 2000; Joseph, Yule & Williams, 1993).

Time since injury

Time since injury explained some of the variance in the intrusion subscale revealing that the shorter the time since injury the stronger the association with intrusions. This concurs with a recent study looking at psychosocial adaptation to SCI (Livneh & Martz, 2003) which found that participants who were more recently injured displayed more shock, denial and generalised anger resulting in a non-acceptance of SCI.

The authors further speculated that the symptoms of shock, experienced by the more recently injured, and the symptoms of anxiety, which were higher for those who had been injured longest, may reflect intrusive, repetitive and unresolved issues akin to those experienced by PTSD sufferers (Livneh & Martz, 2003), reflecting an impairment in the cognitive and emotional processing of the trauma (Foa, Steketee & Rothbaum, 1989).

Social support

Lack of perceived family support consistently explained small, but important, amounts of variance in the total IES scores and in the intrusion and avoidance subscales. A recent study found receiving social support positively effected coping in those with SCI (McColl, Lei, & Skinner, 1995).

Perceived lack of support from friends was not associated with PTSD symptoms at all which is interesting as social support from friends has been found to be important for those with SCI (Heinemann, 1995) although those with SCI have reported a substantial decrease in social interaction with friends after they sustained their injury (Taricco et al., 1992).

Negative cognitions of the world

When neuroticism was removed from the regression equation on the avoidance subscale negative cognitions of the world explained some of the variance. It is known that dysfunctional cognitive appraisals about the world are involved in mediating and maintaining PTSD (Brewin et al., 1996; Foa & Riggs, 1993; Horowitz, 1997; Janoff-Bulman, 1985). Those that feel the world is now a more frightening, unpredictable place may respond by engaging in avoidance and safety behaviour in an attempt to cope with the feelings of ongoing threat produced by such dysfunctional appraisals (Dunmore et al., 1999; Dunmore, Clark, & Ehlers, 2001; Ehlers & Clark, 2000). As a result, such behaviour sustains this sense of threat as the dysfunctional appraisals are never disconfirmed (Dunmore et al., 1999, 2001;

Ehlers & Clark, 2000). Unsympathetic responses from others confirm the view that the world is a dangerous place (Foa & Riggs, 1993; Foa & Rothbaum, 1998). This may be particularly difficult for those with a SCI given the fact that, due to their injury, the world may now appear more dangerous. If the reported decrease in social interaction with friends after SCI (Taricco et al., 1992) is due to how others view them, rather than the individual with a SCI changing how they relate to others, this could be seen as further evidence of how dangerous the world has become.

Clinical implications

The development of PTSD is a significant problem for the SCI population in terms of rehabilitation, adjustment and long-term management of the injury (Williams, 1997). That all the participants in this study reported medium to high levels of distress highlights the need for services to be aware of the psychological difficulties experienced by this client group and to respond by ensuring rehabilitation staff assess not only the physical effects of the SCI but also the emotional effects (Williams, 1997).

That women experienced greater difficulty with acceptance of injury, negative cognitions of self and difficulty identifying their feelings reveals their greater vulnerability to developing PTSD, highlighting the need for staff to monitor female patients closely. There were a comparably low number of females in the study, which may be considered problematic in regard to generalising the findings; however this disparity is representative of the SCI population (Partridge, 1994; Trieschmann, 1988; Whalley Hammell, 1995).

The highly significant association between participant's negative cognitions of self and PTSD symptoms supports the empirical literature (Dunmore et al., 1999; Ehlers & Clark, 2000; Foa & Riggs, 1993). Cognitive factors are known to mediate and maintain PTSD, with the severity of PTSD symptoms linked to dysfunctional cognitive appraisals about the self, world and the nature of the traumatic memory (Brewin et al, 1996; Foa & Riggs, 199). Such dysfunctional appraisals produce a sense of current threat, eliciting dysfunctional behaviour and cognitions in an attempt to control or reduce this perceived threat (Ehlers & Clark, 2000). Such control strategies may exacerbate PTSD symptoms and maintain the problem by preventing change in the appraisal and trauma memory. This finding has implications for early screening in an attempt to thwart the onset of chronic PTSD and for treatment, particularly the use of cognitive behavioural therapy to modify negative appraisals (Ehlers & Clark, 2000).

Two personality factors, neuroticism and alexithymia, were both strongly associated with PTS symptoms, demonstrating their importance. Those whose personalities tend to be more negative are more likely to elicit negative cognitive appraisals of self, world and trauma memory while those with alexithymia may struggle with emotional processing of the trauma. This confirms the findings of previous research of both neuroticism (Holeva & Tarrier, 2001; Joseph et al., 1996; Lauterbach & Vrana, 2001) and alexithymia (Yehuda et al, 1997) as being risk factors for the development of PTSD. Although the measurement of personality traits post-trauma may detect changes in personality that have occurred as a consequence of the experience, it may also reflect enduring personality traits, particularly as there is substantial evidence for the long-term stability of such traits (Watson & Walker,

1996). Identification of those with these personality traits may lead to early detection and treatment of PTSD.

Limitations

This study had some methodological drawbacks that must be borne in mind when interpreting the results and considering future research. This study was cross-sectional and therefore findings can only provide circumstantial evidence for the identification of risk factors of PTSD. The cross-sectional design precludes any conclusions about causation.

It is also important to note that some of the variables identified as strongly association with PTSD symptoms could also be regarded as correlates or even consequences of PTSD. Only a few of the variables, such as age at injury and gender, could be established with certainty prior to the condition and therefore their measurement was not potentially influenced by PTSD symptoms.

The TAS 'difficulty describing feelings' factor was behaving so contrary to expectation in the initial analysis it was removed. It would have been interesting to have performed a factor analysis on the TAS scale to see if the factors were loading in a similar way to the validation study results. However, this was beyond the scope of this study.

Had the IES (Revised) (Weiss & Marmar, 1997) been used to establish PTSD symptoms of hyperarousal, a prevalence rate of PTSD within the study sample

could have been established. It would also have been interesting to explore the differences between coping with life since SCI and acceptance of SCI.

Although using self-report measures is a common occurrence in this type of research, the fact that they were used for both dependent and independent measures does introduce the possibility of reporting bias, such as a tendency to give mainly negative responses. This may be more of a problem when taken in conjunction with the self selecting participants, as described above. In-depth interviews may have been a more reliable method of gaining information.

Few participants attending the outpatients' clinic responded, possibly because the investigations they undergo at the clinic are quite intense and lengthy. The added time needed to meet with the researcher and complete questionnaires may have influenced response rates. There was also a poor response rate for postal questionnaires, even though physical difficulty in completing questionnaires had been considered and alternatives methods devised.

The participants were self-selecting, leading to potential bias. It is possible those experiencing high levels of PTSD did not participate, (perhaps due to avoidance symptoms). Therefore the numbers experiencing symptoms of PTSD may actually be much higher. Alternatively, the participants may have been those that were especially treatment seeking given the high indicators of distress in this group.

Although using self-report measures is a common occurrence the fact that they were used for both the independent and dependent measures does introduce the

possibility of reporting bias, such as a tendency to give mainly negative responses. This may be more of a problem when taken in conjunction with the self selecting participants, as described above. In depth interviews may have been a more reliable method of gaining information.

Suggestions for future research

Further research, employing prospective, longitudinal designs, is required to demonstrate causal links between variables. Future studies should include both a measure of PTSD, to establish a prevalence rate, and potential predictor variables that are less likely to be responses to the SCI, such as previous exposure to trauma. It would also be interesting to explore differences in level of SCI more closely to determine whether those who still experience biological feedback from their site of injury are more likely to experience intrusions and why this should be.

It was unknown whether the PTSD symptoms in this study were due to the traumatic event that caused the SCI or the trauma of living with a SCI. Future research should attempt to differentiate between the two as this may lead to a better understanding of the interactions and mediating effects of individual difference, injury and pretrauma variables on PTSD symptoms.

The construct 'negative affect' subsumes a broad range of aversive mood states, including distress and depression (Watson & Pennebaker, 1989), which may have been what the PANAS scale was measuring, rather than neuroticism. As a strong association has been found between PTSD and depression in those with SCI

(Nielsen, 2003b), future studies should also consider including a measure of depression.

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Appendices

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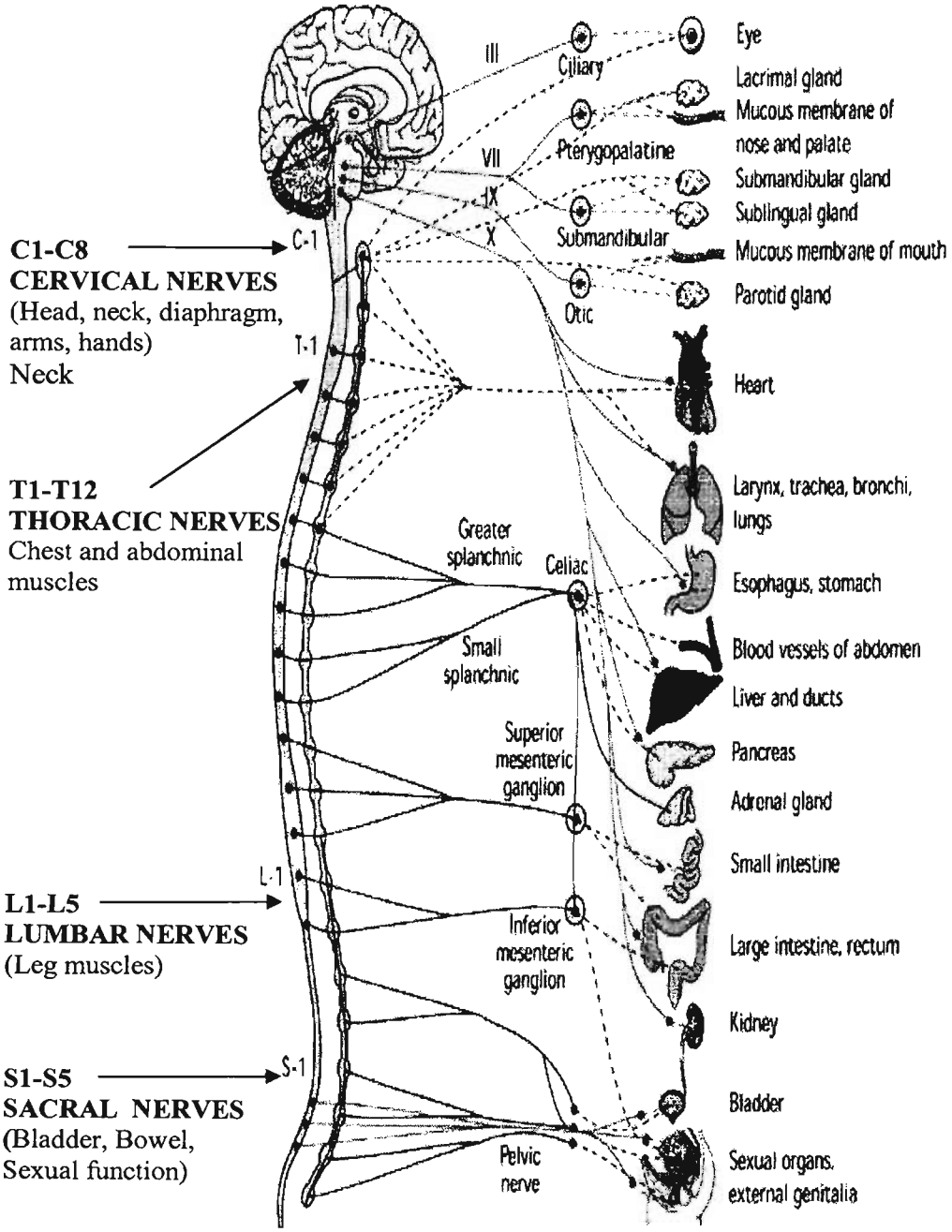
Appendix 2: Areas affected by Damage to Motor and Sensory Nerves in SCI



Appendix 3: DSM-IV Criteria for Posttraumatic Stress Disorder

A. The person has been exposed to a traumatic event in which both of the following have been present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others (2) the person's response involved intense fear, helplessness, or horror.

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.
(2) recurrent distressing dreams of the event.
(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur upon awakening or when intoxicated).
(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma
(2) efforts to avoid activities, places, or people that arouse recollections of the trauma
(3) inability to recall an important aspect of the trauma
(4) markedly diminished interest or participation in significant activities
(5) feeling of detachment or estrangement from others
(6) restricted range of affect (e.g., unable to have loving feelings)
(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep
(2) irritability or outbursts of anger
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

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

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

Acute if duration of symptoms is less than 3 months
Chronic if duration of symptoms is 3 months or more
With delayed onset if onset of symptoms is at least 6 months after the stressor

Appendix 4: Notes for Contributors: British Journal of Health Psychology

Notes for Contributors

The aim of the British Journal of Health Psychology is to provide a forum for high quality research relating to health and illness. The scope of the journal includes all areas of health psychology across the life span, ranging from experimental and clinical research on aetiology and the management of acute and chronic illness, responses to ill-health, screening and medical procedures, to research on health behaviour and psychological aspects of prevention. Research carried out at the individual, group and community levels is welcome, and submissions concerning clinical applications and interventions are particularly encouraged.

The following types of paper are invited:

- papers reporting original empirical investigations;
- theoretical papers which may be analyses or commentaries on established theories in health psychology, or presentations of theoretical innovations;
- review papers, which should aim to provide systematic overviews, evaluations and interpretations of research in a given field of health psychology;
- methodological papers dealing with methodological issues of particular relevance to health psychology.

1. Circulation

The circulation of the Journal is worldwide. Papers are invited and encouraged from authors throughout the world.

2. Length

Papers should normally be no more than 5,000 words, although the Editor retains discretion to publish papers beyond this length in cases where the clear and concise expression of the scientific content requires greater length.

3. Reviewing

The journal operates a policy of anonymous peer review. Papers will normally be scrutinised and commented on by at least two independent expert referees (in addition to the Editor) although the Editor may process a paper at his or her discretion. The referees will not be aware of the identity of the author. All information about authorship including personal acknowledgements and institutional affiliations should be confined to the title page (and the text should be free of such clues as identifiable self-citations e.g. 'In our earlier work...').

4. Online submission process

1) All manuscripts must be submitted online at http://bjhp.edmgr.com .

First-time users: click the REGISTER button from the menu and enter in your details as instructed. On successful registration, an email will be sent informing you of your user name and password. Please keep this email for future reference and proceed to LOGIN. (You do not need to re-register if your status changes e.g. author, reviewer or editor).
Registered users: click the LOGIN button from the menu and enter your user name and password for immediate access. Click 'Author Login'.

2) Follow the step-by-step instructions to submit your manuscript.

3) The submission must include the following as separate files:

- Title page consisting of manuscript title, authors' full names and affiliations, name and address for corresponding author – Editorial Manager Title Page for Manuscript Submission
- Abstract
- Full manuscript omitting authors' names and affiliations. Figures and tables can be attached separately if necessary.

4) If you require further help in submitting your manuscript, please consult the Tutorial for Authors - Editorial Manager - Tutorial for Authors
Authors can log on at any time to check the status of the manuscript.

5. Manuscript requirements

- Contributions must be typed in double spacing with wide margins. All sheets must be numbered.
- Tables should be typed in double spacing, each on a separate page with a self-explanatory title. Tables should be comprehensible without reference to the text. They should be placed at the end of the manuscript with their approximate locations indicated in the text.
- Figures can be included at the end of the document or attached as separate files, carefully labelled in initial capital/lower case lettering with symbols in a form consistent with text use. Unnecessary background patterns, lines and shading should be avoided. Captions should be listed on a separate page. The resolution of digital images must be at least 300 dpi.
- For articles containing original scientific research, a structured abstract of up to 250 words should be included with the headings: Objectives, Design, Methods, Results, Conclusions. Review articles should use these headings: Purpose, Methods, Results, Conclusions - British Journal of Health Psychology - Structured Abstracts Information
- For reference citations, please use APA style. Particular care should be taken to ensure that references are accurate and complete. Give all journal titles in full.
- SI units must be used for all measurements, rounded off to practical values

if appropriate, with the Imperial equivalent in parentheses.

- In normal circumstances, effect size should be incorporated.
- Authors are requested to avoid the use of sexist language.
- Authors are responsible for acquiring written permission to publish lengthy quotations, illustrations etc for which they do not own copyright.

For guidelines on editorial style, please consult the APA Publication Manual published by the American Psychological Association, Washington DC, USA (http://www.apastyle.org).

6. Publication ethics

Code of Conduct - Code of Conduct, Ethical Principles and Guidelines
Principles of Publishing - Principle of Publishing

7. Supplementary data

Supplementary data too extensive for publication may be deposited with the British Library Document Supply Centre. Such material includes numerical data, computer programs, fuller details of case studies and experimental techniques. The material should be submitted to the Editor together with the article, for simultaneous refereeing.

8. Post acceptance

PDF page proofs are sent to authors via email for correction of print but not for rewriting or the introduction of new material. Authors will be provided with a PDF file of their article prior to publication for easy and cost-effective dissemination to colleagues.

9. Copyright

To protect authors and journals against unauthorised reproduction of articles, The British Psychological Society requires copyright to be assigned to itself as publisher, on the express condition that authors may use their own material at any time without permission. On acceptance of a paper submitted to a journal, authors will be requested to sign an appropriate assignment of copyright form.

10. Checklist of requirements

- Abstract (100-200 words)
- Title page (include title, authors' names, affiliations, full contact details)
- Full article text (double-spaced with numbered pages and anonymised)
- References (APA style). Authors are responsible for bibliographic accuracy and must check every reference in the manuscript and proofread again in the page proofs.
- Tables, figures, captions placed at the end of the article or attached as separate files.

Appendix 5: Demographic Information Sheet

Demographic Information

Potential predictors of stress symptoms in spinal cord injury

Researcher: Mal Hatcher, Trainee Clinical Psychologist

(Please tick the boxes where required)

Name: _____

Gender: Male ☐ Female ☐ Date of Birth: _____/_____/_____ (dd/mm/yy)

Consultant: _____

Date of Injury: _____/_____/_____ (dd/mm/yy)

Type of Injury: Paraplegia ☐ Tetraplegia ☐

Level of Injury: Complete ☐ Incomplete ☐

Cause of Injury: _____

_____

(Continue on back of this page if necessary)

Educational level:
less than or up to age 16 ☐ between age 17 and 18 ☐ older than age 18 ☐

Employment status (prior to injury): Full time ☐ Part time ☐

Non-manual/professional ☐

Manual ☐

Homemaker ☐

Student ☐

Unemployed ☐

Appendix 6: Impact of Event Scale

Impact of Event Scale (IES)

On (date): _____

You experienced (life event): an accident resulting in a SCI

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**. If they did not occur during that time, please mark the "not at all" column.

	Frequency			
	Not at all	Rarely	Sometimes	Often
1. I thought about it when I didn't mean to.				
2. I avoided letting myself get upset when I thought about it or was reminded of it.				
3. I tried to remove it from memory.				
4. I had trouble falling asleep or staying asleep, because of the pictures or thoughts about it that came into my mind.				
5. I had waves of strong feelings about it.				
6. I had dreams about it.				
7. I stayed away from reminders of it.				
8. I felt as if it hadn't happened or it wasn't real.				
9. I tried not to talk about it.				
10. Pictures about it popped into my mind.				
11. Other things kept making me think about it.				
12. I was aware that I still had a lot of feelings about it, but I didn't deal with them.				
13. I tried not to think about it.				
14. Any reminder brought back feelings about it.				
15. My feelings about it were kind of numb.				

Appendix 7: Positive and Negative Affect Scale

POSITIVE AND NEGATIVE AFFECT SCHEDULE

Name: .....

Date: ..... Record Number: .....

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you generally feel this way, that is, how you feel on average.

Use the following scale to record your answers.

1	2	3	4	5
Very slightly or not at all	*a little*	*moderately*	*quite a bit*	*extremely*

.....interested

.....distressed

.....excited

.....upset

.....strong

.....guilty

.....scared

.....hostile

.....enthusiastic

.....proud

.....irritable

.....alert

.....ashamed

.....inspired

.....nervous

.....determined

.....attentive

.....jittery

.....active

.....afraid

Appendix 8: Acceptance of Injury Scale

Name: .....

Date: ..... Record Number: .....

Instructions

Please respond to each of the following items by choosing a number from 1 to 5 on the scale adjacent to the item which you feel best describes you. Then **circle** the number you have chosen. There are no right answers to any of the questions.

1. I have a hard time adjusting to the limitations of the injury.

Strongly agree 1 2 3 4 5 Strongly disagree

2. Because of the injury, I miss the things I like to do most.

Strongly agree 1 2 3 4 5 Strongly disagree

3. The injury makes me feel useless at times.

Strongly agree 1 2 3 4 5 Strongly disagree

4. Health problems make me more dependent on others than I want to be.

Strongly agree 1 2 3 4 5 Strongly disagree

5. The injury makes me a burden on my family and friends.

Strongly agree 1 2 3 4 5 Strongly disagree

6. The injury does not make me feel inadequate.

Strongly agree 1 2 3 4 5 Strongly disagree

7. I will never be self-sufficient enough to make me happy.

Strongly agree 1 2 3 4 5 Strongly disagree

8. I think people are often uncomfortable being around me because of the injury.

Strongly agree 1 2 3 4 5 Strongly disagree

Appendix 9: Perceived Social Support for Families

PERCEIVED SOCIAL SUPPORT SCALE

DIRECTIONS: The statements which follow refer to feelings and experiences which occur to most people at one time or another in their relationships with their ***FAMILIES***. For each statement there are three possible answers: **YES, NO, DON'T KNOW.**

Please choose your answer by ticking the relevant box for each item.

	YES	NO	DON'T KNOW
1. My family gives me the moral support I need.			
2. I get good ideas about how to do things or make things from my family.			
3. Most other people are closer to their family than I am.			
4. When I confide in the members of my family who are closest to me, I get the idea that it makes them uncomfortable.			
5. My family enjoys hearing about what I think.			
6. Members of my family share many of my interests.			
7. Certain members of my family come to me when they have problems or need advice.			
8. I rely on my family for emotional support.			
9. There is a member of my family I could go to if I were just feeling down, without feeling funny about it later.			
10. My family and I are very open about what we think about things.			
11. My family is sensitive to my personal needs.			
12. Members of my family come to me for emotional support.			
13. Members of my family are good at helping me solve problems.			
14. I have a deep sharing relationship with a number of members of my family.			
15. Members of my family get good ideas about how to do things or make things from me.			
16. When I confide in members of my family, it makes me uncomfortable.			
17. Members of my family seek me out for companionship.			
18. I think that my family feel that I'm good at helping them solve problems.			
19. I don't have a relationship with a member of my family that is as close as other people's relationships with family members.			
20. I wish my family were much different.			

Appendix 9a: Perceived Social Support for Friends

PERCEIVED SOCIAL SUPPORT SCALE

DIRECTIONS: The statements which follow refer to feelings and experiences which occur to most people at one time or another in their relationships with ***FRIENDS***. For each statement there are three possible answers: **YES, NO DON'T KNOW.**

Please choose your answer by ticking the relevant box for each item.

	YES	NO	DON'T KNOW
1. My friends give me the moral support I need.			
2. Most other people are closer to their friends than I am.			
3. My friends enjoy hearing about what I think.			
4. Certain friends come to me when they have problems or need advice.			
5. I rely on my friends for emotional support.			
6. If I felt that one or more of my friends were upset with me, I'd just keep it to myself.			
7. I feel that I'm on the fringe in my circle of friends.			
8. There is a friend I could go to if I were just feeling down, without feeling funny about it later.			
9. My friends and I are very open about what we think about things.			
10. My friends are sensitive to my personal needs.			
11. My friends come to me for emotional support.			
12. My friends are good at helping me to solve problems.			
13. I have a deep sharing relationship with a number of friends.			
14. My friends get good ideas about how to do things, or make things, from me.			
15. When I confide in friends, it makes me feel uncomfortable.			
16. My friends seek me out for companionship.			
17. I think that my friends feel that I'm good at helping them solve problems.			
18. I don't have a relationship with a friend that is as intimate as other people's relationships with friends.			
19. I've recently had a good idea about how to do something from a friend.			
20. I wish my friends were much different.			

Appendix 10: Copyright permission to use the Toronto Alexithymia Scale

From: Graeme Taylor (graeme.taylor@utotonto.ca)
To: Mal Hatcher
Subject: Copyright from gtaylorpsychiatry.org

Dear Mrs Hatcher:

This message is to confirm that we received from the University of Southampton a cheque for US$40.00 in October, 2004 as payment of the copyright fee for the 20-Item Toronto Alexithymia Scale (TAS-20). At that time we granted you permission to use the TAS-20 in research for your dissertation.

Yours truly,

Graeme J. Taylor, MD
Professor of Psychiatry
University of Toronto
email: graeme.taylor@utoronto.ca
www.gtaylorpsychiatry.org

Appendix 11: Post Traumatic Cognitions Inventory

PTCI (Foa, Ehlers, Clark, Tolin & Orsillo, 1999, Psychological Assessment)

This questionnaire lists different thoughts which people may have after a traumatic experience. In this questionnaire we are interested in the way YOU thought, IN THE LAST MONTH, in regard to the traumatic event that you have experienced.

Please read each statement carefully and decide how much you have AGREED or DISAGREED with each statement during the last month.

For each of the thoughts, please show your answer by choosing the number from the scale below which BEST DESCRIBES HOW MUCH YOU AGREE WITH THE STATEMENT and placing the number next to that statement. People react in many different ways; there are no right or wrong answers to these statements.

1	2	3	4	5	6	7
Totally Disagree	Disagree Very Much	Disagree Slightly	Neutral	Agree Slighty	Agree Very Much	Totally Agree

___ **1. My reactions since the event mean that I am going crazy.**
___ **2. Somebody else would have stopped the event from happening.**
___ **3. I feel like an object, not a person.**
___ **4. I have to be on guard all the time.**
___ **5. Nothing good can happen to me anymore.**
___ **6. I will not be able to control my anger and will do something terrible.**
___ **7. The event happened to me because of the sort of person I am.**
___ **8. The world is a dangerous place.**
___ **9. I feel like I don't know myself anymore.**
___ **10. If I think about the event, I will not be able to handle it.**
___ **11. People can't be trusted.**
___ **12. My life has been destroyed by the event.**
___ **13. Somebody else would not have gotten into this situation.**
___ **14. I can't deal with even the slightest upset.**
___ **15. I feel dead inside.**
___ **16. People are not what they seem.**
___ **17. I can't rely on myself.**
___ **18. There is something wrong with me as a person.**
___ **19. I will never be able to feel normal emotions again.**
___ **20. I have to be especially careful because you never know what can happen next.**
___ **21. My reactions since the event show that I am a lousy coper.**
___ **22. I am inadequate.**
___ **23. You can never know who will harm you.**
___ **24. I feel isolated and set apart from others.**
___ **25. I have no future.**
___ **26. There is something about me that made the event happen.**
___ **27. I have permanently changed for the worse.**
___ **28. I can't rely on other people.**
___ **29. I can't trust that I will do the right thing.**
___ **30. I am a weak person.**
___ **31. The event happened because of the way I acted.**
___ **32. I used to be a happy person but now I am always miserable.**
___ **33. I can't stop bad things from happening to me.**
___ **34. I will not be able to tolerate my thoughts about the event, and I will fall apart.**
___ **35. I will not be able to control my emotions, and something terrible will happen.**
___ **36. You never know when something terrible will happen.**
___ **37. I should be over this by now.**

Appendix 12: University of Southampton Ethics Committee Approval

Ethics Application

From: Smith K.M. [K.M.Smith@soton.ac.uk]

Sent: 28 October 2004 15:08

To: mh1002@.soton.ac.uk

Subject: Ethics Application

Dear Mal

Re: Potential predictors of PTSD symptoms in spinal cord injury

The above titled application was approved by the School of Psychology Ethics Committee on 28 October 2004.

Should you require any further information, please do not hesitate in contacting me. Please quote reference CLIN/03/59.

Best wishes,

Kathryn

Secretary to the Ethics Committee

Appendix 13: Salisbury Hospital Ethics Committee Approval

Salisbury Health Care NHS
Trust South Wiltshire Primary
Care Trust

R&D Management Committee
South Wiltshire R&D Consortium
Room 9, Level 4
Salisbury District Hospital
Salisbury
Wiltshire
SP2 8BJ

Telephone: (01722)425027
Fax: (01722425037
email: stef.scott@salisbury.nhs.uk
or myra.stevens@salisbury.nhs.uk

8 February 2005

Dear Mrs Hatcher

RDMC15/04/05: Potential predictors of Post Traumatic Stress Disorder (PTSD) Symptoms in Spinal Cord injury

Thank you for submitting the amendments regarding the above project to the South Wiltshire Consortium R&D Management Committee (Consortium RDMC). The Consortium RDMC noted the amendments through Chairman's Actions. The documents reviewed were as follows:

1. Letter from Mal Hatcher dated 24/1705
2. LREC approval letter dated 21/12/04 and 12/1/05 regarding amendment to study
3. Amendment to Participant Information Sheet
4. Amendment to Inpatient letter
5. Amendment to Outpatient letter
6. Amendment to Postal letter

The study was approved subject to the following conditions:

a) The study is conducted in accordance with the Research Governance Framework for Health and Social Care. The responsibilities of the Chief Investigator and Local Researchers are appended for ease of reference. A copy of the Framework may be found in the project file;

b) All clinical trials involving medicines are conducted in accordance with The Medicines for Human Use (Clinical Trials) Regulations 2004.

c) The Local Principal Investigator maintains the project file for this research project (attached to Local Principal Investigator copy of this letter) on behalf of the local team;

d) Salisbury Health Care' NHS Trust and South Wiltshire PCT do not have any research governance responsibilities for this project over and above the responsibilities described in the Research governance Framework for Health and Social Care for 'Organisations providing care', and, where appropriate, for 'Universities and other organisations employing researchers';

e) The Consortium is informed of any proposed changes or amendments to or deviations from the protocol;

f) All local serious adverse events are handled in accordance with the Trust and PCT policies for reporting and handling adverse events. Copies of Trust & PCT policies may be found on the intranet, Integrated Clinical Information Database, the Freedom of Information websites, and the Library;

g) Progress reports will be submitted to the Consortium RDMC on an annual basis, and a final report at the conclusion of the study. Wherever possible, these reports will be the same as that submitted elsewhere (e.g. to funding bodies, research sponsors and research ethics committees);

h) At the end of the study, the results of the research must be made available to the research participants. A copy of this report should also be sent to the CRDMC, for further dissemination as appropriate.

The project has been allocated a Local Start Date 8/2/05 and Local End Date 7/8/05.

The Consortium is required by the Department of Health to record details of all R&D projects on a database and submits details of all non-commercial R&D activity to the National Research Register. Details of the above project will be recorded on the Consortium R&D database, and details of all non-commercial projects will be included in the Consortium's next submission to the National Research Register.

If you do not intend to proceed with this project then please let the Consortium RDMC know so that we can amend our records.

If you have any questions about this letter, then please do not hesitate to contact Stef Scott, R&D Manager for the R&D Consortium, on 01722 425027

Yours sincerely

Dr. Sally Nelson/Mr. Alistair Flowerdew
Chair of the Consortium R&D Management Committee

Copy to:

Research Sponsor	University of Southampton
Chief Investigator	Mrs Mal Hatcher
Local Principal Investigator	Mrs Mal Hatcher
Local Researcher(s)	Messrs Tromans, Soopramanien, El-Shafei

Appendix 14: Consent Letter from Consultants, SCI Unit, Salisbury

Salisbury Health Care
NHS Trust
The Duke of Cornwall Spinal Treatment Centre
Salisbury District Hospital
Salisbury
Wiltshire, SP2 8BJ
Direct Line: (01722) 429007
Fax: (01722)336550

25 November 2004

Our Ref: AS/sep

Mrs Mal Hatcher BSc MSc
Clinical Psychologist in Training
School of Psychology
University of Southampton
Highfield
Southampton
S017 1BJ

Dear Mal

Potential predictors of post traumatic stress disorder in spinal cord injuries

Having read the proposal and discussed the study in meetings with you we are agreed that we are happy for you to recruit patients from the Duke of Cornwall Spinal Treatment Centre and to co-operate with you in order to bring this research to a successful conclusion.

Yours sincerely,

Mr A Tromans
Consultant in Spinal Injuries

Dr A Soopramanien
Consultant in Spinal Injuries and Rehabilitation Medicine

Mr A El-Shafei
Consultant in Spinal Injuries

Appendix 15: Introductory Letter from Consultants

Salisbury Health Care NHS
NHS Trust

Dr A Soopramanien
The Duke of Cornwall Spinal Treatment Centre
Salisbury District Hospital
Salisbury
Wiltshire, SP2 8BJ
Direct Line: (01722) 429007
Fax: (01722) 336550

«Name»
«Address_Line_1»
«Line_2»
«Line3»
«Postcode»

«Date»

«Dear»

Re: Potential predictors of stress symptoms in spinal cord injury (Ethics No 04/Q2008/26)

A doctoral student at the University of Southampton is conducting a research project with which she would like to ask your help. The purpose of the project is to look at whether it is possible to predict patients who develop symptoms of posttraumatic stress after a spinal cord injury compared to those who do not. The researcher on this study would like to ask you to take part and her letter of invitation is attached. This explains the purpose of the project and what would be involved if you decide to take part. It is entirely your choice whether you would like to take part or not and your decision will not affect your treatment in my clinic in any way.

If you have any questions about the project please contact the researcher, Mrs Mal Hatcher (023 8059 5321).

Yours sincerely,

Dr A Soopramanien[1]
Consultant in Spinal Injuries

[1] The name of the relevant consultant will appear here and in the address block at the top of the letter

Appendix 16a: Invitation Letter to Inpatient Participants

Date[1]

Dear Patient,

Re: Potential predictors of stress symptoms in spinal cord injury (Ethics No 04/Q2008/26)

I would like to ask you for your help in the above research project. The project has been reviewed by the Salisbury Research Ethics Committee.

The purpose of the research is to find out about the experiences of people, like you, who have suffered spinal cord injuries from an accident. The study will look at whether there are differences in how people are feeling, or what they are experiencing, depending on the type of spinal cord injury they sustained or when the accident happened.

If you are interested in taking part in this research I would be grateful if you could return the attached reply slip, using the enclosed envelope, to a member of staff. I will then contact you to arrange a time to meet. You will have the opportunity to ask questions during our meeting. This meeting will be in the spinal treatment centre in a quiet place where we cannot be overheard. If you are happy to take part, I will ask you to sign a consent form. You will then be asked to provide some demographic information, (for example your age, the type of injury you have) and complete a number of questionnaires. The questionnaires will be looked at to find out what people who have suffered a spinal cord injury after an accident might have experienced or be feeling. The demographic information will be looked at to see if there are any differences between what people experience or feel depending upon, for example, their age or the type of injury they have.

Taking part will take approximately 40 minutes. All the information gathered in this project will be made anonymous and no individual will be identifiable from the summarised results. Your involvement in the research may not have direct benefit on your care but it may influence the care given to future patients.

You are under no obligation to take part in the research and you are free to withdraw from the research at any time without needing to give an explanation. If you decide not take part, or to withdraw from the project, your health care would not be affected in any way.

Please contact me on the above number if you have any questions.

Thank you for taking the time to read this letter.

Yours sincerely,

Mrs Mal Hatcher
Trainee Clinical Psychologist

Supervised by: Dr Anne Waters, Consultant Clinical Psychologist
Mr A Tromans, Consultant in Spinal Injuries

[1] Letter printed on university headed paper

Appendix 16b: Invitation Letter to Outpatient Participants

Date[1]

Dear Patient,

Re: Potential predictors of stress symptoms in spinal cord injury (Ethics No 04/Q2008/26)

I would like to ask you for your help in the above research project. The project has been reviewed by the Salisbury Research Ethics Committee.

The purpose of the research is to find out about the experiences of people, like you, who have suffered spinal cord injuries from an accident. The study will look at whether there are differences in how people are feeling, or what they are experiencing, depending on the type of spinal cord injury they sustained or when the accident happened.

If you are interested to take part in this research I would be grateful if you could return the attached reply slip, using the pre-paid envelope. I will then ring you to arrange a time to meet, either before or after your appointment with your doctor in out-patients. You will have the opportunity to ask questions over the phone and during our meeting. This meeting will be in the out-patients clinic in a quiet place where we cannot be overheard. If you are happy to take part, I will ask you to sign a consent form. You will then be asked to provide some demographic information, (for example your age, the type of injury you have) and complete a number of questionnaires. The questionnaires will be looked at to find out what people who have suffered a spinal cord injury after an accident might have experienced or be feeling. The demographic information will be looked at to see if there are any differences between what people experience or feel depending upon, for example, their age or the type of injury they have.

Taking part will take approximately 40 minutes. All the information gathered in this project will be made anonymous and no individual will be identifiable from the summarised results. Your involvement in the research may not have direct benefit on your care but it may influence the care given to future patients.

You are under no obligation to take part in the research and you are free to withdraw from the research at any time without needing to give an explanation. If you decide not take part, or to withdraw from the project, your health care would not be affected in any way.

Please contact me on the above number if you have any questions.

Thank you for taking the time to read this letter.

Yours sincerely,

Mrs Mal Hatcher
Trainee Clinical Psychologist

Supervised by: Dr Anne Waters, Consultant Clinical Psychologist
Mr A Tromans, Consultant in Spinal Injuries

[1] Letter printed on university headed paper

Appendix 16c: Invitation Letter to Postal Participants

Dear Patient,[1]

Re: Potential predictors of stress symptoms in spinal cord injury (Ethics No 04/Q2008/26)

I would like to ask you for your help in the above research study. The study has been reviewed by the Salisbury Research Ethics Committee.

The purpose of the research is to find out about the experiences of people, like you, who have suffered spinal cord injuries from an accident. The study will look at whether there are differences in how people are feeling, or what they are experiencing, depending on the type of spinal cord injury they sustained or when the accident happened.

If you are interested in taking part in this research I would be grateful if you could complete and return the enclosed consent form, demographic information (for example your age, the type of injury you have) and questionnaires, using the pre-paid enclosed envelope, by ____[2]

The questionnaires will be looked at to find out what people who have suffered a spinal cord injury after an accident might have experienced or be feeling. The demographic information will be looked at to see if there are any differences between what people experience or feel depending upon, for example, their age or the type of injury they have.

All the information gathered in this study will be made anonymous and no individual will be identifiable from the summarised results. Your involvement in the research may not have direct benefit on your care but it may influence the care given to future patients.

You are under no obligation to take part in the research and you are free to withdraw from the research at any time without needing to give an explanation. If you decide not take part, or to withdraw from the project, your health care would not be affected in any way.

Please contact me on the above number if you have any questions.

Thank you for taking the time to read this letter.

Yours sincerely,

Mrs Mal Hatcher
Trainee Clinical Psychologist

Supervised by: Dr Anne Waters, Consultant Clinical Psychologist
Mr A Tromans, Consultant in Spinal Injuries

[1] Letter printed on university headed paper

[2] Date pack to be returned by inserted here

Appendix 17: Participant Information Sheet

Potential predictors of stress symptoms in spinal cord injury (Ethics No 04/Q2008/26)

PARTICIPANT INFORMATION SHEET

You are being asked to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Please contact Mal Hatcher at the number at the end of this information sheet if there is anything that is not clear or if you would like more information. Thank you for reading this.

What is the purpose of the study?

This study is trying to find out about the experiences of people, like you, who have suffered spinal cord injuries from an accident. The study will look at whether there are differences in how people are feeling, or what they are experiencing, depending on the type of spinal cord injury they sustained or when the accident happened.

Why have I been chosen?

In order to gain a cross-section of opinions a number of individuals have been asked to take part. In this way, I hope to gather information from a representative sample of people.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care you receive.

What will happen if I take part?

You will be asked to fill in some questionnaires and to provide demographic information, for example your age or the type of spinal injury you have, and return them all either to the researcher, if she is present, or in a prepaid envelope. The results of the questionnaires will be looked at to find out what people who have suffered a spinal cord injury after an accident might have experienced or be feeling.

How do I consent to take part?

You consent to take part by signing and returning two of the three consent forms either to the researcher, if she is present, or in the prepaid envelope. The researcher will keep one of the consent forms and the other will go into your hospital notes. The third consent form is for you to keep.

Your participation is voluntary and you are free to withdraw from this study at any time, without giving any reason, without your medical care or legal rights being affected. To do this just contact the number at the end of this information sheet and ask for your answers to the questionnaires to be removed from the computer.

Will my taking part in the study be kept confidential?

All information will be kept strictly confidential. The information used in the study will only have a study code number on. Nobody will be able to identify you from this code number. Immediately I receive the questionnaires and consent form from you I will separate them and code the questionnaires so that no one will know that they are yours.

What are the potential benefits of taking part?

Your involvement in the research will not have any direct benefit on your care but it may influence the care given to future patients.

What are the potential risks of taking part?

The questionnaires being used are well established and standardised but in the very unlikely event that you find completing them distressing you can contact me, Mal Hatcher, on the number below, during working hours, and I will be happy to discuss these issues with you on the phone. If you then require further help with these issues I can advise you of the appropriate channels to go through.

What will happen to the results of the study?

A report of the study will be written. A summary of the results will be made available on request.

Who is organising and funding the research?

I am a third year clinical trainee at the University of Southampton, Doctoral Programme in Clinical Psychology. This research is being conducted as part of my training.

Who has reviewed the study?

The Department of Psychology Research Ethics Committee, University of Southampton and an NHS Research Ethics Committee have reviewed the study.

If you have any questions about your rights as a participant in this research or you feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton SO17 1BJ. Tel: 023 8059 3995

Contact for further information

If you have any questions, or you wish to request a summary of the study please contact: Mrs Mal Hatcher, Department of Clinical Psychology, University of Southampton, SO17 1PN.

Tel: 023 8059 5321. Email: mh1002@soton.ac.uk

Thank you.

Appendix 18: Participant Consent Form

CONSENT FORM

Potential predictors of stress symptoms in spinal cord injury (Ethics No 04/Q2008/26)

Name: _____

I have read the information sheet provided about the above study. I know that I can discuss this further with the researcher, Mal Hatcher, should I wish to do so.

I also understand that any information I give will be treated as strictly confidential.

I understand that I am free to withdraw from this study at any time, without giving a reason, and without this affecting my medical care or legal rights in any way.

Please tick:

☐ I *agree* to participate in this study.

☐ I *do not agree* to participate in this study.

☐ I wish to receive a copy of the summary report of the research. My contact details are:

_____

_____

Signed: _____
(If a representative is signing on your behalf, please ask them to tick this box ☐)

Date: _____

Please post this form back to me in the prepaid envelope provided.
Thank you.

Mal Hatcher
Trainee Clinical Psychologist

Supervised by: Dr. Anne Waters, Consultant Clinical Psychologist.
Mr. A Tromans, Consultant in Spinal Injuries.

(1 copy for patient; 1 copy for researcher; 1 copy to be kept with hospital notes)

Appendix 19: Mean, standard deviation and range of scores for questionnaires

Scale or subscale name	Mean	St Dev (Range)
IES total score (IES_{total})	26.38	11.18 (15-57)
IES avoidance scale (IES_{avoid})	12.66	5.7 (7-28)
IES intrusion scale (IES_{intru})	13.73	6.27 (8-29)
PANAS Negative Affect (NA)	20.22	8.57 (10-46)
Acceptance of Injury ($Accpt_{Inj}$)	22.42	7.91 (8-40)
Alexithymia total score (TAS_{total})	53.57	13.40 (28-82)
Alexithymia difficulty identifying feelings (TAS_{dif})	19.30	7.34 (7-35)
Alexithymia difficulty describing feelings (TAS_{ddf})	13.61	5.22 (5-25)
Alexithymia externally oriented thinking (TAS_{eot})	20.66	4.69 (10-32)
Post Traumatic Cognitions Inventory ($PTCI_{total}$)	99.17	39.75 (37-220)
Post Traumatic Cognitions Inventory ($PTCI_{self}$)	2.35	1.29 (1-6)
Post Traumatic Cognitions Inventory ($PTCI_{selfblame}$)	2.56	1.53 (1-7)
Post Traumatic Cognitions Inventory ($PTCI_{world}$)	3.52	1.35 (1-7)
Perceived social support from family (Fam_{Sppt})	12.71	5.86 (0-20)
Perceived social support from friends ($Frnd_{Sppt}$)	12.16	5.61 (1-20)

Appendix 20: Spearman's correlation matrix for independent and dependent variables

Variable[a]	NA	AIS	SOC -fam	SOC -fr	TAS -dif	TAS -eot	PTCI -self	PTCI -world	PTCI -blame	AGE -inj	Time -since	IES -total	IES -avoid
NA	1												
AIS	-.640*	1											
SOC-fam	-.145	.132	1										
SOC-fr	-.154	.276**	.432**	1									
TAS-dif	.534**	-.414*	-.051	-.096	1								
TAS-eot	.022	-.118	-.075	-.239*	.150	1							
PTCI-self	.622**	-.610**	-.321**	-.421**	.564**	.180	1						
PTCI-world	.454**	-.407**	-.173	-.287**	.517**	.008	.636**	1					
PTCI-blame	.149	-.178	-.142	.029	.247*	.009	.388**	.297**	1				
AGE-inj	-.056	-.096	.112	.091	.058	-.046	.038	-.024	-.009	1			
Time-since	-.203*	.186	-.158	-.222*	-.179	-.143	-.114	-.039	-.012	-.582**	1		
IES-total	.600**	-.492**	-.062	-.129	.476**	.085	.576**	.459**	.182	.154	-.321**	1	
IES-avoid	.606**	-.453**	-.062	-.123	.425**	.015	.536**	.459**	.231*	.019	-.257**	-.944**	1
IES-intru	.524**	-.469**	.066	-.102	.475**	.142	.557**	.398**	.149	.190	-.344**	.933**	.780**

[a] NA = Negative affect; AIS = Acceptance of Injury Scale; SOCfam = Perceived social support from family; SOCfr = Perceived social support from friends; TASdif = difficulty identifying feelings; TASeot = externally oriented thinking; PTCIself = negative cognitions of self; PTCIworld = negative cognitions of world; PTCIblame = negative cognitions of selfblame; Ageinj = age at injury; Timesince – time since injury; IEStotal = total scores of Impact of Event Scale; IESavoid = avoidance subscale of Impact of Event Scale; IESintru = intrusion subscale of Impact of Event Scale.
* p<.05 **p< .01

Appendix 21: Stepwise regression of predictors of symptoms of PTSD

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	12.91	1.74	
	PTCIself	5.73	0.65	0.66***
Step 2				
	(Constant)	8.49	2.05	
	PTCIself	3.85	0.80	0.45***
	NAscore	0.44	0.12	0.34***

Step 1 - $R^2 = .44$, $F(1,100) = 78.24$, $p < .001$
Step 2 - $\Delta R^2 = .50$, $F(1,99) = 12.9$, $p < .001$

Overall model summary – $R^2 = .50$, $F(2,99) = 50.31$, $p < .001$

*p<.05, **p<.01, ***p<.001

Appendix 22: Summary of the hierarchical regression of predictors of symptoms of PTSD

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	8.49	2.05	
	PTCIself	0.44	0.12	0.34***
	NAscore	3.85	0.80	0.45***
Step 2				
	(Constant)	1.54	2.91	
	PTCIself	0.40	0.12	0.31 **
	NAscore	4.72	0.81	0.55***
	SOCFAMscore	0.45	0.14	0.23 *
Step 3				
	(Constant)	-2.25	3.28	
	PTCIself	0.42	0.11	0.32***
	NAscore	4.55	0.80	0.53***
	SOCFAMscore	0.40	0.14	0.21 **
	Ageinj	0.15	0.06	0.16 *

Step 1 - $R^2 = .50$, $F(2,99) = 50.31$, $p < .001$
Step 2 - $\Delta R^2 = .05$, $F(1,98) = 10.42$, $p < .01$
Step 3 - $\Delta R^2 = .02$, $F(1,97) = 5.451$, $p < .05$

Overall model summary – $R^2 = .58$, $F(4,97) = 32.89$, $p<.001$

*p<.05, **p<.01, ***p<.001

Appendix 23: Stepwise regression of predictors of symptoms of PTSD without negative affect

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	12.91	1.74	
	PTCIself	5.73	0.65	0.66***
Step 2				
	(Constant)	8.65	2.33	
	PTCIself	4.74	0.73	0.55***
	TASdif	0.34	0.13	0.23 **

Step 1 - $R^2 = .44$, $F(1,100) = 78.24$, $p < .001$
Step 2 - $\Delta R^2 = .48$, $F(1,99) = 7.04$, $p < .01$

Overall model summary – $R^2 = .48$, $F(2,99) = 45.01$, $p < .001$

*p<.05, **p<.01, ***p<.001

Appendix 24: Summary of the hierarchical multiple regression of predictors of symptoms of PTSD without negative affect

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	8.6	2.33	
	PTCIself	4.74	0.73	0.55***
	TASdif	0.34	0.13	0.23 **
Step 2				
	(Constant)	2.26	3.07	
	PTCIself	5.62	0.76	0.65***
	TASdif	0.28	0.13	0.18 *
	SOCFAMscore	0.44	0.14	0.23 **

Step 1 - $R^2 = .48$, $F(2,99) = 45.01$, $p < .001$
Step 2 - $\Delta R^2 = .52$, $F(1,98) = 9.27$, $p < .05$
Step 3 - $\Delta R^2 = .54$, $F(1,97) = 4.13$, $p < .05$

Overall model summary – $R^2 = .54$, $F(4,97) = 28.58$, $p<.001$

*p<.05, **p<.01, ***p<.001

Appendix 25: Stepwise multiple regression of predictors of symptoms of avoidance

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	3.80	1.10	
	NAscore	0.44	0.05	0.66***
Step 2				
	(Constant)	3.21	1.04	
	NAscore	0.29	0.06	0.43***
	PTCIself	1.55	0.41	0.35***

Step 1 - $R^2 = .43$, $F(1,100) = 76.57$, $p < .001$
Step 2 - $\Delta R^2 = .07$, $F(1,99) = 14.31$, $p < .001$

Overall model summary – $R^2 = .51$, $F(2,99) = 50.54$, $p < .001$

*p<.05, **p<.01, ***p<.001

Appendix 26: Summary of hierarchical regression of predictors of symptoms of avoidance

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	3.21	1.04	
	NAscore	0.29	0.06	0.43***
	PTCIself	1.55	0.41	0.35***
Step 2				
	(Constant)	0.47	1.51	
	NAscore	0.27	0.06	0.41***
	PTCIself	1.89	0.42	0.43***
	SOCFAMscore	0.18	0.07	0.18*

Step 1 - $R^2 = .51$, $F(2,99) = 50.54$, $p < .001$
Step 2 - $\Delta R^2 = .03$, $F(1,98) = 6.01$, $p < .05$

Overall model summary – $R^2 = .53$, $F(3,98) = 37.40$, $p<.001$

*p<.05, **p<.01, ***p<.001

Appendix 27: Summary of the stepwise regression of avoidance without negative affect

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	6.12	0.92	
	PTCIself	2.78	0.34	0.63***
Step 2				
	(Constant)	4.43	1.23	
	PTCIself	2.30	0.41	0.52***
	PTCIworld	0.80	0.40	0.19*

Step 1 - $R^2 = .40$, $F(1,100) = 65.86$, $p < .001$
Step 2 - $\Delta R^2 = .02$, $F(1,99) = 4.11$, $p < .05$

Overall model summary – $R^2 = .42$, $F(2,99) = 36.01$, $p<.001$

*p<.05, **p<.01, ***p<.001

Appendix 28: Summary of hierarchical regression of avoidance without negative affect

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	4.43	1.23	
	PTCIself	2.30	0.41	0.52***
	PTCIworld	0.80	0.40	0.19*
Step 2				
	(Constant)	1.13	1.72	
	PTCIself	2.64	0.42	0.60***
	PTCIworld	0.77	0.38	0.18*
	SOCFAMscore	0.21	0.08	0.21**

Step 1 - $R^2 = .42$, $F(2,99) = 36.01$, $p < .001$
Step 2 - $\Delta R^2 = .04$, $F(1,98) = 7.06$, $p < .01$

Overall model summary – $R^2 = .46$, $F(3,98) = 27.83$, $p<.001$

*p<.05, **p<.01, ***p<.001

Appendix 29: Stepwise multiple regression of predictors of symptoms of Intrusion

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	6.79	1.03	
	PTCIself	2.95	0.39	0.61***
Step 2				
	(Constant)	4.02	1.38	
	PTCIself	2.31	0.43	0.48***
	TASdif	0.22	0.08	0.26**
Step 3				
	(Constant)	5.87	1.60	
	PTCIself	2.23	0.43	0.46***
	TASdif	0.21	0.08	0.24**
	timesinc	-0.09	0.04	-0.17*

Step 1 - $R^2 = .37$, $F(1,100) = 58.64$, $p < .001$
Step 2 - $\Delta R^2 = .05$, $F(1,99) = 8.49$, $p < .01$
Step 3 - $\Delta R^2 = .03$, $F(1,98) = 4.62$, $p < .05$

Overall model summary – $R^2 = .45$, $F(3,98) = 26.26$, $p < .001$

*p<.05, **p<.01, ***p<.001

Appendix 30: Hierarchical multiple regression of predictors of symptoms of Intrusion

	Variable	*B*	*SE B*	β
Step 1				
	(Constant)	5.87	1.60	
	PTCIself	2.23	0.43	0.46***
	TASdif	0.21	0.08	0.24**
	Timesinc	-0.09	0.04	-0.17*
Step 2				
	(Constant)	2.26	2.06	
	PTCIself	2.70	0.45	0.56***
	TASdif	0.17	0.07	0.20*
	Timesinc	-0.07	0.04	-0.14*
	SOCFAMscore	0.23	0.09	0.21**

Step 1 - $R^2 = .45$, $F(3,98) = 26.26$, $p < .001$
Step 2 - $\Delta R^2 = .04$, $F(1,97) = 7.16$, $p < .01$

Overall model summary – $R^2 = .48$, $F(4,97) = 22.72$, $p<.001$

*p<.05, **p<.01, ***p<.001