

Past, Present and Future Directions in Posttraumatic Stress Disorder Research

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1.1 General Introduction

Natural and man-made disasters are part of humanity's past, present and future. These traumatic events and associated suffering are part of the human experience. The majority of people appear to successfully negotiate through these experiences and overcome them adaptively. However, for reasons not yet fully understood, some people do not successfully negotiate traumatic events. They go on to develop maladaptive ways of coping and various trauma-related disorders. One such trauma-related disorder is posttraumatic stress disorder (PTSD), a debilitating psychobiological disorder that is characterised by three core symptom clusters of intrusion, avoidance and hyperarousal.

These symptom clusters of intrusion, avoidance and hyperarousal are common in a large number of people in the immediate aftermath of a traumatic event but usually subside over subsequent weeks and months (Carr, Lewin, Webster, & Kenardy, 1997; Blanchard et al., 1996; Shalev, 2002). In people with PTSD the symptom clusters do not subside and may go on for months, years or a lifetime. Over time as these symptoms of intrusion, avoidance and hyperarousal are maintained, additional problems follow. The clinical picture that develops over time with PTSD is that of an individual who suffers not only psychologically but also physically, emotionally and socially.

This chapter provides a brief overview of PTSD that includes diagnostic criteria and associated problems, the history of PTSD and the current state of knowledge in PTSD research. Proposed theories of PTSD, treatment modalities and future directions in PTSD research are also outlined.

1.2 Diagnostic Criteria

The three symptom clusters of intrusion, hyperarousal, and avoidance that form the core diagnostic criteria for PTSD are gated by an additional criterion in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). This criterion (Criteria A) in the DSM-IV specifies that in order for a person to meet criteria for PTSD they must first have been exposed to a traumatic event in which both of the following were present:

“(1) The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.

(2) The person's response involved intense fear, helplessness, or horror" (American Psychiatric Association, 2000:467).

Criterion A sets PTSD apart from every other psychological disorder in the DSM-IV. It is the only disorder in which a diagnosis has to be connected to a concrete identifiable event. If a life-threatening event is identified then a specified threshold of symptoms related to the three core symptom clusters (criterion B, C & D) must be then experienced in order to meet diagnostic criteria for PTSD. These symptoms and related thresholds are outlined in the DSM-IV as follows:

Criteria B. The traumatic event is persistently re-experienced 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 though the traumatic event were occurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashbacks episodes, including those that occur on waking or when intoxicated.
- (4) Intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.
- (5) Physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

Criteria 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 foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

Criteria 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.

In children, criteria B, C, D may manifest differently in the form of disorganised and agitated behaviour, nightmares having no recognisable content and trauma themes identifiable in play. Criteria B, C, and D need to last more than one month (Criterion E) and the disturbance caused by the symptom clusters must cause “clinically significant distress or impairment in social, occupational, or other important areas of functioning” (Criterion F).

If the above criteria are met then the disorder is further classified in terms of the length of time the person has suffered from these symptoms since the traumatic event or the length of time between the development of symptoms and the traumatic event. A duration of symptoms less than three months is identified as acute PTSD, a duration of symptoms for three months or more is identified as chronic PTSD and the onset of symptoms at least 6 months after the traumatic event is identified as PTSD with delayed onset (American Psychiatric Association, 2000).

1.3 Additional Problems

In addition to the suffering associated with the core symptom clusters of intrusion, hyperarousal, and avoidance, an individual suffering from chronic PTSD is also likely to meet DSM-IV criteria for additional diagnoses. Personality disorders (Shea et al., 2000; Dunn, Yanasak, Schillaci, Simotas, Rehm, et al., 2004), particularly borderline personality disorder (Hidalgo & Davidson, 2000; Bolton, Mueser, Rosenberg, 2006), drug and alcohol abuse (Shipherd, Stafford & Tanner, 2005; Reynolds et al., 2005), nicotine dependence (Hapke et al., 2005; Koenan et al., 2006), anxiety disorders (Lawford, Young, Noble, Kann & Ritchie, 2006), psychosis (Kilcommons & Morrison, 2005) and major depression (O’Donnell, Creamer, Pattison,

2004) all share a high comorbidity with PTSD. Comorbidity of PTSD, major depression and a personality disorder is additionally associated with an increased risk of suicidal behaviour (Oquendo et al., 2005). Suicidal ideation is also a problem in older adults above 65 years of age with comorbid PTSD and depression (Rauch, Morales, Zubritsky, Knott & Oslin, 2006).

At present it is unclear whether a vulnerability to psychiatric illness underlies the high comorbidity or whether PTSD is itself a vulnerability to other disorders. What we do know, based on the available studies, is that somewhere between 50% and 90% of both adults (Yehuda and McFarlane, 1995) and children (Carrion, Weems, Ray & Reiss, 2002) who meet criteria for PTSD develop comorbid psychiatric disorders. Children with PTSD appear particularly susceptible to the future development of other anxiety disorders (see Cortes et al., 2005).

It is also common for chronic PTSD sufferers to experience problems with verbal memory (Bustamante, Mellman, David & Fins, 2001), working memory (Weber et al., 2005), the encoding of information and ability to inhibit irrelevant information (Cottencin et al., 2006; Vasterling et al., 2002) and problems with overall executive functioning (Koso & Hansen, 2005; Kanagaratnam & Asbjørnsen, 2006). Consistent with McEwan's research (1998; 2003; 2004) on the impact of sustained stress on the body there is also an increased risk of physical illness associated with PTSD. High rates of physical illness have been reported in both war veterans (Boscarino, 2004; Spiro, Hankin, Mansell, & Kazis, 2006; Marshall, 2006) and civilians with PTSD (Ouimette, Cronkite, Hensen, Prins, Gima et al., 2004; Lauterbach, Vora & Rakow, 2005). A study in the USA on the use of health services by people with PTSD, revealed that high users of medical health care (27.5%) were almost twice as likely to meet criteria for PTSD as low users (14.8%) of medical health care due to a direct relationship between PTSD and a number of health conditions (Deykin et al., 2001).

Given the above outlined problems it is not surprising that unstable family relationships (Galovski & Lyons, 2004), impaired social relationships (Cook, Riggs, Thompson, Coyne & Sheikh, 2004; Schnurr, Hayes, Lumney, McFall & Uddo, 2006) and difficulty with employment (Nicholls, Abraham, Conner, Ross & Davidson, 2006) are all associated with chronic PTSD. Galovski & Lyons (2003) identified that a diagnosis of PTSD increased the likelihood of unemployment by 150% and marital instability by 60%. Similarly, children with PTSD struggle in their interpersonal relationships and ability to work effectively. Verbal memory impairments and general memory impairments are specifically associated with trauma-exposed youth with PTSD compared with trauma-exposed youth without PTSD (Yasik, Saigh, Oberfield &

Halamandaris, 2006). In light of the memory difficulties it is not surprising that impaired learning is common in children (Saigh, Mroueh & Bremner, 1997; Scrimmin et al., 2006) and adults (Emdad, Söndergaard & Theorell, 2005) with PTSD.

1.4 History of PTSD

Although some authors (e.g., Bracken, 1998) view PTSD as a recent unhelpful construct of Western psychiatry, Lipton (1994) claims that the symptom clusters we now know as PTSD have been identified and written about for centuries in various literary sources such as the Old Testament and Greek mythology. Similarly, literature from the 1800's pertaining to the American civil war identifies an enduring condition known as '*soldier's heart*' that has similarities to PTSD (Vieweg et al., 2006). Observations recorded regarding '*war neuroses*', a condition identified in soldiers and World War II concentration camp survivors, in particular have a striking resemblance to PTSD criteria (Yehuda and McFarlane, 1995). References in early medical literature to '*railroad spine syndrome*' a disorder seen in survivors of early railway accidents and '*shell-shock syndrome*' a disorder identified in World War I soldiers, also bear a marked resemblance to PTSD symptoms (Lamprecht & Sack, 2002).

In psychological literature the core elements of PTSD can be found in the early psychoanalytic writings of Freud and Janet on neurosis (Wilson, 1995). Sigmund Freud writes in *The Introductory Lectures on Psychoanalysis* about a *traumatic neurosis*, identified by symptoms similar to PTSD symptoms and also connected to a concrete traumatic event. He states, "The traumatic neuroses give a clear indication that a fixation to the traumatic accident lives at their root" (1917/1966:274).

In subsequent years the core symptoms of PTSD continued to have a place in psychological writings as an identifiable response to a variety of traumatic events. They were given a variety of descriptive labels such as *rape trauma syndrome*, *the battered woman syndrome*, *the Vietnam veteran's syndrome* and *the abused child syndrome* (van der Kolk, Weisaeth & van der Hart, 1996). The first *Diagnostic and Statistical Manual of Mental Disorders* (DSM) referred to the cluster of symptoms as *Gross Stress Reaction* and then re-classified them in the next DSM edition as adjustment reactions of adult life (Brown, Fulton, Wilkeson & Petty, 2000).

The majority of the above disorders were viewed in their historical settings as an extreme stress response. In 1980 all of the above was subsumed under the label Post Traumatic Stress

Disorder and officially recognised as a distinct psychological disorder (Briere, 2004) operationalised by the criteria outlined earlier. In more recent years there has been increasing pressure to once again redefine the DSM criteria in order to keep pace with the growing body of research on the disorder (e.g., Maier, 2006; Spitzer, First & Wakefield, 2006).

1.5 Current State of Knowledge in PTSD Research

Official recognition of PTSD as a legitimate psychological diagnosis in 1980 opened the way for more specialised research into the disorder. Over ensuing years PTSD research has proliferated and provided a vast array of information relating to prevalence, course, memory and information processing, predictors/risk factors, relevant brain structures and their functional changes, and underlying neurobiology and physiology. The following pages provide a brief overview of the current state of knowledge in PTSD research.

1.5.1 Prevalence

Following official recognition of PTSD, initial research focused almost entirely on war veterans. The obvious trauma experienced in war and the link to *war neurosis* that had many parallels to PTSD criteria provided an obvious starting point for more specialised research into the disorder. Although a narrow focus, this provided a fertile field for researchers as prevalence rates of PTSD were found to be high in this population. For example, in Vietnam veterans alone the lifetime prevalence of PTSD was found to be 31%, with more than half of the veterans still suffering from it twenty years later (Creamer & Forbes, 2004).

However, subsequent research has identified that PTSD is also prevalent in the general population (Brown, Fulton, Wilkeson, & Petty, 2000) with sexual abuse being the highest cause of PTSD amongst women (Kessler, Sonnega, Bromet, Hughes & Nelson, 1995). A substantial proportion of individuals who experience other types of major trauma such as a severe car accident (Blanchard et al., 2003), physical abuse (Nicholls, Abraham, Conner, Ross & Davidson, 2006), natural disasters such as earthquakes (Livanou et al, 2005) and floods (Chae, Kim, Rhee & Henderson, 2005), man-made disasters such as terrorism (Blanchard, Rowell, Kuhn, Rogers & Wittrock, 2005; Gabriel et al., 2006; Ferrando et al., 2007), partner violence (Basile, Arias, Desai & Thompson, 2004), burns (El hamaoui, Yaalaoui, Chihabeddine, Boukind & Moussaoui, 2002) or a diagnosis of a life threatening illness (Kangas, Henry & Bryant, 2005) will also develop PTSD.

Child abuse alone contributes to a substantial number of people developing PTSD. Bremner (2000) estimates that in America alone four million women suffer from PTSD as a result of child abuse. This rate may be even higher in countries such as the Democratic Republic of the Congo where violent rape of children is a common weapon of war (see report by Internal Displacement Monitoring Centre, 2006 and Médecins Sans Frontières, 2004). Unfortunately there is little research carried out with such populations, so broader cross-cultural prevalence rates of child-abuse related PTSD are still unknown.

Refugees appear to be particularly vulnerable to the development of PTSD (see Lie, 2002; Nicholl & Thompson, 2004; Ai, Peterson & Uebelhor, 2002; Carlsson, Mortensen & Kastrup, 2005). Marsella, Friedman & Spain (1996) in their literature review of ethnocultural aspects of PTSD report PTSD rates of 54% to 93% in Indochinese refugees and rates of 25% to 52% among Central American immigrants. Bosnian refugee children had similarly high rates of PTSD (65%) one year after settlement in the USA (Weine et al., 1995).

Lifetime prevalence rates appear to vary across countries. In America the lifetime prevalence rate of PTSD is estimated to be approximately 8% (American Psychiatric Association, 2000). In non-Western countries where risk of exposure to war and violence is often higher, lifetime prevalence rates of PTSD are also higher. For example, available lifetime prevalence rates in four post-conflict countries (Algeria, Cambodia, Ethiopia, and Gaza) range from 16% to 37% (De Jong et al., 2001). Australia reports much lower prevalence rates of PTSD with 12-month prevalence rates reported to be only 1.33% (Creamer, Burgess, & McFarlane, 2001). However, whilst prevalence rates may seem low overall in Australia, the rate shoots up dramatically in psychiatric populations. A study by McFarlane, Bookless and Air (2001) revealed that in Australia, 50% of cases in a general psychiatric in-patient population had PTSD as the incident disorder. The high rates in this population highlight the broader clinical picture associated with PTSD.

Gender differences have been reported in PTSD rates with women at a higher risk of developing PTSD (Kessler et al., 1995; Olf, Langeland, Draijer & Gersons, 2007). However, data collected from the Australian National Survey of Mental Health and Well Being found no gender differences in prevalence rates (cited in Creamer, Burgess & McFarlane, 2001). Peters, Issakidis, Slade and Andrews (2006), researching this difference in prevalence rates, report that the gender differences found in previous studies can be attributable to differential responding in males and females according to the measurement instrument and culture.

1.5.2 Course

It is estimated that 60% of people suffering from PTSD will go on to recover in the absence of treatment (Kessler, Sonnega, Bromet, Hughes, Nelson, 1995). Although this statistic highlights that a natural recovery process can take place, it also highlights that a staggering number of people around the world continue to suffer the debilitating symptoms outlined previously. Sub-populations of PTSD sufferers, such as refugees and children, show even poorer natural recovery rates. The limited longitudinal studies available on the course of PTSD in children and adolescents suggest that PTSD is maintained over time if there are ongoing stressors (e.g., Shaw, Applegate & Schorr, 1996) or no interventions offered (Goenjian et al., 2005).

Similarly, refugees also appear to be at a higher risk of PTSD symptoms being maintained over time (see Hunt & Gakenyi, 2004; Steel, Frommer & Silove, 2004; Steel et al., 2006). They also appear particularly vulnerable to delayed onset PTSD. A prospective study conducted by Roth, Ekblad and Agren (2006) with adults who were evacuated from Kosovo revealed a dramatic increase in PTSD rates over three follow-ups as measured by both the Harvard Trauma Questionnaire and clinical assessment (37% to 80% at final follow-up 1.5 years later).

1.5.3 Memory Dysfunction and Information Processing Difficulties in PTSD

An additional problem to the core symptom clusters, particularly as the disorder becomes more chronic in nature is memory dysfunction. The burgeoning research literature shows that individuals with PTSD have impairments in remembering trauma neutral information, affecting their verbal, short-term, visual and long-term memory (see Buckley, Blanchard & Neill, 2000 for a review of information processing in PTSD).

In contrast, memory for trauma specific information in PTSD individuals appears to be heightened. For example, Creamer & Kelly (1997) found that although there was evidence for a subliminal (unconscious) avoidance of trauma related material in PTSD there was a supraliminal (conscious) bias towards threat-related material. Similarly, Buckley et al. (2000:1054), in their review of information processing in PTSD individuals concluded “individuals with PTSD evince an attentional bias toward trauma relevant stimuli at a post recognition stage of information processing”. In support of a bias at the post recognition stage of processing are studies that show an association between ex-consequencia reasoning (e.g., “I feel anxious therefore there must be danger”) and the maintenance of PTSD (see Engelhard & Arntz, 2005 for discussion on ex-consequencia reasoning and PTSD).

1.5.4 Predictors and Protective Factors for PTSD

The available research literature on PTSD offers a range of predictive factors and protective factors for the development and maintenance of PTSD. These identified predictors and protective factors for PTSD are presented in Tables, 1, 2, 3 and 4. For the purpose of this thesis the identified predictors are separated into factors that pre-exist the traumatic event (pre-trauma predictors), factors that are part of the traumatic event itself (peri-trauma predictors), factors that follow the traumatic event (post-trauma predictors) and general protective factors.

Table 1. Pre-trauma Predictors of PTSD

Pre-trauma Predictors
Prior psychological adjustment (Ozer, Best, Lipsey & Weiss, 2003).
Family history of psychopathology (Ozer, Best, Lipsey & Weiss, 2003).
A harm-avoidance pre-trauma personality (Gil, 2005).
Prior trauma (Ozer, Best, Lipsey & Weiss, 2003); chronic environmental adversity (Koenen, Moffitt, Poulton, Martin & Caspi, 2006)
Lower intelligence as measured by standardised measures of IQ (McNally & Shin, 1995; Vasterling et al., 2002). After controlling for extent of combat exposure in Vietnam veterans, lower intelligence still emerged as a risk factor for PTSD (Macklin et al., 1998).
Low self-efficacy (Heinrichs et al., 2005)

Table 2. Peri-trauma Predictors of PTSD

Peri-trauma Predictors
Peri-traumatic emotional responses and peri-traumatic dissociation (Harvey & Bryant, 2002; Ozer, Best, Lipsey & Weiss, 2003). However, research by Holbrook Hoyt, Stein, and Sieber (2001) revealed that if perceived threat to life at the time of the trauma is taken into account, dissociation is not a significant predictor.
Trauma severity (Bryant & Harvey, 1995; Brewin, Andrews & Valentine, 2000).
Torture (Silove, Steel, McGorry & Mohan, 2002).
Higher life threatening trauma and trauma severity associated with chronicity of PTSD in Vietnam veterans (Roy-Byrne et al., 2004; Schnurr, Lunney & Sengupta, 2004).
Threat exposure (Carr et al., 1997; Parslow, Jorm & Christensen, 2006)
The extent of physical injuries sustained (Altindag, Ozen & Sir, 2005; Koren, Norman, Cohen, Berman & Klein, 2005).
Loss or injury of a family member (Pfefferbaum, et al., 1999).
The experience of powerful emotions at the time of the trauma (Creamer, McFarlane & Burgess, 2005).
Fear of dying during the traumatic event (Blanchard et al., 1997).
Cognitive processing during trauma (assault) such as mental defeat, mental confusion and detachment (Dunmore, Clark & Ehlers, 2001).

Table 3. Post-trauma Predictors of PTSD

Post-trauma Predictors
Heart rate increased in car accident PTSD subjects when trauma specific cues were used. The severity of the accident and current symptomatology discriminated between PTSD subjects who did and did not respond with increased heart rate (Veazey, Blanchard, Hickling & Buckley, 2004).
Hyperarousal in the immediate aftermath of trauma (Ginzberg et al., 2003; Kutz & Dekel, 2006). A prospective study by Schell, Marshall, and Jaycox (2004) with survivors of community violence found that hyperarousal was the predominant predictor of the subsequent severity of all other symptoms and poor recovery.
Initial PTSD symptom severity (Blanchard et al., 2003).
After controlling for initial symptoms, trauma narrative disorganisation (repetition, non-consecutive chunks, and coherence) at one-week post trauma predicted trauma severity at 3 months post trauma (Jones, Harvey & Brewin, 2007).
Emotion-based ex-consequencia reasoning (e.g., intrusions trigger anxiety and subject concludes “I feel anxious therefore there must be danger”) predicted both acute and chronic PTSD (Engelhard & Arntz, 2005; Engelhard, van den Hout, Arntz, & McNally, 2002).
Characteristics of intrusions such as perceived ‘nowness’, vividness of the memories, and level of distress are predictive of persistent PTSD (Speckens et al., 2006).
Avoidance behaviour post-trauma (Zoellner, Jaycox, Watlington & Foa, 2003; Marx & Sloan, 2005).
Emotional numbing post-trauma predicts pervasive PTSD symptom disturbance (Breslau, Reboussin, Anthony & Storr, 2005), chronic PTSD (Marshall et al., 2006) and comorbid major depression (Kashdan, Elhai & Frueh, 2006).
Ongoing dissociation (Halligan & Yehuda, 2002)
Lack of post trauma social support (Ozer, Best, Lipsey & Weiss, 2003; Brewin, Andrews & Valentine, 2000).
Ongoing chronic system stress such as poverty, discrimination, poor social standing, and social inequalities related to race, gender or class (Kubiak, 2005).
Additional life stressors such as loss of job, serious illness, death or illness of close friends/family, broken relationships (Bryant & Harvey, 1995; Brewin, Andrews & Valentine, 2000; Maes, Mylle, Delmeire & Janca, 2001; Mayou, Ehlers & Bryant, 2002).
Persistence of traumatic physical injury (Blanchard et al., 1997). Severity of injury (Schynder, Moergeli, Klaghofer, & Buddberg, 2001).
Negative interpretations of symptoms such as dissociation, anger and intrusive memories (Ehlers, Mayou & Bryant, 2003; Mayou, Ehlers & Bryant, 2002).
Negative beliefs about self and world (Dunmore, Clark & Ehlers, 2001).
Lack of resilience, mastery and life satisfaction (Heilemann, Kury & Lee, 2005).

Pre-trauma predictors of posttraumatic stress disorder appear to be pre-existing low psychological and intellectual skills in the individual, and an unsupportive pre-trauma environment (i.e. family psychopathology, prior trauma and chronic environmental adversity).

Peri-trauma predictors appear to centre on the intensity of the trauma experience. The identified poor cognitive processing during the trauma may also link to the intensity of the

trauma as diminished cortical involvement is linked to perceived imminent threat to one's life (see paper 'An exploratory study into the development and maintenance of posttraumatic stress symptoms following large-scale disasters' by Dawson, 2007 for further elaboration).

Post-trauma predictors appear to be associated with the initial severity of avoidance, hyperarousal and distress symptoms, intrusions that are experienced as 're-living' phenomena, type of reasoning (e.g., negative, ex-consequencia), low levels of individual skills and abilities (e.g., lack of resilience, mastery and life satisfaction, physical injuries, inability to organise trauma narrative) and an unsupportive environment (e.g., additional post-trauma stressors, chronic system stress, lack of social support).

Of the three types of predictors, pre-trauma variables have been identified as having the weakest association with PTSD (see Schnurr, Lunney & Sengupta, 2004; Maes et al., 2001). This may be due to the pre-trauma predictors (low individual psychological and intellectual skills and an unsupportive environment) merely being predictive of critical post-trauma predictors (low individual skills and an unsupportive environment).

As already noted, the peri-trauma factors outlined in the available literature centre on the intensity of the traumatic event. This makes the identified peri-trauma factors descriptive rather than predictive, as the DSM-IV criteria for a diagnosis of PTSD is a life-threatening event involving powerful emotions of fear, helplessness or horror. To claim these as predictors presents a circular logic. Post-trauma predictors may therefore provide the most promise for understanding why some people develop PTSD and others return to pre-trauma functioning. With the exception of initial severity of avoidance, hyperarousal and distress symptoms and re-living type intrusions that are also descriptive of PTSD rather than predictive, the remainder of the identified post-trauma predictors offer additional information to the diagnostic criteria of PTSD.

In support of the importance of post-trauma variables in the development and maintenance of PTSD, Schnurr et al. (2004) found that whilst pre-trauma and peri-trauma factors contributed to the development of PTSD, that post-trauma factors were most predictive of current PTSD. Schnurr et al. (2004:93) states "the maintenance of PTSD was primarily associated with variables relating to the current time frame - current emotional sustenance, current structural social support, and recent life events". Maes et al. (2001) similarly reported that in a group of disaster and accident survivors' pre-trauma and peri-trauma stressors were unrelated to current PTSD course and severity, whereas post-trauma stressors were highly related to course and severity of PTSD.

Whilst the traumatic event itself may trigger initial PTSD symptoms, it appears that post-trauma factors underlie the maintenance of PTSD. Thus future PTSD research may benefit from focusing more on the impact of post-trauma variables than the traumatic event itself.

Similar to predictive factors, the identified protective factors (over page) also appear to suggest that what occurs after the traumatic event is important in determining whether a person will go on to develop PTSD or not. Positive social and emotional support in addition to personal resources such as resilience, purpose and the ability to cognitively process the event in a meaningful way in the aftermath of a trauma (perhaps enhanced by higher education) appear to act as a protective buffer against the development of PTSD. Although as the following research on brain imaging shows, the ability to cognitively process the traumatic event may be the result of not having PTSD rather than it being a protective buffer against PTSD.

Table 4. Protective Factors for PTSD

Protective Factors
Social/emotional support following trauma (Joseph, 1999; Ozer et al., 2003; Vranceanu, Hobfoll & Johnson, 2007)
Self-esteem and appraisal support following trauma (Hyman, Gold & Cott, 2003)
High sense of coherence (SOC). SOC refers to the ability to perceive a traumatic event as comprehensible, manageable and meaningful (Engelhard, vanden Hout & Vlaeyen, 2003).
Higher education associated with lower risk of PTSD (Schnurr, Lunney & Sengupta, 2004).
Higher intelligence quotient (IQ) was a predictor of resilience against PTSD in child and adolescent trauma survivors (Silva et al., 2000)
Personal hardiness and functional social support following trauma (King, King, Foy, Keane & Fairbank, 1999).
Secure attachments (Fraley, Fazzari, Bonanno & Dekel, 2006)

1.5.5 Relevant Brain Structures and Functional Changes in PTSD

Nancy Andreasen (2001:41) describes the human brain as a “living computer” that is “continually updating its software and keeping its systems running smoothly with a minimum of glitches and incompatibilities”. It appears that in PTSD individuals this ‘living computer’ is not able to keep its systems running smoothly. Brain functioning in PTSD individuals is marked by clear functional differences across brainstem, limbic and cortical regions of the brain when compared to non-PTSD individuals. They also show differences in their management of the body’s various systems when compared to non-PTSD individuals.

1.5.5.1 Brainstem Region

The brainstem region is involved in the automatic regulation of breathing, heartbeat, blood pressure, attention and the organisation of sensory information that guides movement (Carter, 2002). It includes structures such as the midbrain, pons, medulla and cerebellum. Subjects with PTSD exhibit increased functioning in this region during flashbacks of trauma memories when measured by regional cerebral blood flow in comparison to non-PTSD subjects (Osuch et al., 2001). Bonne et al. (2003) further demonstrated that PTSD subjects also exhibit an elevated cerebral blood flow in this region under non-threatening conditions. This suggests an increased baseline arousal of the autonomic nervous system.

1.5.5.2 Limbic Region

The limbic region includes structures such as the amygdala, hippocampus, caudate nucleus, hypothalamus, and thalamus, with the thalamus and hypothalamus also identified as 'between brain' structures due to their regulatory information relaying functions between the various brain regions (Andreasen, 2001). This brain region is considered to be a primitive brain region involved in emotions, emotional memory and the facilitation of an organism's response to the environment, including fear and anxiety-related behaviours (Kim & Gorman, 2005; Le Doux, 1998). In PTSD individuals, the limbic region exhibits increased functioning when compared to non-PTSD individuals (Liberzon et al., 1999). Following is a brief outline of how these structures function in PTSD compared to non-PTSD individuals.

The amygdala is a crucial brain structure involved in the detection of danger, learning about potential threats, recalling stressors and initiating survival responses (Le Doux, 1998; Knight, Nguyen, & Bandettini, 2005). In PTSD subjects, the amygdala exhibits an exaggerated response to both masked (Suslow, 2006) and overt fearful conditions (Armony, Corbo, Clement & Brunet, 2005). This suggests that subjects with PTSD have a heightened sensitivity to threatening stimuli both at an unconscious and a conscious level. The exception to this finding is PTSD subjects who show numbing rather than hyperarousal symptoms. During symptom provocation, Lanius et al. (2003) reported that when numbing rather than hyperarousal was evidenced no amygdala activation occurred.

In individuals with PTSD, the right putamen also shows increased functioning when experiencing flashbacks (Osuch et al., 2001). The right putamen is involved in unconscious

processes and controlling familiar skills such as riding a bike (Carter, 2002). This function is consistent with flashbacks experienced by a PTSD sufferer. During a flashback, physiological and behavioural responses familiar to the original trauma occur automatically (Briere, 2004).

A key structure in the limbic system is the hippocampus. This structure is known to be involved in contextual fear conditioning (Phillips & Le Doux, 1992). It is reported to unite contextual information with sensory information (auditory, tactile, olfactory) and to consolidate memory (Le Doux, 2002). The hippocampus is also involved in shutting down the hypothalamic-pituitary-adrenal (HPA) axis after a stressful psychological event is over (McEwan, 2003) and is susceptible to damage from chronic stress (Sala et al., 2004). In PTSD, changes are evidenced not just in the functioning of the hippocampus but in the brain structure itself. Neuroimaging studies have shown that in PTSD subjects, the hippocampal volume is significantly less than non-PTSD subjects (see Kitayama, Vaccarino, Kutner, Weiss & Bremner, 2005; Pavić et al., 2007 and Karl et al., 2006 for a meta analysis of hippocampal functioning in PTSD). Given the functions of the hippocampus, damage to this structure would impair context conditioning, memory processes and the ability to shut down the HPA axis, which are all consistent with PTSD phenomenology.

The finding of smaller hippocampal volume in PTSD subjects may be a result of pre-existing familial risk factors (Gilbertson, et al., 2002) and/or a result of ongoing chronic stress related to PTSD. The latter explanation is supported by studies that show that hippocampal volume is generally only smaller in chronic PTSD subjects and not in subjects following acute short-term trauma (e.g., Woodward et al., 2006). Although a recent study by Jatzko et al. (2006) on long-term changes in hippocampal volume (comparing chronic PTSD subjects to matched controls) suggests that hippocampal volume reduction may not be due to PTSD. The Jatzko (2006) study's exclusion criteria included alcohol addiction, a common comorbid condition with PTSD. When they controlled for alcoholism in PTSD, they found no significant differences in hippocampal volume between PTSD and non-PTSD subjects. As alcohol is increasingly consumed to manage distressing PTSD symptoms the disorder may be further consolidated through alcohol damaging the hippocampus.

In contrast to other limbic structures that generally show increased functioning, the thalamus displays significantly less activation when individuals with PTSD are recollecting traumatic and negative events compared to individuals who do not have PTSD (Lanius et al., 2003). Given that the thalamus is involved in regulating pre-attentive gating processes and is the principal structure

for relaying signals and sensory information to the cortex for conscious evaluation (Hazlett et al., 2001), significantly less activation suggests decreased pre-attentive gating (Lanius et al., 2003) and decreased information being relayed to the cortex (i.e., problems filtering out irrelevant information at pre-attentive levels and problems sending relevant information for conscious evaluation).

1.5.5.3 Cortical Region

The cortex is involved in higher executive processes such as thinking, planning and reasoning (Lee, 2006). A brief literature review on cortical functioning in PTSD suggests that these higher executive processes are impaired in PTSD subjects. For example, Chae et al. (2004) conducted EEG studies and identified diffuse cortical information processing disturbance in PTSD subjects. A growing body of research suggests that one cortical area in particular, the medial prefrontal cortex (mPFC), may be involved in the aetiology of PTSD. Correlational neuroimaging studies show that the mPFC has a negative covariation interaction with the amygdala, limiting the automaticity of fear responses (Das et al., 2005). The mPFC also contains the HPA response to stress (Jacobsen & Sapolsky, 1991) and has a critical role in higher order emotional evaluation processes, memory, and consciously planned responses to environmental stimuli rather than reactive responses (Dolcos, LaBar & Cabeza, 2004). Neuroimaging studies reveal reduced mPFC engagement in PTSD subjects compared to non-PTSD subjects during trauma-related cortical processing (e.g., Shin et al., 2004; Williams et al., 2006), non trauma-related cortical processing (e.g., Phan, Britton, Taylor, Fig, & Liberzon, 2006; Matsuo et al., 2003) and cortical processing of threat-related distracters that are not trauma-specific (Kim et al., 2007).

The Williams et al. (2006) study also found that in the context of reduced mPFC functioning in individuals with PTSD there was also an absence of coupling (i.e., the activity of one structure being linked to the activity of another) between the amygdala and the anterior cingulate cortex (ACC) over trials, as evidenced in non-PTSD subjects. Williams et al. (2006:355) conclude that this absence of coupling between amygdala and ACC activity over time in PTSD subjects highlights a “breakdown in the normal pattern of medial prefrontal and amygdala modulation”. These outlined differences in mPFC functioning suggest that in PTSD (compared to non-PTSD subjects), there is a reduction of higher order emotional evaluation processes and consciously planned behaviours, and a lack of cortical inhibition over amygdala driven fear and the HPA axis.

In general, the outlined differences in brain functioning between individuals who have PTSD and individuals who do not have PTSD suggest that PTSD is associated with increased functioning in limbic and brainstem regions, and decreased functioning in higher cortical regions both when the individual is at rest and undergoing symptom provocation. There also appears to be reduced communication and modulation between cortical and limbic structures in PTSD resulting in the domination of automatic responses over conscious responding.

Figure 1 (over page) provides a diagrammatic representation of the increased functioning in limbic and brainstem regions and decreased functioning in cortical regions in PTSD subjects compared to non-PTSD subjects.

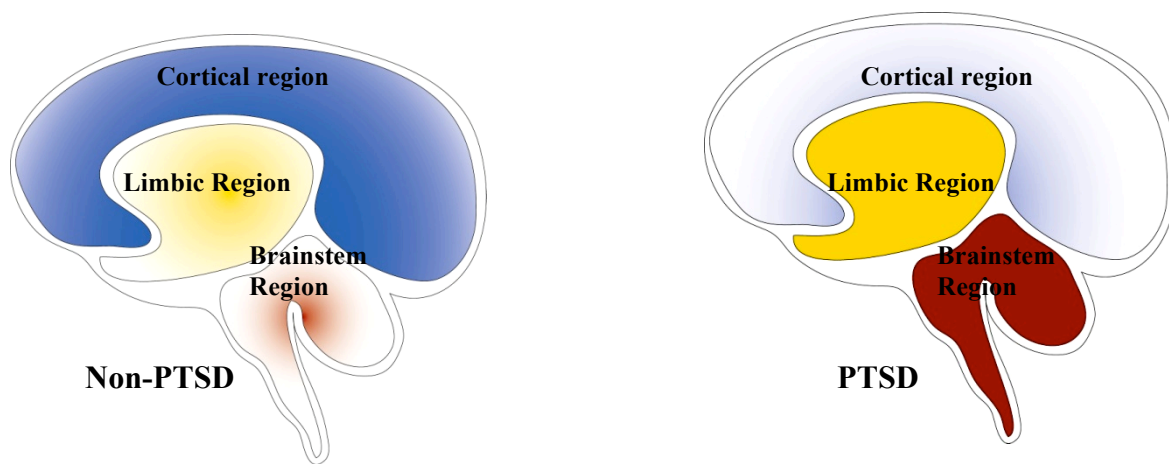


Figure 1. Diagrammatic representation of increased functioning in limbic and brainstem regions and decreased functioning in cortical regions in PTSD subjects compared to non-PTSD subjects. Solid colours represent increased functioning and lighter shaded areas represent decreased functioning.

The brain research outlined highlights the involvement of similar brain structures in PTSD to those identified by Le Doux (1998) as underlying fear mechanisms. The involvement of fear mechanisms in PTSD is not surprising as fear mechanisms facilitate an adaptive rapid response to a perceived threat so as to enhance the chance of survival. Le Doux (1998; 2002) demonstrated that fear mechanisms facilitate rapid survival responses through the relaying of sensory threat information via two neural routes, an indirect cortical route and a direct subcortical route (see diagram on opposite page). The fast direct subcortical route from the thalamus to the lateral amygdala sends crude information, is pre-attentive and triggers rapid defensive responding. In contrast, the slower indirect route sends more detailed threat information to higher order cortical

processing systems for conscious elaboration and evaluation of the threat (Le Doux, 1998; Adolphs, 2002).

Le Doux (1998) notes that the presence of these two neural routes has obvious survival value in that a fast primitive response to a dangerous stimulus via the subcortical route facilitates an immediate survival response whereas a slower cortical route allows for more detailed information about the threat for later evaluation and planning once out of immediate danger. Thus the rapid and slow systems work together to facilitate optimal short and long-term responses to threat. For example, if a fire is raging through a house, a rapid defensive response is required by occupants to avoid imminent death. However, once imminent death is avoided, the situation requires evaluation and planned action for either ongoing survival or adaptation to present circumstances. Research suggests that this latter conscious process is facilitated by the modulation of amygdala activity by the mPFC (Hariri, Mattay, Tessitore, Fera & Weinberger, 2003; Miller, Taber, Gabbard & Hurley, 2005; Quirk & Gehlert, 2003).

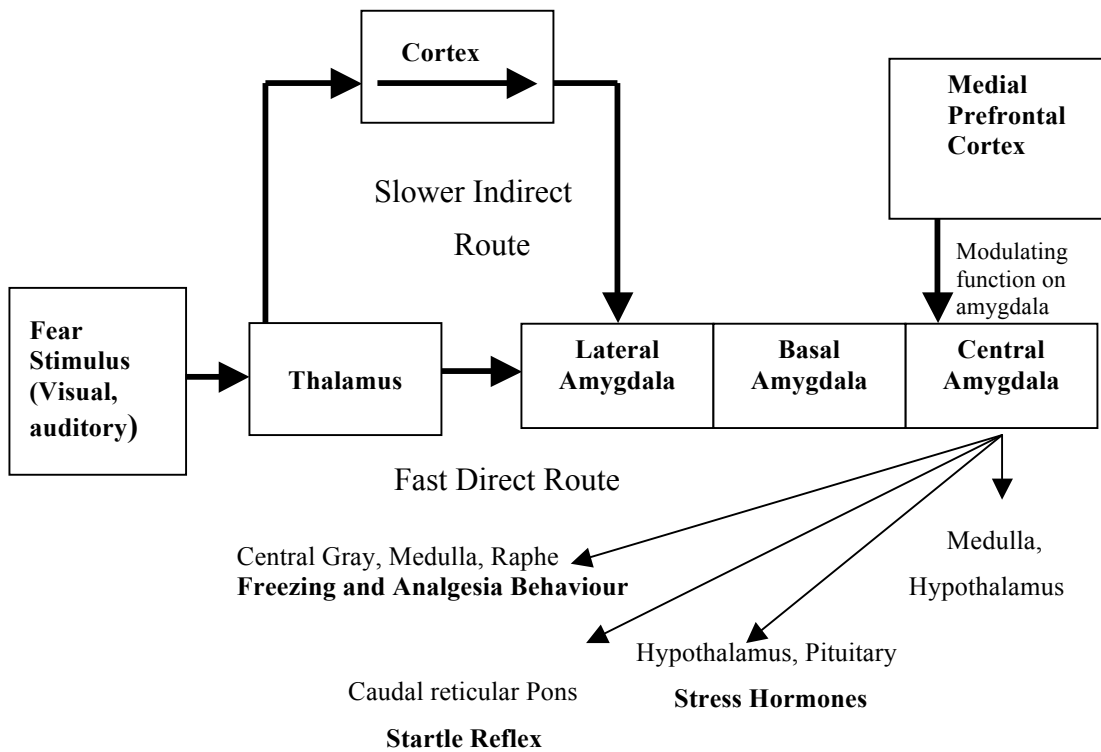


Figure 2. Information about an emotional stimulus is sent via a fast and a slow route to the amygdala.

1.5.6 Neurobiological Alterations in PTSD

As PTSD progresses, the disorder becomes increasingly marked by neurobiological alterations. Surprisingly, although PTSD has a high comorbidity with major depression and clearly involves a stress response, the underlying neurobiological alterations appear to be in the opposite direction to those seen in stress and depression. For example, psychosocial stress is known to increase cortisol levels (Gaab, Rohleder, Nater & Ehlert, 2005) but in PTSD basal cortisol levels are commonly found to be low (e.g., Rohleder, Joksimovic, Wolf & Kirschbaum, 2004; Neyla et al., 2005). Although it should be noted that cortisol lability (i.e., both high and low cortisol levels over time in the same individual) has also been shown in PTSD subjects (see Mason et al., 2002).

A further difference is that low glucocorticoid receptor levels mark major depression with an erosion of negative feedback whilst in PTSD there is an increase in glucocorticoid receptor sensitivity and stronger negative feedback inhibition (Yehuda, Golier, Yang & Tischler, 2004). Progressive sensitisation of the hypothalamic-pituitary-adrenal (HPA) axis in PTSD also stands in stark contrast to the progressive desensitisation of the HPA axis in depression and chronic stress (Yehuda, 1998). The behavioural outcome of these specific neurobiological changes in PTSD is that an individual with PTSD will become progressively more sensitive and responsive to lower levels of threat and environmental stimuli (Yehuda, 1998).

Consistent with a heightened responsiveness to environmental stimuli, PTSD subjects also exhibit higher noradrenergic activity under baseline conditions (Geraciotti et al., 2001), reduced pre-pulse inhibition (Grillon, Morgan, Southwick, Davis & Charney, 1996), diminished P50 sensory gating (Ghisolfi et al., 2004) and an exaggerated startle reflex (Grillon, Morgan, Davis & Southwick, 1998; Grillon & Morgan II, 1999; Shalev et al., 2000). Together, these neurobiological alterations reduce the filtering out of environmental information at a pre-attentive level and diminish the ability of higher brain regions to cognitively process and block threat information being sent throughout the body (see Mears, Klein & Cromwell, 2006 for function of P50 sensory gating and Braff, Geyer & Swerdlow, 2001 for functions of pre-pulse inhibition). These changes facilitate rapid automatic defensive responses to life-threatening stimuli (see Yeomans et al., 2002 for survival function of startle reflex and Morilak, Barrera, Echevarria, Garcia, Hernandez et al., 2005 for function of norepinephrine). The automaticity of responses in PTSD is consistent with research by Hendler et al. (2003) who demonstrated that combat veterans with PTSD displayed greater activation in sensory processing areas of the brain than

non-PTSD combats when trauma-related images were presented below conscious recognition threshold.

1.6 Theories of PTSD

As reflected by the research outlined in previous sections, the picture that has evolved in the research literature over the past 25 years is that PTSD is a complex psychological and neurobiological disorder. Early learning theories and cognitive behavioural theories of PTSD have similarly evolved over the years in an attempt to accommodate the expanding body of knowledge. At present there are three dominant overlapping theoretical viewpoints in the psychological literature. These current theories of PTSD have developed out of earlier theoretical views of PTSD such as those proposed by Horowitz (1986), Foa, Steketee and Rothbaum (1989), Litz and Keane (1989), Chemtob, Roitblat, Hamada, Carlson and Twentyman (1988), Janoff-Bulman (1985) and McFarlane (1989) and incorporate various combinations of fear conditioning principles, memory and information processing problems, and idiosyncratic cognitive distortions and schemas.

The three viewpoints overlap in that they all include aspects of memory and information processing to explain the aetiology of PTSD. They can be delineated from each other in simple terms as being either: emotional processing theories that focus primarily on strong emotional connections formed to elements of the trauma, information processing theories that focus primarily on how the trauma is processed and laid down in memory, or theories that focus primarily on cognitive factors. Biological theories are less common in the psychological literature but perhaps should be developed further given the identified neurobiological alterations. Following is a brief overview of three psychological theories that highlight the core elements of current theoretical viewpoints and a brief outline of biological accounts of PTSD.

1.6.1 Emotional Processing Theories

The core elements of current emotional processing theories (e.g., Foa, Huppert and Cahill (2006) have built on the original theoretical position of Foa and Kozak (1986) and Foa, Steketee and Rothbaum (1989). Foa et al. (1989) proposed that PTSD symptoms occur because of the strong emotional connections that are formed between behavioural responses, physiological responses and subjective meanings associated with the trauma. These emotional connections form a fear network that is readily re-activated by similar features of the trauma, particularly

environmental cues that represent assumptions about safety that were fractured at the time of the trauma. Symptoms persist because exposure to all the connecting elements of the trauma is not achieved.

Building on this model Foa and Rothbaum (1998) incorporated the idea that rigid beliefs and schemas that were present before, during and after the trauma also contribute to the development and maintenance of PTSD. For example, a person who rigidly believes the world to be dangerous, herself/himself incompetent and interprets their trauma symptoms and the reactions of other people negatively will potentiate and expand the fear network. Rigid beliefs and schemas have the additional effect of creating biased information processing of the traumatic event that leads to disorganised trauma narratives. Disorganised narratives are also developed and maintained by dissociation. Avoidance and numbing symptoms are seen as a strategy to avoid activating the fear network and the resulting re-experiencing symptoms.

The theory has indirect support for the existence of a fear structure in that therapeutic improvements have been shown to result from the activation of fear through exposure therapy (refer to Riggs, Cahill & Foa, 2006 for review of effective reduction of symptoms through prolonged exposure). There is also support for the view that dissociation creates disorganised and fragmented narratives (see Harvey & Bryant, 2002).

Treatment goals in this model of PTSD include activating the fear structures and providing corrective information to modify the connecting elements of the trauma so as to form a new, more realistic structure (Rauch, Foa, Furr & Filip, 2004). The intervention advocated to meet treatment goals is prolonged exposure/emotional flooding procedures or the same procedure combined with Cognitive Behaviour Therapy (outlined in next section).

1.6.2 Information Processing Theories

One of the more popular information processing theories is that offered by Brewin, Dagleish & Joseph (1996): the dual representation theory of PTSD. Brewin et al. (1996) proposes two separate types of memory systems, verbally accessible memories (VAM) and situationally accessible memories (SAM), to explain PTSD symptomatology. VAM are normal autobiographical memories that are retrieved deliberately through higher cognitive processes. They can be edited and are situated in a personal context of past, present and future. In contrast to VAM, SAM are image-based and are processed at a sub-cortical level.

According to this theory, PTSD results when memories from the SAM system are not processed sufficiently to enter the VAM system. They remain encoded as images, which the brain attempts unsuccessfully to transfer again to the VAM system. In this image format they are easily triggered in the brain and seen by individuals with PTSD as ‘flashbacks’ of the trauma. Avoidance from aversive flashbacks, along with the large discrepancies between the two memory systems, inhibits the SAM system from transferring its memories as it usually would, resulting in PTSD (Brewin, 2001). There is some indirect evidence for the presence of two memory systems as outlined in the dual representation theory (see Hellawell & Brewin, 2004; Holmes, Brewin & Hennessy, 2004; Lanius et al., 2005; Stuart, Holmes, & Brewin, 2006).

Treatment goals in this model of PTSD are to incorporate into the VAM system information provided by the SAM system during flashbacks, thus inhibiting the ability of trauma cues to trigger the SAM. Negative cognitions are also targeted through cognitive restructuring where necessary (Brewin, 2001). Similar to emotional processing accounts of PTSD, exposure therapy and cognitive behaviour therapy (outlined in next section) are used to achieve treatment goals.

1.6.3 Cognitive Behavioural Theories

Ehlers and Clark’s (2000) theory is one of the more comprehensive cognitive behavioural theories in the research literature, in that it attempts to explain the full range of PTSD symptoms. Similar to the emotional processing and information processing theories, Ehler and Clark (2000) see memory processes as a critical element of PTSD but also focus on the role of perceived current threat. They propose that idiosyncratic negative cognitions about the trauma and its sequela, plus trauma memory deficits (poor memory elaboration and inadequate memory integration), trigger a sense of current threat. This sense of current threat is then accompanied by arousal, anxiety, intrusions and other characteristic PTSD symptoms.

Due to incomplete cognitive processing during the trauma and a predominance of sensory memories associated with the trauma, intrusive memories are easily retrieved by trauma cues. In order to manage the intrusions, the sufferer then engages in avoidant behaviours and avoidant cognitive strategies (e.g., thought suppression and rumination). Thus avoidance maintains PTSD by preventing evaluation and subsequent alterations in negative cognitions. Later Ehlers et al. (2002) added a warning signal hypothesis to the original theory. The warning signal hypothesis proposes that intrusions represent memory fragments of the trauma experience that occurred just

prior to the moment of highest emotional impact. These memory fragments then intrude into memory as a warning signal.

Subsequent research has supported key elements of the Ehler & Clark (2000) theory. For example, Ehlers, Mayou and Bryant (2003) conducted a longitudinal study that tested the hypothesis derived from the Ehlers & Clark's (2000) theory that incomplete processing and negative appraisal thoughts lead to a sense of current threat and dysfunctional cognitive strategies. They found that in children that had experienced a car accident, variables testing incomplete processing and negative appraisal thoughts were correlated with PTSD severity. A more recent study by Ehring, Ehlers and Glucksman (2006) compared seven cognitive predictors from Ehlers & Clark's (2000) PTSD theory with predicted cognitions drawn from travel phobia and depression theories and found that each theory's predicted cognitions were correlated but distinct to the respective disorder. Studies by Halligan, Clark and Ehlers (2002), Halligan, Michael, Clark and Ehlers (2003), and Fairbrother & Rachman (2006) also support Ehler and Clark's (2000) assertion that negative cognitive appraisals of trauma and its sequela are correlated with PTSD symptoms.

Treatment goals within this framework are to reduce the sense of current threat through challenging negative cognitions and to promote the integration of trauma memory into a coherent narrative located in time and place through cognitive behaviour therapy (outlined in next section).

1.6.4 Biological Theories

Essentially, biological theories such as that proposed by Pitman (1989) and Elzinga and Bremner (2002) postulate that an excess of epinephrine is released at the time of the trauma and this in turn leads to (a) high levels of fear conditioning (i.e., a neutral stimulus becomes paired with an aversive event) and (b) an over consolidation of emotional memories (resulting in declarative memory deficits and the symptom clusters characteristic of PTSD).

Research within biological frameworks correspondingly focus on biological parameters such as D2 dopamine receptor (DRD2) genes (Lawford, et al., 2003), plasma levels of gamma amino-butyric acid (GABA) (Vaiva, et al., 2004), plasma neuropeptide Y (Yehuda, Brand & Yang, 2006), norepinephrine (Geraciotti et al., 2001) and physiological parameters of PTSD such as auditory startle (Elsesser, Sartory & Tackenberg, 2004, Guthrie & Bryant, 2005), heart rate (Veazey, et al., 2004; Zatzick, et al., 2005) blood pressure (Orr, Meyerhoff, Edwards & Pitman,

1998), cardiac vagal tone (Sack, Hopper & Lamprecht, 2004) and eye blink and skin conductance responses (Guthrie & Bryant, 2005).

Treatment goals within this framework usually focus on the prevention and treatment of PTSD primarily through pharmacological interventions such as propranolol (Pitman et al., 2002; Vaiva et al., 2003), mirtazapine (Won, Jeong-Ho, Tae-Youn, Won-Myong, 2005) reboxetine (Spivak et al., 2006) and paroxetine (Vermetten, Vythilingam, Southwick, Charney, & Bremner, 2003). To date research directed by biological theories has provided a wealth of understanding about the underlying neurobiological aspects of PTSD but an overall effective intervention has remained elusive.

1.7 Interventions for PTSD

There are a range of therapeutic interventions that have been promoted for the prevention and treatment of PTSD. However, for the purpose of this thesis, only those therapies with sufficient empirical research to evaluate their effectiveness are included. Currently, the dominant interventions applied clinically and researched empirically are: psychological debriefing procedures, prolonged exposure/emotional flooding procedures, eye movement desensitisation and reprocessing (EMDR), cognitive behavioural therapy (CBT) and pharmacotherapy.

1.7.1 Psychological Debriefing Following Trauma

Psychological debriefing is defined in broad terms by Kenardy (2000b: 1032) as being “a set of procedures including counselling and the giving of information aimed at preventing psychological morbidity and aiding recovery after a traumatic event”. These procedures are administered by either professionals or lay people trained in specific models such as critical incident debriefing. Debriefing procedures are commonly one-off interventions, 24-72 hours after a traumatic event (Raphael, 2000). Due to the debilitating nature of PTSD, psychological debriefing has been used extensively over the years in attempt to prevent its development and that of additional comorbid psychiatric disorders.

However, despite its wide use, psychological debriefing is currently a controversial practice. For although recipients of psychological debriefing have expressed satisfaction in the procedure (see Robinson, Sigman & Wilson, 1997), concerns have been expressed by various mental health professionals about its efficacy (see Kenardy, 2000a; Raphael, 2000; Bisson, 2003; Gist & Devilly, 2002). Central to their concerns are outcome studies that reveal that psychological

debriefing was ineffective in mitigating trauma symptoms and in some cases was even shown to be harmful. For example, a systematic review by Wessely, Rose and Bisson, (1999) on the outcomes of early debriefing procedures concluded that debriefing in the immediate aftermath of trauma was ineffective and even harmful in some cases. Studies conducted since this watershed review continue to suggest the ineffectiveness of its use and the potential for harm (see Small, Lumley, Donohue, Potter, & Waldenstrom, 2000; Mayou, Ehlers & Hobbs, 2000; Van Emmerik, Kamphuis, Hulsbosch & Emmelkamp, 2002; Rose, Bisson, Churchill & Wessely, 2002; Sijbrandij, Olf, Reitsma, Carlier & Gersons, 2006). A recent meta-analysis on preventative interventions by Cuijpers, Van Straten and Smit (2005) reported that in contrast to preventative interventions for depression and general anxiety disorders that reduce risks, the risk of developing PTSD was somewhat increased after debriefing procedures.

Despite the problems outlined above there is some evidence that psychological debriefing may be beneficial in organisational settings (see review by Arendt & Elklit, 2001) or if administered months after the event. A study by Chemtob, Tomas, Law & Cremniter (1997) that was conducted counter to general practice and administered 6 months after the traumatic event, evidenced a significant reduction in PTSD symptoms. At present the controversy surrounding the practice of psychological debriefing continues, as does its application in multiple settings following trauma.

1.7.2 Prolonged Exposure / Emotional Flooding

The rationale behind flooding is based on knowledge gained from fear conditioning procedures and learning theory. Repeated re-living of the trauma either through imaginal or in-vivo experiences is claimed to promote habituation to the feared stimulus. The subject is exposed to the feared stimulus (i.e. trauma memories or trauma cues) in the absence of danger (e.g., in the context of a supportive therapeutic relationship) and instructed to put the trauma into words at the same time. This procedure is carried out for an extended period of time until anxiety reduces, thus promoting extinction of the fear response (Saigh, Yule & Inamdar, 1996). Zoellner, Fitzgibbons and Foa (2001) claim that research supports the efficacy of prolonged exposure with PTSD and advocate its use as an effective treatment for PTSD. Subsequent research supports their faith in the procedure (see Rothbaum, Meadows, Resick & Foy, 2000). For example, a recent randomised clinical trial by Foa et al. (2005) comparing treatment outcomes (waitlist vs. exposure therapy vs. exposure therapy + cognitive treatment) found that both treatment groups

fared better than the waitlist group, but adding cognitive restructuring to prolonged exposure gave no additional therapeutic benefit.

Despite the reported benefits, van Minnen, Arntz & Keijsers (2002) report that 25 to 45% still meet diagnostic criteria for PTSD at the end of exposure treatment. Prolonged exposure is also contraindicated in some PTSD subjects due to possible harm. Pitman et al. (1991,1996) conducted imaginal prolonged exposure as advocated by emotional processing theories with Vietnam veterans. He found that prolonged exposure either had no therapeutic outcome (Pitman, 1996a) or adverse consequences such as heightened levels of guilt, shame, failure, panic and substance abuse in this PTSD population (Pitman, 1991). Schnyder (2005) warns of using exposure therapy with individuals who are severely disturbed by the traumatic experience due to a risk of increasing symptom levels. Caution has also been expressed by King (2002) regarding the use of exposure based treatments with PTSD individuals if head injuries and dysexecutive impairment are present, as perseveration of re-experiencing symptoms was found to result from its use in such a case. A recent study by Speckens et al. (2006) also suggests that exposure therapy is not the optimal treatment for all PTSD subjects. They report that subjects who experience a 'here and nowness' re-living of intrusions exhibit poorer outcomes with exposure techniques than PTSD subjects who don't have this type of intrusions.

Poor client compliance has also been reported to be a common problem with exposure therapy. For example, a study by Scott and Stradling (1997) reported that only 57% of subjects complied with exposure treatment. Interestingly, a study by Becker, Zayfert and Anderson (2004) on 217 licensed psychologists' attitudes to exposure therapy found that a significant number of those interviewed did not use exposure therapy for PTSD clients due to concerns over the clients' responses to the procedure. The finding of both client and clinician resistance to exposure therapy suggests that the distress levels triggered by the procedure may lead to avoidance in both client and therapist. Together the above suggests that exposure techniques are not always the treatment of choice and are effective only with certain populations.

1.7.3 Eye Movement Desensitisation and Reprocessing (EMDR)

EMDR is a procedure developed by Shapiro (1995) that, described simply, involves a PTSD subject tracking a stimulus with their eyes that is moving bilaterally, while at the same time holding an image in their mind of the traumatic event that triggered PTSD symptoms. The therapy was not developed from a theoretical framework but rather a random experience by the author. As a result of the lack of theoretical underpinnings, the therapy has been received

sceptically, with mixed reviews on its efficacy. There is some evidence that the procedure does indeed have a therapeutic benefit (see Chemtob, Tolin, van der Kolk, & Pitman, 2000).

However, Devilly (2002) in his review of EMDR concludes, whilst there is strong evidence that EMDR is better than no treatment, eye movements are a superfluous addition to the exposure techniques that also feature in EMDR therapy.

Similarly, a meta-analysis by Davidson & Parker (2001) found treatment benefits when compared to non-exposure treatments but no difference when compared to exposure therapies. A recent RCT study by Rauthbaum, Astlin, & Marsteller, (2005) confirmed this conclusion. An EMDR group was compared to a prolonged exposure group and a waitlist group. The EMDR treatment group (eye movement plus imaginal exposure) showed similar significant gains to the prolonged exposure group compared to the waitlist group, suggesting that the eye movements did not add any extra benefits.

1.7.4 Cognitive Behavioural Therapy

The major component of cognitive behaviour therapy (CBT) is cognitive restructuring of dysfunctional beliefs and schemas that trigger a fear response to trauma related cues. PTSD symptoms are reduced through helping clients gather as much information as possible about the fear-invoking object or event (Dietrich et al., 2000). The gathered information along with counterarguments is then used to correct and restructure related dysfunctional beliefs and schemas (Massad & Hulseley, 2006). The other main ingredient in CBT is imaginal or in vivo exposure (see Zoellner, Fitzgibbon & Foa, 2001 for outline of cognitive-behavioural treatments of PTSD).

At present there is convincing evidence for the efficacy of CBT in treating PTSD (see van Etten & Taylor's meta analysis, 1998; review by Harvey, Bryant & Tarrier, 2003; Taylor, 2006). However, a closer look suggests that even though CBT is better than no treatment or supportive counselling, its clinical efficacy appears to be limited to a sub population of individuals with PTSD. For example, a longitudinal study by Blanchard et al. (2004) found that there were some clients who showed only minimal improvement at the end of treatment and continued to be highly symptomatic throughout follow ups at one and two years post treatment.

As with prolonged exposure therapy, the drop out rate with CBT is commonly high (Tarrier, 2001; Hembree et al., 2003). A recent study by Zayfert et al. (2005) on dropout rates in clinical practice found that only 28% of people with a principal diagnosis of PTSD (n=115) completed

CBT treatment for PTSD. Ehlers, Clark, Hackman, McManus & Fennel (2005) also note that dropout rates in published studies on CBT range from 15-25% with a high proportion (35-47%) still meeting diagnostic criteria for PTSD at the end of treatment.

Ehlers and Clark (2000) have proposed a more refined version of CBT that appears to have overcome some of the problems leading to high subject drop out. Although similar to other CBT interventions, the key difference with the Ehlers and Clark (2000) approach is that exposure features much less in treatment. Exposure is only used to identify the points of peak emotional distress ('hot spots') in the trauma narrative that need elaboration and processing. Similar to other CBT approaches, this approach identifies idiosyncratic negative appraisals and avoidance strategies. It then uses behavioural experiments and cognitive restructuring to change the individuals negative appraisals and strategies into positive ones. A study by Ehlers et al. (2005) utilising this approach reported a drop out rate of only 3% in contrast to the majority of published CBT outcome studies.

It should be noted however, that the number of subjects in the Ehlers et al. (2005) study was small (1st trial n = 20 PTSD condition; 2nd trial n = 28: 14 PTSD condition, 14 waitlist condition). A further limitation of this study is that in line with most other clinical trials of CBT, individuals with drug and alcohol addictions, depression, history of psychosis and borderline personality disorder were excluded. Given the high comorbidity between PTSD and these disorders outlined previously, this limits any general claims of treatment efficacy.

1.7.5 Pharmacotherapy

Pharmacotherapy therapy for PTSD is still in its infancy and at present no specific drug has been developed for the disorder. Neither is there a well-established treatment protocol for the use of available pharmaceutical interventions in the prevention and treatment of PTSD. However, of the various drugs currently available it would appear that serotonin selective reuptake inhibitors (SSRI's) are the most efficacious in reducing PTSD symptoms. Stein, Ipser and Seedat (2006) reviewed 35 randomised controlled studies (n = 4,597) of PTSD subjects treated with SSRI's, placebo or other types of medication. They concluded that, despite gaps in the evidence base, SSRI's should be the treatment of choice in PTSD as the data suggest that SSRI's have some efficacy in reducing core symptoms of intrusion, avoidance and hyperarousal. An overview of recent PTSD literature by Cooper, Carty and Creamer (2005) and a comparative study of SSRI's vs. non-SSRI's in the treatment of PTSD by Asnis, Kohn, Henderson and Brown (2004) also

supports the use of SSRI's as the first drug of choice in the treatment of PTSD. However, even though SSRI's show promise as a treatment modality their application as a broad intervention is limited. Asnis et al. (2004:64) highlights this limitation stating, "50% of people taking SSRI's continue to have significant residual symptoms", thus leaving a large percentage of people still suffering the debilitating symptoms of PTSD.

1.7.6 Comment on Theories and Interventions for PTSD

There is evidence to support elements of each theoretical viewpoint. However, a possible weakness is that they all propose, in one form or another, that traumatic memories in PTSD are somehow qualitatively different to other types of memory. There is some evidence to suggest that this may not be the case. Studies undertaken both with war veterans (Geraerts et al., 2007) and civilians (Bernstein, Willert & Rubin, 2003) have found no qualitative differences in trauma memories. The Geraerts et al. (2007) study of 121 Croatian war veterans diagnosed with PTSD found that there was qualitatively no difference between neutral memories and traumatic memories. Subjects attributed similar amounts of visual, auditory, olfactory and bodily sensations to both types of memories.

Bernstein et al. (2003) looked at a different aspect of memory, the fragmentation of trauma memories between PTSD subjects and non-PTSD subjects. Their results revealed no significant difference between the two groups in the amount of fragmentation in their trauma memories. In contrast, Halligan, Michael, Clark & Ehlers (2003) did find an association between PTSD and trauma memory disorganization, but they found no evidence to support a relationship between recovery and more coherent trauma memories. Based on the premise that vivid trauma memories underlie PTSD symptoms, Rauch, Foa, Furr & Filip (2004) measured treatment outcomes after modifying the vividness of trauma memories. Contrary to their hypothesis they reported that modifying trauma vividness was unrelated to treatment outcomes. An event-related functional magnetic resonance imaging (fMRI) study also lends support to the possibility that vivid trauma memories do not underlie PTSD. Dolcos, LaBar and Cabeza (2004) demonstrated that successful encoding of both positive and negative pictures was equally enhanced by emotion compared to neutral pictures. Together these studies suggest that the trauma memory itself may not play a key role in PTSD pathology. This raises potential problems for the dominant psychological theories of PTSD.

Disorganised and/or fragmented narratives due to incomplete processing also feature in the dominant psychological theories. If these factors underlie PTSD, then a logical assumption is

that changes in the trauma narrative (i.e. if they become organised and coherent) will reduce symptoms. However, empirical evidence suggests that changes in disorganised trauma narratives are not associated with a reduction of symptoms as current theories predict (see Jones, Harvey & Brewin, 2007, Gidron et al., 2007). A recent study by Briere, Scott & Weathers (2005) on dissociation also causes problems for emotional processing and information processing theories. They reported that dissociation at the time of trauma, hypothesised by emotional and information processing theories to contribute to the disruption of information encoding at the time of the trauma, did not predict PTSD.

A further problem with the above theories is that they are based on PTSD being a discrete clinical syndrome with a discrete causal origin. However, taxometric studies provide evidence that this is not the case. Recently, Broman-Fulks et al. (2006) conducted a study utilising three mathematically independent taxometric procedures to investigate the latent structure of PTSD. They found that across two large nationally represented samples of women ($n = 2684$ and 3033), PTSD was not a discrete clinical syndrome but rather the extreme end of a continuum of posttraumatic stress responses. This finding replicates previous taxometric studies by Ruscio, Ruscio and Keane (2002) and Forbes, Haslam, Williams and Creamer (2005).

Ruscio et al. (2002) found that the latent structure of PTSD was of a dimensional nature rather than a discrete clinical disorder in their taxometric study of 1230 male combat veterans. Similarly Forbes et al. (2005) reported that their taxometric study of 692 Australian combat veterans, randomly selected from community samples, favoured a dimensional rather than a categorical model of PTSD. The importance of these findings is that PTSD is unlikely to have a discrete etiological origin as suggested by current theories, but rather multiple causal determinants, that in an additive manner place a person further along a posttraumatic stress continuum (Broman-Fulks et al., 2006; Forbes et al., 2005).

Neurobiological evidence also reveals that regions of the brain involved in pre-attentive automatic survival processes drive large portions of PTSD symptomatology. The neurobiological data outlined previously suggests that in PTSD, the fast (subcortical) and slow (cortical) threat processing and responding breaks down, with pre-attentive subcortical processes continuing to dominate over cortical processes due to reduced engagement of the prefrontal cortex (PFC). Of relevance is research that suggests that the PFC is taken “off-line” or reduced in functioning when under threat so that sympathoexcitatory circuits are disinhibited and energy reserves

released for the domination of automatic survival enhancing defensive responses (Arnstein & Goldman-Rakic, 1998; Thayer & Brosschot, 2005).

Taken at face value, the changes in brain functioning and associated neurobiological alterations present a picture of PTSD as the outcome of a 'living computer' that has updated its software after a life-threatening experience to enhance survival in a dangerous environment. Fear-driven automatic survival responses thus remain operational, leaving the individual biologically 'wired' to rapidly detect and respond to any additional life-threats in the post-trauma environment. In such a context, cortical engagement may diminish the chances of survival as cortical engagement facilitates slower cognitively determined responses over rapid automatic defensive responses. Within this framework, working memory and cognitive dysfunction evidenced in PTSD is a secondary effect of enhanced survival functions, rather than the primary cause of PTSD as proposed by current theories.

Several theorists have similarly proposed that PTSD may be a result of fear driven survival responses. Derek Silove (1998) suggested that PTSD may be an over-learned survival response, Eberly, Harkness & Engdahl (1991) proposed that the core symptoms of PTSD are all adaptive evolutionary behaviours to extreme threats. Chemtob et al. (1988) proposed that PTSD results from a low threat threshold activating biologically predisposed survival modes and more recently, Chris Cantor (2005) proposed that PTSD is a maladaptive use of adaptive evolutionary defensive responses. Aside from these few sources, the majority of PTSD theorists do not view PTSD as an adaptive survival response to a dangerous environment but a disorder of memory and information processing. This emphasis on the manner in which the trauma memory is laid down at the time of the trauma and subsequently processed and integrated fails to address the involvement of pre-attentive automatic survival processes and the prominent role of post-trauma environmental variables.

1.8 Summary and future directions in PTSD research.

In summary, PTSD is clearly a complex disorder that progressively impacts a person's functioning across all aspects of their life. Deleterious effects are evident in social, emotional, biological and psychological domains as the posttraumatic symptoms become more chronic. It appears to particularly impact vulnerable groups, such as children and refugees, making this an important focus area for research.

Contemporary psychological theories and relevant psychological data suggest that PTSD is a disorder of memory, information processing and dysfunctional cognitions. Whilst there is partial evidentiary support for these claims, a weakness in this approach to PTSD is the strong emphasis on trauma memories being qualitatively different to other forms of memory as there is evidence to the contrary. Evidence also suggests that changes in trauma narratives do not reduce symptoms (as would be expected if incomplete processing contributed to PTSD) and that cognitive therapies that target dysfunctional cognitions appear to only work for a subset of PTSD sufferers. Taxometric studies also suggest that PTSD is not a discrete clinical syndrome thus challenging the assumptions underlying the dominant psychological theories.

Certain therapies developed from the outlined theories, exposure therapy and cognitive behavioural therapy, have been shown to be an effective treatment for PTSD. However, the high dropout rate associated with these therapies, only partial remissions of symptoms and exclusion criteria for clinical trials means that the efficacy of the treatments is limited. It is not clear at present why these therapies do not meet the needs of more individuals who suffer from PTSD. Nor is it clear why early interventions for PTSD are ineffective and potentially harmful.

Current theories of PTSD and the interventions that these theories propose are limited in that they are trauma-centric and person-centric. They tend to overlook the clear role of post-trauma variables that encompass social and environmental factors in addition to individual factors. Based on the available psychological and neurobiological data, PTSD presents more as a functional survival response that is maintained or even enhanced over time by post-trauma variables. Research that explores this possibility may elucidate the critical determinants that underlie the development and maintenance of posttraumatic symptom clusters in PTSD and the development of safer, more effective interventions for the disorder.

1.9 Aims of Thesis

The broad aims of this thesis are to elucidate the critical determinants that underlie the development and maintenance of posttraumatic symptom clusters in PTSD and to outline a trauma theory with a conceptual framework for the future development of safe and effective interventions. In order to achieve these aims, factors that maintain the core PTSD symptom clusters of intrusion, avoidance and hyperarousal are investigated both cross-species and cross-culturally. Based on the possibility suggested by the neurological data that PTSD involves

survival mechanisms, an ecological animal model of survival is used as a framework for identifying factors to be investigated.

The major aim of the first experimental paper comprising this thesis is to validate that PTSD exists in African trauma populations (in which future cross-cultural studies are to be conducted). This aim is addressed through qualitative interviews with African people-helpers who experienced or witnessed recent horrific war atrocities carried out in their communities.

The aim of the second experimental paper is to validate a posttraumatic symptom measurement scale, the Impact of Events Scale Revised (IES-R), for use in Sri Lanka. This aim is addressed by:

- (1) Translating the validated English version of the IES-R to be used with Africans into the local Sri Lankan dialect (Tamil and Sinhala).
- (2) Validating the psychometric properties of the Sri Lankan version of the IES-R (IES-R-SL) in a large sample of Sri Lankan adults exposed to the 2004 tsunami.

The aim of the third experimental paper is to conduct an exploratory study into possible maintaining and mitigating factors of posttraumatic intrusion, hyperarousal and avoidance symptom clusters in trauma survivors from large-scale disasters (natural and man-made). This aim is addressed through conducting a correlational study between self-report measures of intrusion, hyperarousal and avoidance symptoms with variables derived from Kavaliers and Choleris's (2001) animal predator-prey model of survival in a population exposed to war atrocities (Africa) and a population exposed to a tsunami (Sri Lanka).

The aim of the fourth experimental paper is to explore the role of the environment in the maintenance of PTSD. Specifically, to explore whether geographical proximity of current residence to an earlier large-scale disaster site modulates posttraumatic avoidance, hyperarousal and intrusion symptoms. This aim is addressed by comparing IES-R-SL scores of Sri Lankan people-helpers exposed to the 2004 Asian tsunami whose permanent residence is in one of two geographical locations (one close to where the tsunami struck and one further away). IES-R-SL scores were collected at three weeks post-tsunami and six months post-tsunami.

The aim of the fifth experimental paper is to explore the possibility raised in the fourth experimental paper that the environment and discrete trauma cues interact to produce ongoing fear-related symptoms in PTSD. As experimentally manipulating these variables in humans is considered to be problematic, this aim is addressed by conducting two fear-conditioning

experiments in rats. Context/cue interactions are explored using variations of forward and backward fear conditioning procedures (i.e. pairings of a brief tone and shock in a chamber).

The aim of the sixth experiment paper is to extend the findings of the fourth experimental paper and the fifth experimental paper on the modulating role of the environment on posttraumatic avoidance, hyperarousal and intrusion symptoms. Specifically, whether (a) currently living in a dangerous environment maintains high levels of posttraumatic symptoms (b) whether a failure to identify a local context of safety following a traumatic event is associated with a global excitatory context (i.e. a perception of threat across contexts) and (c) whether a global excitatory context is positively correlated with posttraumatic symptoms. These aims are addressed by administering a modified version of the IES-R and a questionnaire rating subjective levels of threat and safety across contexts (local to global) to trauma survivors who continue to live within a dangerous context (a war zone with the constant threat of gunfire, abduction by rebel armies and/or rape) and exploring the correlations between the scores obtained.

Based on the results from these six experiments and relevant literature, a trauma theory is then outlined with suggestions for future research and safe intervention practices.

1.10 Methodological Considerations: Cross Cultural and Cross Species Research

There are two main methodological considerations that arise from the proposed research:

(i) Can PTSD be researched cross culturally given that it was historically operationalised in a Western context?

Even though PTSD was operationalised in a Western context, PTSD has been identified across vastly diverse cultures. For example, PTSD has been diagnosed in American Indians (Beals et al., 2005) Guatemalan refugees (Sabian, Cardozo, Nackerud, Kaiser & Varese, 2003), Kalahari bushman (McCall & Resick, 2003), Vietnamese refugees (Hinton, Safren & Pollack, 2006), a Kenyan refugee community (Kamau, et al., 2004), Indian women (Mehta, Vankar & Patel, 2005) and Russian youth (Ruchkin, Schwab-Stone, Jones, Cicchetti, Koposov & Vermeiren, 2005). In fact Marsella, Friedman, Gerrity and Scurfield (1996) stated in their review of cross-cultural PTSD that they could not find an ethnocultural group in which PTSD could not be diagnosed. Further, as the focus of the study involves indirectly investigating universal survival mechanisms, it is critical to the study to include cross-cultural trauma groups.

An additional benefit of researching outside of a Western context is that, sadly, large numbers of people within other cultural groups experience similar trauma and receive little intervention, thus providing a more homogenous trauma sample than found in Western populations. It is acknowledged that aspects of a traumatic event and traumatic responses may be culture-specific and may carry different subjective meanings and expressions. This issue is addressed in part through consulting with local community leaders at each stage of the experiments conducted so that relevant information can be gained and the risk of cultural ignorance and insensitivity minimised.

(ii) Can meaningful PTSD research be conducted cross-species?

Although there are species-specific responses to a life-threatening event, both adaptive and maladaptive survival mechanisms are essentially the same across all mammalian species (Rosen & Schulkin, 1998). Neurological and neuroimaging studies have shown that fear-related emotional patterns in humans activate the same adaptational defence mechanisms as in other mammals (Lang, Davis & Öhman, 2000). Similarly, Blanchard, Hyne, Minke, Minemoto & Blanchard (2001) identified that human defensive behaviours to threat scenarios showed parallels to fear-related and anxiety-related defense patterns of non-human mammals. Phelps, Delgado, Nearing and Le Doux (2004) have also identified that similar brains regions and mechanisms are involved in fear extinction in both humans and animals. These similarities between humans and other mammals are largely responsible for our present understanding of the brain structures and neurological pathways involved in human defensive responses to threat (Le Doux, 1998).

If PTSD is underpinned by cross-species survival mechanisms as suggested by the neurological data, then principles gained from the proposed animal studies may be able to provide critical information as a guide for future human research. Whilst the more complex cognitive components of PTSD obviously cannot be replicated in animals, fear and fear extinction processes such as that investigated in this thesis can be. In order to gain new information about the maintenance of fear-related mechanisms it is necessary to (i) experimentally manipulate a threat context and then (ii) record the resulting behavioural response to both the context and threat cue in an experimentally controlled manner. The manipulation and collection of such data is essential to understanding fear extinction processes in PTSD and this can only be obtained with the use of experimental animals, such as rats.

1.11 References

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