EXPOSURE IN THE TREATMENT OF POSTTRAUMATIC STRESS DISORDER

Johanna Christina Theron

Dissertation presented for the degree of Doctor of Psychology at Stellenbosch University

Promotor: Prof A.T. Möller

Co-Promotor: Prof B.B. Van Heerden

December 2007

DECLARATION

I, the undersigned, hereby declare that the work contained in this dissertation is my own original work and that I have not previously in its entirety or in part submitted it at any university for a degree.

Signature:

Date: 2007-11-01

ABSTRACT

A review of the literature revealed that several well-controlled outcome studies found prolonged exposure effective in the treatment of posttraumatic stress disorder (PTSD). However, all these studies were based on either American or British samples. The present study, therefore, investigated the effectiveness of exposure treatment for PTSD in a South African sample of female survivors of sexual violence. Fifteen patients participated in manualized exposure treatment, consisting of nine sessions of 90 minutes each, while 14 patients served as delayed treatment controls. Results showed that prolonged exposure significantly reduced all the PTSD symptom clusters (re-experiencing, avoidance, and arousal) from pretreatment to post-treatment, and that this improvement was maintained at follow-up after three months. At the end of treatment no treated patient met the diagnostic criteria for PTSD, as assessed by an independent, blind evaluator by means of the Clinician Administered PTSD Scale. The untreated controls all retained the diagnosis of PTSD. The results also showed a gradual reduction in posttraumatic stress symptoms and the associated symptoms of depression, anxiety and dysfunctional cognitions from sessions two to four, again from four to six, and again from sessions six to eight. In addition, based on four case studies, there were indications that prolonged exposure treatment also facilitated a reduction in regional cerebral blood flow (rCBF) bilaterally in the superior and mid frontal regions, as well as mixed bilateral changes in perfusion in the cerebellum and parieto-occipital regions.

OPSOMMING

Uit 'n oorsig van die literatuur blyk dit dat verskeie goed gekontroleerde uitkomsstudies aangetoon het dat langdurige blootstelling 'n effektiewe behandelingsprosedure vir posttraumatiese stresversteuring (PTSV) is. Omdat al hierdie studies egter op Amerikaanse of Britse steekproewe gebaseer was, het die huidige studie ten doel gehad om die effektiwiteit van langdurige blootstelling in 'n Suid-Afrikaanse steekproef van vroulike slagoffers van PTSV as gevolg van seksuele geweld te ondersoek. Vyftien pasiënte het deelgeneem aan 'n handleidingsgebaseerde blootstellingsprogram bestaande uit nege sessies van 90 minute elk, terwyl 14 pasiënte in 'n vertraagde behandeling kontrolegroep ingesluit is. Die resultate het aangetoon dat langdurige blootstelling al die PTSV simptoomgroepe beduidend van voor tot na behandeling verminder het, en dat hierdie verbetering gehandhaaf is by opvolg drie maande na behandeling. Aan die einde van die behandeling het geen van die behandelde pasiënte meer aan die diagnostiese kriteria vir PTSV voldoen nie, terwyl die pasiënte in die kontrolegroep almal die diagnose behou het. Die resultate het ook aangetoon dat daar 'n geleidelike verbetering in posttraumatiese stressimptome sowel as die geassosieerde simptome van depressie, angs en disfunksionele kognisies vanaf sessies twee na vier, weer van vier na ses, en weer vanaf sessies ses na agt plaasgevind het. Hierbenewens het dit ook op grond van vier gevallestudies geblyk dat langdurige blootstelling 'n vermindering in serebrale streekbloedvloei bilateraal in die superior en mid-frontale areas, sowel as gemengde bilaterale veranderinge in perfusie in die serebellum en pariëto-oksipitale areas gefasiliteer het.

ACKNOWLEDGEMENTS

I would like to express my sincere appreciation to the following persons:

- Prof A.T. Möller, my promotor, for his excellent guidance and continuous support.
- Prof B.B. van Heerden, my co-promotor, for his continuous guidance, interest and support.
- Dr J. Warwick for his assistance with the SPECT scans throughout the study.
- Mr H. Steele for his assistance with the statistical analysis.
- Dr P.D. Carey for his assistance with the analysis and interpretation of the SPECT results.
- The doctors and nursing staff at various community hospitals for their help with the identification of potential participants.
- The SAPS for their assistance in identifying potential participants.
- The staff from Rape Crisis and Mosaïk for their support and help in the identification of potential participants.
- Dr M.F. Gerber and my colleagues at Stikland Hospital and Tygerberg Hospital for their continuous support and encouragement.
- Mrs T. van Greunen for the pre-, post-, and follow-up evaluations of the potential participants.
- My mother, Elma, for her time and effort with the typing of the dissertation and for her consistent support and encouragement.
- My brother, Jan, for his support and encouragement.
- My friends for their interest and support.
- My Heavenly Father, without whose strength this dissertation would not have been possible.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

1. INTRODUCTION AND OBJECTIVES

1.1 POSTTRAUMATIC STRESS DISORDER

The concept of a trauma-related emotional disturbance has existed for over a century. A condition resembling posttraumatic stress disorder (PTSD) was first described among veterans of the American Civil War by a Philadelphia physician Jacob Mendez Da Costa (Vaisrub, 1975). It was then referred to as "soldier's heart" due to the presence of autonomic cardiac symptoms. During World War I the syndrome was called "shell shock", and was hypothesized to result from brain trauma due to the explosion of mortar shells. This syndrome was also seen during and after World War II, particularly in the survivors of Nazi concentration camps as well as in survivors of the Hiroshima and Nagasaki atomic bombings in Japan. At this stage the syndrome was referred to as "operational fatigue" or "combat neurosis".

However, the relatively high prevelance of this condition among veterans of the Vietnam War served as the important impetus for the burgeoning of PTSD research over the last decades (Nemeroff et al., 2006). This contributed to the recognition of PTSD as a formal disorder, and its official categorization in the diagnostic nomenclature occured in 1980, when it was first introduced in the third edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM III; APA,1980).

Official recognition of PTSD as a formal disorder resulted in intensive clinical and scientific interest in this syndrome. For example, in 1989 the National Centre for Posttraumatic Stress Disorder was established in the United States of America in response to a congressional mandate to address the needs of combat veterans with PTSD. Since then the Centre has initiated and carried out numerous research projects on PTSD, including studies on assessment, PTSD symptoms such as numbing (Litz, 1992) and attention and memory (Litz et al., 1996), risk factors (Schnurr, Rosenberg, & Friedman, 1993), and the etiology of PTSD (King, King, Foy, & Gudanowski, 1996). Similarly, an international interest in PTSD stimulated a congruence of disparate areas of research on different types of trauma, resulting in rapid developments in theory and treatment (Foa & Meadows, 1997; Friedman & Rosenheck, 1996; Schnurr & Friedman, 1999).

The DSM-IV-TR (APA, 2000) describes PTSD as a set of typical symptoms following a traumatic stressor. A traumatic stressor is an essential prerequisite for a diagnosis of PTSD, and entails that an individual must have witnessed, experienced, or otherwise have been confronted with an event that involved actual or possible death, grave injury, or threat to physical integrity of self or others. In addition, the individual's response to the event must include severe helplessness, fear or horror. Apart from a traumatic stressor, the DSM-IV-TR (APA, 2000) lists 17 symptoms which are organized into three symptom clusters. The re-experiencing cluster consists of symptoms such as recurrent and intrusive distressing recollections of the event (including images, thoughts, or perceptions), recurent distressing dreams of the event, acting or feeling as if the traumatic event were recurring, and intense psychological distress and physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event. The second cluster of symptoms, referred to as the avoidance of stimuli associated with the trauma and numbing of general resposiveness, includes efforts to avoid thoughts, feelings, or conversations associated with the trauma, and avoidance of activities, places or people, that arouse recollections of the trauma, inability to recall important aspects of the trauma, diminished interest or participation in meaningful activities, feelings of detachment or estrangement from others, a restricted range of affect, and a sense of a foreshortened future. The third symptom cluster, increased arousal, refers to difficulties in falling or staying asleep, irritability or outbursts of anger, difficulty in concentrating, hypervigilance, and an exaggerated startle response. For a diagnosis of PTSD to be made, these symptoms must be present for at least one month following the traumatic event. In addition, PTSD is associated with significant distress or impairment in social, occupational or other important areas of functioning.

Reactions to major trauma may vary extensively, depending on the victim's individual characteristics and circumstances, the intensity and severity of the specific trauma, as well as the duration of the trauma exposure. Foa, Stein, and McFarlane (2006) stated that victims of disaster, where homes and livelihoods were destroyed, may present a response pattern different from that of victims of more individual and personal traumas. Similarly, PTSD may not be the only response to trauma. For example Norris, Friedman, and Watson (2002) reviewed studies of disaster victims and identified six categories of outcomes following major trauma. These include specific psychological disorders (such as depression, anxiety, or PTSD), nonspecific distress, health problems, chronic problems in living, resource loss, and unique problems specific to young victims. The most commonly observed disorder,

PTSD, was identified in 68% of the studies, while major depressive disorder was identified in 36% of the studies, and anxiety (including generalized anxiety disorder and panic disorder) in 20%.
Several studies indicated that the development of PTSD may depend on specific trauma characteristics such as intensity and duration of the trauma (Yehuda & McFarlane, 1995). Different prevelance rates are therefore reported for different trauma populations (McFarlane, 2002). For example, several studies reported prevelance rates of 70% and above for PTSD after rape and sexual violence (McFarlane, 2002), while a prevelance rate of 43% following motor vehicle accidents is reported (Coffey, Gudmundsdottir, Beck, Palyo, & Miller, 2006). Surveys of the general USA population indicated that PTSD affects about one in every 12 adults at some stage. This is about 15% to 24% of those exposed to some kind of traumatic event (Breslau, Davis, Andreski, & Peterson, 1991; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Although exposure to trauma is lower in women than in men, a 2:1 ratio of female to male prevalence for PTSD is typically reported (Breslau, 2001a).

Symptoms of PTSD developing within four weeks after a trauma represent an acute stress response, which is considered a normal response to stress. The majority of patients showing these symptoms do not subsequently develop any psychological disorders. The symptoms of PTSD therefore do not commence immediately after the trauma, but rather represents a lack of resolution of the acute stress response to the trauma. Although PTSD eventually resolves without treatment in approximately 60% of cases (McFarlane, 2000), some individuals go on to chronic, unremitting PTSD (Foa et al., 2006).

Not all individuals exposed to trauma develop PTSD (Foa et al., 1999; Foa, Hearst-Ikeda, & Perry, 1995; Foa, Rothbaum, & Furr, 2002; Kaplan & Sadock, 1998). This led researchers to focus on risk factors for the development of PTSD, as well as possible protective factors that may inhibit its development. However, there is at present no concensus on possible risk factors that may contribute to the development and maintenance of PTSD.

1.2 PSYCHOLOGICAL TREATMENT OF POSTTRAUMATIC STRESS DISORDER

Even before the official recognition of PTSD as a formal disorder, trauma-related emotional distress was treated by means of psychological procedures such as systematic desensitization (Schindler, 1980; Wolff, 1977) and psychodynamic therapy (Fox & Scherl, 1972). A review of the literature showed that a broad spectrum of psychotherapeutic approaches and techniques are currently used for treating

PTSD. These include psychodynamic therapy (e.g., Lindy, Green, Grace, & Tichener, 1983; Scarvalone, Cloitre, & Difede, 1995), group therapy (e.g., Najavits & Garber, 1989; Zaidi, 1994), hypnotherapy (e.g., Jiranek,1993; Peebles, 1989), eye movement desensitization and reprocessing (e.g., Shapiro, 1989, 1995), as well as various cognitive-behavioural procedures. The latter include cognitive restructuring (e.g., Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998), cognitive processing therapy (e.g., Resick & Schnicke, 1992), stress inoculation training (e.g., Kilpatrick, Veronen, & Resick, 1982; Veronen & Kilpatrick, 1982), systematic desensitization (e.g., Brom, Kleber, & Defares, 1989) and prolonged exposure (e.g., Cooper & Clum, 1989; Foa et al., 1999; Taylor et al., 2003).

Relatively few treatment outcome studies for PTSD from a psychodynamic perspective have been reported. Apart from yielding conflicting results on the effectiveness of this approach for the treatment of PTSD, most of these studies showed methodological shortcomings. The same applies for hypnotherapy as a treatment for PTSD. Despite a number of case reports, only one controlled study on the effectiveness of hypnotherapy for PTSD was published (Brom et al., 1989). More outcome studies on the effectiveness of eye movement desensitization and reprocessing in the treatment of PTSD are available. Although positive results were generally reported, this procedure cannot at this stage be conclusively credited with proven efficacy in the treatment of PTSD.

The most studied psychosocial treatments for PTSD are the cognitive-behavioural interventions (Foa & Meadows, 1997). From a cognitive-behavioural perspective, cognitive restructuring as a treatment procedure for PTSD was investigated in only a few studies (Marks et al., 1998; Rieckert & Möller, 2000). Despite positive results, more outcome studies as well as comparative studies are needed. The same applies to cognitive processing therapy as only a few outcome studies (Resick & Schnicke, 1992; Resick, Nishith, & Astin, 1998), characterised by methodological limitations, are available. Only one controlled study investigated the effectiveness of systematic desensitization in the treatment of PTSD (Brom et al., 1989), comparing it to psychodynamic therapy and hypnotherapy. No significant differences in treatment effect between the treatments were found. Several well controlled studies (Foa, Riggs, & Gershuny, 1995; Foa, Rothbaum, Riggs, & Murdock, 1991; Resick, Jordan, Girelli, Hutter, & Marhoefer-Dvorak, 1988; Veronen & Kilpatrick, 1982) provided support for stress inoculation training as a treatment for PTSD. It produced significant improvement on all three clusters of PTSD symptoms and these improvements were maintained at follow-up.

A review of outcome studies on exposure treatment for PTSD showed that since 1989 about 11 well controlled studies were published (Boudewyns & Hyer, 1990; Boudewyns, Hyer, Woods, Harrison, & McCraine, 1990; Cooper & Clum, 1989; Foa et al., 1991; Foa et al., 1995; Foa et al., 1999; Keane, Fairbank, Caddell, & Zimering, 1989; Marks et al., 1998; Richards, Lovell, & Marks, 1994; Taylor et al., 2003; Thompson, Charlton, Kerry, Lee, & Turner, 1995).

These studies investigated the effectiveness of exposure treatment for PTSD resulting from a broad spectrum of traumas, including sexual assault, criminal violence, motor vehicle accidents, etc. The results of these studies showed that the effectiveness of exposure treatment does not depend on the type of trauma preceding PTSD.

Inspection of the available outcome studies revealed large variations in design. Only a few studies compared exposure treatment to a control condition (either a waiting-list control or a standard psychosocial treatment or counselling). Most studies compared prolonged exposure to other cognitive-behaviuoral procedures such as stress inoculation training, cognitive restructuring, relaxation training, or combinations of these treatments.

In general, these studies are methodologically sound and to a large extent met the accepted methodological criteria for outcome studies. The most common exceptions were the absence of a control condition, blind evaluators to assess treatment outcome not being used, ratings of adherence to treatment not being specified, symptom severity ratings not provided, and treatment not manualized.

These studies supported prolonged exposure as an effective treatment for PTSD. Not only is exposure probably the most researched psychological treatment for PTSD, but Foa et al. (2002) also concluded that prolonged exposure was more effective when conducted alone and not in combination with other cognitive-behavioural procedures such as stress inoculation training.

1.3 PROBLEM STATEMENT, OBJECTIVES AND HYPOTHESES

All available outcome studies supporting the effectiveness of exposure as psychological treatment for PTSD were based on American and British samples. No studies were identified in which the effectiveness of exposure treatment for PTSD was investigated outside the USA or UK, also not in South Africa with its culturally diverse and less affluent population. It is generally accepted that cultural variables may impact on psychological procedures, and that a procedure valid and proven in one culture, may not be effective and valid in a different cultural context (APA, 2000; Cantlie, 1994; Eagle, 2002; Kakar, 1985; Nordstrom, 1992; Roland, 1996; Rosenthal & Feldman, 1992; Stamm & Friedman, 2000). There is consequently a need to investigate the effectiveness of exposure treatment for PTSD in a South African sample.

In additon, the majority of studies investigating the effectiveness of exposure treatment on PTSD did so by examining possible changes in PTSD symptoms at post-treatment and follow-up, mostly using the Clinician-Administered PTSD Scale administered by blind evaluators. Only a few studies (e.g., Cooper & Clum, 1989; Foa et al., 1995; Keane et al., 1989) also examined the effect of exposure treatment on symptoms such as depression, anxiety, and dysfunctional beliefs, thereby providing insight into the process of change.

Thirdly, none of the available outcome studies investigated the possible effect of exposure treatment on brain functioning. Kolb (1987) suggested that possible changes in brain functioning, associated with PTSD, could be expected in regions that have previously been implicated in the pathophysiology of PTSD. Nadel and Moscovitch (1998) showed that the superior frontal regions, medial-temporal area, cerebellum, neocortex and hipocampus are all implicated in implicit memory and the retrieval of specific episodes and their contextual framework. It can therefore be expected that initial changes in these areas as shown on pre-treatment SPECT scans may be reversed on post-treatment SPECT scans. Schwartz, Stoessel, Baxter, Martin and Phelps (1996) showed that symptoms of obsessive-compulsive disorder coincided with significant correlations of brain activity between the orbital gyri and the head of the caudate nucleus and the orbital gyri and the thalamus on the right. By means of positron emission tomography (PET scans) they were able to demonstrate that these correlations decreased significantly after effective behaviour modification for obsessive-compulsive disordered patients. The question may therefore be posed as to whether effective exposure treatment for PTSD will similarly be

associated with changed brain activity in the brain regions implicated in PTSD, namely the superior frontal region, the medial-temporal area, the cerebellum, neocortex, and the hipocampus.

Based on these considerations the objectives of the present study were to investigate the effectiveness of prolonged exposure treatment

- in significantly reducing PTSD symptoms in a South African sample of female rape survivors;
- in significantly reducing the co-morbid symptoms of depression anxiety, and dysfunctional thinking associated with PTSD;
- in altering changed brain functioning in specific brain areas implicated in PTSD such as the superior frontal region, medial-temporal area, cerebellum, neocortex, and hipocampus.

The study therefore examine the following hypotheses:

Hypothesis 1: Prolonged exposure treatment will result in a significant reduction of all three symptom clusters at post-treatment and this improvement will be maintained at follow-up after three months.

Hypothesis 2: Prolonged exposure treatment will result in a significant reduction of the co-morbid symptoms of depression, anxiety, and dysfunctional thinking, associated with PTSD.

Hypothesis 3: Prolonged exposure treatment will result in altering changed brain functioning in specific brain areas implicated in PTSD such as the superior frontal region, medial-temporal area, cerebellum, neocortex, and hipocampus.

1.4 OUTLINE OF DISSERTATION

Chapter 2 of this discertation presents a description of PTSD in terms of diagnostic criteria, differential diagnosis, co-morbidity, course and prognosis, impact on functining and quality of life, epidemiology and prevalence, trauma memory, cultural variables, and neurobiology and neurochemical systems. Psychological treatments for PTSD as well as research evidence for their effectiveness are discussed in Chapter 3. Chapter 4 deals with prolonged exposure treatment for PTSD. It describes exposure treatment, theories underlying exposure treatment, and outcome studies on its effectiveness for PTSD. The research method, results and discussion are presented in Chapters 5 to 7 respectively.

2. POST-TRAUMATIC STRESS DISORDER

The concept of a trauma-related emotional disturbance has existed for over a century. According to Kaplan and Sadock (1998) a syndrome very similar to what is now called PTSD was noted in soldiers during the American Civil War. It was then called "soldier's heart" due to the presence of autonomic cardiac symptoms. During World War I the syndrome was called "shell shock", and was hypothesized to result from brain trauma due to the explosion of mortar shells. This syndrome was also seen during and after World War II in the survivors of Nazi concentration camps and the Hiroshima and Nagasaki atomic bombings in Japan. At this stage it was sometimes referred to as "operational fatigue" or "combat neurosis". According to Kaplan and Sadock (1998), in all these instances the appearance of the syndrome was correlated with the severity of the stressor – the most severe stressors (e.g., imprisonment in concentration camps) resulting in the appearance of the syndrome in over 75% of the surviving victims.

However, recognition of PTSD as a formal disorder and its official categorization in the diagnostic nomenclature only occurred in 1980, when it was first introduced in the third edition of the DSM (APA, 1980). In previous editions stress reactions were described in terms such as "gross stress reaction" and "transient situational disturbance", but without empirical support or specific criteria.

Official recognition of PTSD as a formal disorder, largely based on studies of combat veterans, stimulated a congruence of disparate areas of research on different types of trauma, resulting in rapid developments in theory and treatment (Friedman & Rosenheck, 1996; Schnurr & Friedman, 1999).

2.1. DIAGNOSTIC CRITERIA

Post-traumatic stress disorder is described by the DSM-IV-TR (APA, 2000) as a set of typical symptoms that develop in response to a traumatic stressor. It therefore follows that a *relevant traumatic stressor* (Criterion A) must be present for a formal diagnosis of PTSD to be made.

Initially the DSM-III (APA, 1980) as well as the DSM-III-R (APA, 1987) described a traumatic stressor as an event outside the range of normal human experience which almost any person would find markedly distressing. This original definition excluded relatively common traumatic experiences such as childhood abuse, domestic violence and criminal assault. This problem was eliminated in the DSM-IV (APA, 1994). According to the updated criteria of the DSM-IV, to qualify for a diagnosis of PTSD an individual must have witnessed, experienced, or otherwise have been confronted with an event that involved actual or possible death, grave injury, or threat to physical integrity. In addition, the individual's response to the event must include severe helplessness, fear or horror. The DSM-IV (APA, 1994) therefore emphasizes that the direct or indirect threat to life or well-being, as well as the individual's response to that threat, are the factors that specifically contribute towards making a given event traumatic. This description of a traumatic event was based on studies showing that the experience of life threat and subjective distress are significant predictors of the development of PTSD (e.g., Blank, 1993; March, 1993). According to Marshall and Rothbaum (2004) a wide range of fairly common experiences fall within this definition of trauma, such as car accidents, industrial accidents, domestic violence, robbery, criminal assault, rape, natural disasters, and war-related experiences. Research has also shown that the same pattern of symptoms follows these divergent traumatic events. This indicates that the determining factor underlying the pattern of symptom formation is a shared response diathesis rather than a trauma-specific pattern of distress (McFarlane, 2002). It is now commonly accepted that the type of trauma experienced is less important in the development of PTSD than trauma severity and individual reactions and vulnerabilities, despite different traumas showing some unique features.

According to the DSM-IV-TR (APA, 2000, p.8) PTSD is characterised by a group of 17 symptoms organized into three symptom clusters. According to the first symptom cluster (Criterion B) the traumatic event is persistently re-experienced in one (or more) of the following ways:

- Recurrent and intrusive distressing recollections of the event, including images, thoughts or perceptions. (Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed)
- Recurrent distressing dreams of the event. (Note: In children, there may be frightening dreams without recognizable content)

- 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 on awakening or when intoxicated). (Note: In young children, trauma-specific re-enactment may occur)
- Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
- Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

Criterion C refers to 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 symptoms:

- Efforts to avoid thoughts, feelings, or conversations associated with the trauma
- Efforts to avoid activities, places, or people that arouse recollections of the trauma
- Inability to recall an important aspect of the trauma
- Markedly diminished interest or participation in significant activities
- Feeling of detachment or estrangement from others
- Restricted range of affect (e.g., unable to have loving feelings)
- Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

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

- Difficulty falling or staying asleep
- Irritability or outbursts of anger
- Difficulty concentrating
- Hypervigilance
- Exaggerated startle response

According to Criterion E, the duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month, while according to Criterion F the disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Feelings of fear, horror and helplessness are some of the initial psychological reactions to trauma, and are essential symptoms for the diagnosis of PTSD. Other associated symptoms include feelings of guilt, shame or despair, withdrawal, social isolation, increased hostility, domestic violence, and changes in belief structures. These symptoms may occur irrespective of whether a specific psychological disorder such as PTSD can be diagnosed (Foa et al., 2006). Certain somatic symptoms may also occur, such as gastro-intestinal, cardio-vascular, respiratory, dermatologic, urological, or neurological problems.

PTSD is specified as acute if the duration of symptoms is less than three months, and as chronic if the duration of symptoms exceeds three months. Delayed onset PTSD is specified if the onset of symptoms is at least six months after the stressor, and is recognised as a discrete subcategory of PTSD. Very little is currently known about this specific condition. Based on available evidence delayed-onset PTSD is considered uncommon following disasters (McFarlane, 1992b, 2000). This was borne out in a study by Grace, Green, and Lindy (1993) where marked fluctuations in PTSD symptoms during long-term follow-up of survivors of natural disasters were reported.

2.2 DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

An essential requirement for a diagnosis of PTSD is the fact that the patient has experienced or witnessed a severely traumatic event as described in the DSM-IV-TR (APA, 2000). As the symptoms of PTSD overlap with those of a broad range of different psychiatric disorders, their association with a specific trauma must be established in order for a diagnosis of PTSD to be made (Marshall & Rothbaum, 2004).

Symptoms of avoidance, numbing and increased arousal which may have been present before the traumatic experience do not warrant a diagnosis of PTSD and should be considered for other diagnoses, e.g., a mood disorder or other anxiety disorder.

The DSM-IV-TR (APA, 2000) diagnostic criteria for PTSD require that the symptoms of re-experiencing, avoidance and hyper-arousal have lasted more than one month. When symptoms have been present for less than a month, the appropriate diagnosis would be acute stress disorder. Acute stress disorder is distinguished from PTSD by the fact that the symptom pattern must occur within four weeks of the trauma and must resolve within that four week period. If the symptoms persist for more than one month and the criteria for PTSD are met, the diagnosis is changed from Acute stress disorder to PTSD.

Obsessive-compulsive disorder also presents with recurrent intrusive thoughts, but the patient experiences these as inappropriate and they are not related to a traumatic event. Flashbacks in PTSD must be distinguished from illusions, hallucinations, and other perceptual disturbances which may occur in schizophrenia, other psychotic disorders, mood disorder with psychotic features, substance-induced disorders, delirium, and psychotic disorders due to a general medical condition (APA, 2000).

In making a differential diagnosis, a major feature in the diagnosis of PTSD is the possibility that the patient also incurred a head injury during the trauma. Other organic conditions that can both cause or exacerbate the presenting symptoms could be epilepsy, alcohol use disorders, and other substance-related disorders. Acute intoxication or withdrawal from some substance must also be eliminated during the initial examination, as these conditions may present a clinical picture that is difficult to distinguish from PTSD until the effects of the substance have worn off (Kaplan & Sadock, 1998).

PTSD must be distinguished from other mental disorders such as borderline personality disorder, dissociative disorders, factitious disorders, and malingering. Borderline personality disorder could be difficult to distinguish from PTSD, as the two disorders may be causally related and may even co-exist. Patients with dissociative disorders do not usually present with the same degree of avoidance behaviour, autonomic hyper-arousal, or a history of trauma that patients with PTSD report (Kaplan & Sadock, 1998). The possibility of a fictitious disorder or malingering should also be considered when making a diagnosis, especially in situations where financial gain, benefit eligibility, and forensic determinations play a role. Conditions such as depression and substance abuse may develop as complications of PTSD, and must be diagnosed as such and not as primary disorders. Ultimately, the distinguishing factor remains the fact that the patient has experienced a severe trauma.

2.3 POSTTRAUMATIC STRESS DISORDER AND COMORBIDITY

As indicated by epidemiological studies, a large spectrum of disorders, such as affective disorders, panic disorder, as well as alcohol and substance abuse, often emerge in conjunction with PTSD. Solomon and Davidson (1997) concluded that individuals with a primary lifetime diagnosis of PTSD are between two and four times more likely to meet criteria for another disorder in their lifetime compared to individuals with any other diagnosis. This finding concurs with data from the National Comorbidity Study which showed that individuals with PTSD are almost eight times more likely to have three or more disorders compared to those without PTSD (Kessler et al., 1995).

Studies of war veterans by Deering, Glover, Ready, Eddleman, and Alarcon (1996) and Keane and Wolfe (1990) found that substance abuse and affective disorders are common comorbid diagnoses in veterans with PTSD. Kessler et al., (1995) also showed that generalized anxiety disorder and phobias often co-exist with PTSD in war veterans.

Data from the National Comorbidity Study demonstrated that 88% of men and 79% of women with a history of PTSD have one or more comorbid psychiatric diagnosis, often major depression, anxiety disorders, or alcohol/substance abuse (Lecrubier, 2004). Studies on PTSD in civilian populations reported generalized anxiety disorder to be the most common comorbid anxiety disorder in PTSD. Rates ranged from 7% in a study by Breslau et al. (1991) to almost 76% in a study by Shore, Vollmer, and Tatum (1989).

Differences in assessment methods and diagnostic criteria may account for some of the discrepancies in reported comorbidity rates. Although it is apparent that PTSD is assosiated with high rates of comorbidity, it is not always clear whether this is unique to PTSD (Zayfert, Becker, Unger, & Shearer, 2002). Few, if any, studies allow comparison of comorbidity rates between PTSD and other similarly severe Axis I disorders (Keane & Wolfe, 1990). Large comorbidity studies typically report on PTSD in a separate report (Kessler et al., 1994; Kessler et al.,1995), and this disorder has often been excluded from studies of comorbidity among other anxiety disorders (Brown & Barlow, 1992; Sanderson, Di Nardo, Rapee, & Barlow, 1990). Studies of comorbidity in PTSD often focus exclusively on traumatised populations (e.g., Hubbard, Realmuto, Northwood, & Masten, 1995; Shore et al., 1989). Keane and Wolfe (1990) consequently pointed out that it was unclear whether comorbidity rates for

PTSD are actually higher than for other anxiety disorders. In a more recent report Marshall and Rothbaum (2004), however, stressed that PTSD has the highest rate of comorbidity of any Axis 1 disorder and the most extensive overlap across the affective, anxiety and personality disorders.

A frequent argument by clinicians is that patients with PTSD present with exceedingly high rates of comorbidity, which make treatment more difficult, and which may be a contra-indication for empirically supported therapies (Pitman et al., 1991; Solomon & Davidson, 1997). Previously available data on this aspect has largely been derived from population-based studies of trauma survivors and may not reflect the diagnostic profiles of individuals who present for treatment (Zayfert et al., 2002). In a recent study to determine whether the clinical viewpoint regarding PTSD and comorbidity in treatment-seeking populations was accurate, Zayfert et al. (2002) found a greater association of Major depressive disorder with PTSD, relative to Panic disorder. Patients with PTSD showed a significantly higher rate of overall comorbidity relative to other patients seeking treatment in an anxiety disorders clinic. The presence of multiple anxiety disorders among treatment-seeking PTSD patients may influence treatment outcomes as comorbid anxiety disorders may contribute to chronicity, which may possibly be associated with resistance to intervention.

According to Brady, Killeen, and Brewerton (2000) a *Major Depressive Disorder* is the most common concomitant psychiatric disorder in patients with PTSD. The core symptoms in a Major Depressive Disorder are usually accompanied by a range of both physical symtoms including appetite changes, sleep disturbances, and loss of energy, as well as psychological symptoms, including excessive guilt, feelings of worthlessness and suicidal thoughts. A history of major depressive disorder may therefore be predictive of PTSD after exposure to trauma. The presence of a major depressive disorder is associated with greater functional impairment in patients with PTSD as well as with less likelihood of showing remission of symptoms over a six months period (Foa et al., 2006).

In *Panic Disorder*, another possible comorbid disorder with PTSD, fear and avoidance are related to the physical symptoms associated with panic attacks (anticipatory anxiety), while in PTSD they are related to specific trauma-related situations and memories (Brady et al., 2000).

According to Breslau, Davis, and Schultz (2003) *abuse of nicotine, alcohol and narcotic drugs* is common among patients with PTSD. They maintained that increased substance use after trauma

appears to be related to PTSD rather than to exposure to stress per se. McFarlane and Papay (1992), in an article on multiple diagnoses in victims of a natural disaster, also concluded that PTSD was associated with both increases and decreases in alcohol consumption and that these changes could be attributed to PTSD rather than to exposure to stress.

Somatic symptoms are often found to overlap with distinctive PTSD symptoms. There is growing evidence that information-processing in PTSD becomes disordered, consequently contributing to the presentation of somatic symptoms (Breslau, 2001a).

McFarlane (2002) stressed that PTSD patients with co-morbid disorders are likely to have a worse long-term prognosis compared to those without co-morbidities, and that they may require chronic maintenance therapy.

2.4 COURSE AND PROGNOSIS

Posttraumatic stress disorder may occur at any age, including childhood. Usually symptoms appear within the first three months after the traumatic experience, although there may be a delay of months, or even years, before symptoms appear (APA, 2000).

Symptoms of PTSD developing within four weeks after the experienced trauma represent an acute stress response, which is a normal response to stress. The majority of trauma victims experiencing these symptoms do not necessarily develop any psychological disorders. Posttraumatic stress disorder therefore does not commence immediately after the trauma, but rather represents a lack of resolution of the acute stress response to the trauma. Initial PTSD symptoms resolve in approximately 60% of cases (McFarlane 2000). Kessler et al. (1995) quoted PTSD as being the fifth most common psychiatric disorder in the United States, with a lifetime prevalence of 7.8% in the National Comorbidity Survey. According to the results of this survey, however, trauma is a much more common occurrence, affecting 61% of men and 51% of women. This clearly shows that not all traumatized individuals develop PTSD.

Symptoms can fluctuate over time and may be most intense during periods of stress. According to Kaplan and Sadock (1998) about 30% of patients recover completely, 40% continue to have mild symptoms, 20% continue to have moderate symptoms, and 10% remain unchanged or become worse.

2.4.1 Risk factors in the development of PTSD

The original conceptualization of PTSD was that it represents a direct consequence of exposure to a traumatic event in otherwise normal individuals. The original emphasis was on establishing the primacy of the trauma as being the etiological agent. Individual vulnerability factors received little emphasis. As all trauma survivors do not develop a permanent disorder and many do recover, the emphasis shifted to possible risk factors that may increase vulnerability to PTSD, and may influence the prognosis of individuals with PTSD.

The severity, duration, and proximity of an individual's exposure to the traumatic event are the most important factors affecting the likelihood of developing this disorder. Posttraumatic stress disorder can develop in individuals without any predisposing conditions, particularly if the stressor is especially extreme (APA, 2000).

Research showed that the risk of developing PTSD varies with a number of pre-traumatic, peri-traumatic, and post-traumatic factors (Breslau et al., 1991; Kessler et al., 1995; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993; Schnurr, Friedman, & Rosenberg, 1993). The development of chronic PTSD may also be related to many of the same risk factors (King, King, Fairbank, Keane, & Adams, 1998; King et al., 1996; Kulka et al., 1990; Schnurr & Vielhauer, 1999; Stein, Koverola, Hanna, Torchia, & McClarty, 1997). Only a few studies of risk factors for PTSD distinguished between chronic and less chronic PTSD (Schnurr, Lunney, & Sengupta (2004). As a result very little is known about specific factors contributing to the development of PTSD, as opposed to factors associated with the maintenance of PTSD.

In a comparison of individuals who developed short-term PTSD and individuals who developed chronic PTSD after a mass shooting, North, Smith and Spitznagel (1997) found that neither demographic characteristics, pretraumatic and acute psychiatric comorbidities, nor exposure characteristics, predicted remission in survivors. McFarlane (1988b, 1992a) also found that the only

difference between these two groups was a greater likelihood of concentration difficulties in the chronic group. Dunmore, Clark and Ehlers (2001) compared chronic and remitted cases of PTSD after sexual and physical assault, and found that the chronic cases had less education and were more likely to be single at the time of the trauma. Chronic cases were less likely to have had pretraumatic emotional problems but they showed more severe PTSD symptoms in the month following the trauma Mental defeat, confusion, negative appraisal of actions and emotions during the assault, negative appraisal of initial symptoms, perceptions of permanent change, avoidant symptom control strategies, and negative global beliefs after the assault, were all cognitive factors which appeared to be strongly associated with the maintenance of PTSD.

In a study by Breslau and Davis (1992) in which acute and chronic cases of PTSD were compared, factors identified as predictors of chronic but not of acute PTSD, were female gender, a family history of antisocial behaviour, pre-existing anxiety or depression, and neuroticism. In a study of motor accident victims with PTSD, Blanchard et al. (1997) found that continued PTSD was associated with higher initial PTSD severity, more severe injury, a greater number of remaining injuries four months after the accident, and familial trauma after the accident. Green, Grace, Lindy, and Gleser (1990) identified exposure to death and mutilation, and a higher number of special assignments, as two prominent indicators of acute PTSD in a sample of male Vietnam war veterans.

Schnurr et al. (2004) analyzed two large, ethnically diverse studies of Vietnam veterans, namely the National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al., 1990) and the Hawaiian Vietnam Veterans Project (HVVP; Friedman et al., 1997), and used a comprehensive set of pretraumatic, peritraumatic and posttraumatic risk factors, and an analytic method that allowed statistical comparison of the effects of these risk factors on the development and maintenance of PTSD. They found premilitary socio-economic status, perceived life threat, peritraumatic dissociation, homecoming emotional sustenance, and post-Vietnam trauma to be associated with the development of PTSD, while gender, ethnicity, atrocity exposure, and current emotional exposure predicted maintenance of PTSD.

In a study with Vietnam veterans, Foy, Sipprelle, Reuger, and Carroll (1984) found that *combat exposure and military adjustment* were significantly related to the developmemt of PTSD. Initial adjustment immediately after the trauma did not seem to be a risk factor. The *characteristics of the trauma exposure* was shown to be of central importance.

Environmental factors may play an important role in the development of PTSD. In addition to trauma severity, a history of prior exposure to trauma or to chronic stress are important risk factors (Davidson, Hughes, Blazer, & George, 1991; Halligan & Yehuda, 2000). This risk is increased if the exposure or stress is experienced at a young age (Bremner et al., 1993). Breslau, Chilcoat, Kessler, and Davis (1999) argued that prior exposure to assault is a particularly potent risk factor. King et al. (1996) pointed out that a history of family instability may be associated with an increase in the prevalence of PTSD, while Solomon, Mikulincer, and Avitzur (1988) found that less severe levels of symptoms can be attributed to good social support.

Certain *demographic factors* may increase the risk for developing PTSD. According to Breslau et al. (1998) gender is an extremely important risk factor as the prevalence of PTSD is nearly twice as high in women than in men. This higher risk for PTSD in women is considered a result of their vulnerability to assaultive violence, and as most perpetrators are physically stronger males, assaultive violence is more injurious and threatening to females (Breslau, Chilcoat, Kessler, Petersen, & Lucia, 1999). Lower levels of income and education as well as being divorced or widowed, were also identified as risk factors for PTSD (Halligan & Yehuda, 2000). Some studies (Breslau et al., 1998) have identified ethnic minorities as at greater risk for developing PTSD, but others (Breslau et al., 1991; Norris, 1992) found that ethnic differences may be attributable to or may interact with other factors but cannot be regarded as risk factors per se. Breslau et al. (1998) argued that some of the identified demographic predictors of PTSD may actually be predictors of trauma exposure and not necessarily predictors for the development of PTSD.

Personality and psychiatric disorders may also constitute risk factors. Several studies investigated the personality dimension of negative emotionality, described by Miller (2004), as a possible predictor of PTSD. Negative emotionality refers to dispositions toward negative mood and emotion and a tendency towards adversarial interactions with others. It is synonymous with neuroticism, subsuming traits relating to anxiety, alienation and aggression. While some studies have revealed a significant

association between pretrauma negative emotionality and the development of PTSD (Bramsen, Dirkzwager, & Van der Ploeg, 2000; Lee, Valiant, Torrey, & Elder, 1995; O'Toole, Marshall, Schureck, & Dobson, 1998; Schnurr et al., 1993), other studies have identified negative emotionality as playing a key role in the etiology of PTSD (Bennett, Owen, Koutsakis, & Bisson, 2002; Breslau et al., 1991; Cox, MacPherson, Enns, & McWilliams, 2004; McFarlane, 1992b; Van Zelst, De Beurs, Beekman, Deeg, & Van Dyck, 2003). These studies pointed to high negative emotionality as the primary personality risk factor for PTSD following exposure to trauma. However, Clark, Watson, and Mineka (1994), as well as Krueger, McGrue, and Iacono (2001) showed high negative emotionality to be a non-specific predictor of a broad spectrum of psychopathology referred to as the "internalizing disorders" or "distress disorders" (Miller, 2004). As such, negative emotionality may rather be classified as a generalized biological vulnerability to anxiety and its disorders, rather than as a specific risk factor for PTSD.

McFarlane (1989) found that a *past history of psychological or behavioural problems* may be associated with the development of PTSD. Breslau et al. (1998) concurred, and found that prior affective, anxiety or substance abuse disorders were risk factors for developing PTSD. They stressed that having a psychiatric history per se was a stronger risk factor than having a history of a specific disorder. Personality dimensions, such as adult avoidant, antisocial, or neurotic personalities prior to the trauma, were shown to present an increased risk for developing PTSD (Breslau et al., 1998; Schnurr et al., 1993; Schnurr & Vielhauer, 1999). According to Koopman, Classen, and Spiegel (1994) *peri-traumatic dissociation* seems to be an important risk factor for PTSD. Several subsequent studies underlined this conclusion (Holen, 1991; Marmar et al., 1999; Shalev, Peri, Canetti, & Schreiber, 1996; Tichenor, Marmar, Weiss, Metzler, & Ronfeldt, 1996). Zoellner, Alvarez-Conrad, and Foa (2002) concluded that peritraumatic dissociation interferes with the encoding of traumatic memories and this may impede recovery from PTSD. Spiegel (1991) argued that dissociative reactions may be adopted as maladaptive coping strategies in response to childhood trauma or chronic stress, and as such, may partially be mediating between prior traumatization and subsequent increased vulnerability to PTSD.

Macklin et al. (1998) assessed *intelligence* in soldiers prior to entering combat and found that lower intellectual functioning is a risk factor for the development of PTSD. Gurvits et al. (2000) reported that PTSD patients showed increased neurological "soft signs", indicating subtle nervous system disfunction. In addition, they reported a larger number of developmental problems in these PTSD

patients, which suggest that there are *pre-existing impairments in neuro-development* which act as risk factors for PTSD.

Although not yet proven as a risk factor, *specific impairments in explicit memory* have been observed in PTSD patients compared to controls (Jenkins, Langlais, Delis, & Cohen, 1998; Yehuda et al., 1995). Halligan and Yehuda (2000) stressed the possibility that lower mnemonic ability may predate the trauma.

Studies of *biological dimensions* of PTSD showed several abnormalities that are present in trauma survivors with PTSD. Recent evidence has suggested that at least some of these biological abnormalities represent risk factors for the development of PTSD. It has been claimed that biological factors may render patients with PTSD to be hyper-responsive to stressful stimuli, especially stimuli that are reminiscent of the trauma. An enhanced or prolonged catecholamine response to trauma, presented in a higher heart rate, was found in a study by Shalev, Freedman, et al. (1998), while Pitman (1989) found that elevated norepinephrine during trauma may result in overconsolidation of memory and the subsequent development of intrusive symptoms.

In the field of neuro-hormonal research it was found that both combat related and civilian PTSD was associated with chronically low levels of cortisol, a gluco-corticoid secreted by the hypothalamic-pituary-adrenal (HPA) axis. This is indicative of systematic alterations in overall HPA axis activity, apparently unique to PTSD. Compelling evidence is presented by Yehuda, Teicher, Trestman, Levengood, and Siever (1996) that such alterations are symptoms of enhanced negative feedback occurring in the HPA axis, mediated by increased gluco-corticoid receptor sensitivity, and acting to keep corticol levels chronically low (Halligan & Yehuda, 2000). Further support for the theory that *HPA axis abnormalities* represent a risk factor for PTSD, was provided by studies of rape victims (Resnick, Yehuda, Pitman, & Foy, 1995) and motor vehicle accident survivors (Yehuda, McFarlane, & Shalev, 1998).

According to Koenen (2003) twin studies provided the strongest evidence thus far for *genetic influences* as risk factors for both trauma exposure and PTSD. True et al. (1993) found substantial genetic influences for all PTSD symptoms in Vietnam veterans, after adjusting for differences in combat exposure. A recent study with Vietnam veterans by Koenen et al. (2003) showed shared genetic influence on PTSD and major depression, and a study by Stein, Jang, Taylor, Vernon and

Livesley (2002) demonstrated that exposure to assaultive violence (e.g., physical assault, combat, rape) and PTSD symptoms were both moderately heritable. A study of monozygotic versus dyzygotic twins (True et al., 1993) demonstrated that 30% of some PTSD symptoms appear to have a genetic basis. Davidson, Swartz, Storck, Krishnan, and Hammett (1985) demonstrated that trauma survivors with PTSD were more likely to have parents and/or first-degree relatives with substance abuse, anxiety, and mood disorders compared to trauma survivors who did not develop PTSD. Halligan and Yehuda (2000) concluded that the extent to which these findings are indicative of a truly genetic basis for PTSD is not yet clear. They believe that family studies may ultimately demonstrate *inter-generational effects of stress and trauma* as the most potent risk factors due to the persistent cognitive and neurobiologic changes they induce. From the point of genetics research, PTSD is considered a complex or polygenetic disorder. Unlike other diseases that are caused by a single gene, such as Huntington's disease, there is probably no single 'PTSD gene'. There are probably several genes, each of which contributes interchangeably and additively, in a probabilistic fashion, to the inherited liability for PTSD (Koenen, 2003).

In a study of familial risk factors for PTSD, Koenen et al. (2002) found that a family history of conduct disorder, major depression, panic disorder, generalized anxiety disorder or substance dependance were each significantly associated with PTSD. However, according to Koenen et al. (2003) this association is indirect, mediated by increased risk of trauma exposure and pretrauma pathology. In studies comparing the prevalence of PTSD in Cambodian refugee children with the prevalence of PTSD in their parents (Sack, Clarke, & Seeley, 1995,) it was found that children with one or more parent who had PTSD were almost five times more likely to be diagnosed with the disorder than children with no family record of PTSD. A study by Yehuda et al. (1995) on adult children of Holocaust survivors with PTSD has shown that these children have a higher risk of PTSD following trauma exposure than adult children of Holocaust survivors without PTSD.

Association studies, utilising the association method which can detect genes of small effect, is usually the method of choice for molecular genetic studies of complex disorders (Koenen, 2003). Humans have been found to be 99.9% genetically identical. Therefore, research aimed at identifying genes that explain individual differences in risk for developing PTSD, focuses on the minute fraction of DNA that differs among individuals. Association studies correlate a DNA marker's alleles (different forms of DNA at a specific place on the chromosome) with an outcome. Association studies may, alternatively,

focus on endophenotypes or markers of the neuro-biological pathways thought to mediate the relationship between the gene and the relevant disorder. In a study by Radant, Tsuang, Peskind, McFall, and Raskind (2001) on the biological markers and diagnostic accuracy in the genetics of PTSD, the authors pointed out obstacles to the genetic analysis of PTSD, and suggested that these obstacles can be overcome by performing genetic analysis of traits associated with PTSD, such as changes in the HPA axis as well as increased startle response. They proposed that *genes involved in the endophenotypes of HPA axis dysregulation, the physiology of hyperarousal, and the acoustic startle response* might influence the development of PTSD.

Some aspects of the initial reaction to trauma exposure may be predictive of the development of PTSD. In the initial aftermath of trauma, intrusive re-experiencing is common. Shalev (1992) found that the mere presence of these symptoms is not a good predictor of PTSD, but that certain characteristics of these intrusive memories may be better at predicting PTSD. Michael (2000) conducted two studies of assault survivors and found that several characteristics of intrusive memories distinguished between survivors with and without PTSD and also predicted subsequent PTSD severity. These characteristics included d*istress caused by the intrusion, lack of time perspective, and lack of context* (Ehlers, Hackmann, & Michael, 2004).

Brewin, Andrews, and Valentine (2000) examined the impact of the following risk factors for PTSD in a meta-analysis: gender, age at the time of the trauma, education, intelligence, race, socio-economic status, previous psychiatric history, reported childhood abuse, reported previous traumatisation, reported adverse childhood experiences, family history of psychiatric disorder, severity of trauma, post-trauma life stress, and post-trauma social support. Statistically each of these variables was found to be highly significant although the size of the effects varied markedly. It appeared that factors that were present before the trauma had a relatively small effect on the risk for PTSD, while the largest effects were noticed for those factors operating during or after the trauma, such as trauma severity, lack of social support, and post-trauma stress.

Another research question deals with factors that may contribute to the development of chronic PTSD. Breslau and Davis (1992) studied risk factors for chronic PTSD in an urban population of young adults. Individuals with chronic PTSD were found to have a higher total number of PTSD symptoms, higher rates of numbing and hyperreactivity to stressors, anxiety or affective disorders, and other

comorbid medical conditions, compared to individuals with non-chronic PTSD (Foa et al., 2006). Independent risk factors for chronic PTSD were found to be female gender and a family history of PTSD. Other factors which influence the development of chronic PTSD included avoidance behaviours in response to stressors. Whereas intrusive symptoms usually decrease with time, avoidance symptoms tend to increase (Blank, 1993; McFarlane, 2000). Avoidance behaviours may limit the patient's exposure to experiences which could correct negative perceptions and beliefs. It may also prevent the re-organisation of memory and the necessary consignment of the trauma memory to the past. Avoidance symptoms may also reinforce the individual's perception of the world as a dangerous place and of himself/herself as unable to cope effectively with stress (Foa et al., 2006). Peritraumatic dissociative experiences, by interfering with the encoding of traumatic memories, may also hamper recovery and thereby increase the risk of perpetuating PTSD to a chronic state (Zoellner et al., 2002).

In conclusion, from the preceding overview it is clear that there is currently no consensus on possible risk factors for the development of PTSD. There seem to be some agreement on the weight of some risk factors in the development of PTSD than with other factors. These include the severity of the trauma, prior exposure to trauma, the age at exposure to trauma, female gender, and prior psychological problems such as affective, anxiety, and substance abuse disorders. The influence of other factors such as level of education, level of income, biological and genetic dimensions, and negative emotionality seem less clear. It is evident that more research is needed on the role of specific risk factors in the development of PTSD.

2.5 IMPACT ON FUNCTIONING AND QUALITY OF LIFE

A large and growing body of literature presents the negative effect of PTSD in terms of distress and dysfunction cased. From a longitudinal study in a clinical setting, Warshaw, Fierman, and Pratt (1993) concluded that PTSD has severe effects on quality of life, affecting virtually all aspects of one's existence. These include high levels of depression, suicide attempts, and alcohol abuse. Furthermore, data from the National Comorbidity Survey showed that individuals with PTSD are six times more likely than those without PTSD to attempt suicide, and that about 19% of PTSD patients will eventually attempt suicide (Foa et al., 2006; Kessler, Borges, & Walters, 1999). Several studies, including studies of Vietnam War veterans as well as of patients with anxiety disorders, found

significantly higher rates of suicide attempts, alcohol abuse or dependence, and hospitalization for psychiatric illness in patients with PTSD compared to patients without a PTSD diagnosis. (Warshaw et al., 1993; Zatzick, Marmar, et al., 1997; Zatzick, Weiss, et al., 1997).

The original National Comorbidity Survey revealed that PTSD was associated with approximately 3.6 days of work impairment per month (Kessler et al., 1995; Kessler et al., 1999). Data from the National Comorbidity Survey Replication study (Kessler, Chiu, Demler, Merikangas, & Walters, 2005) showed the 12-months prevalence of PTSD as 3.5%. Of those with PTSD, 36.6% were rated as 'serious' (based on criteria such as work disability, role impairment, and suicide attempts). Higher severity was significantly associated with greater interference with normal activities and with more psychiatric comorbidities.

In a study by Stein, Walker, Hazen, and Forde (1997) participants with PTSD showed the most substantial interference with occupational or school activities, but those with partial PTSD (i.e. with less than the required number of symptoms on Criteria C and D) showed more occupational impairment when compared to participants who had experienced trauma but did not meet the criteria for either full or partial PTSD. Breslau, Lucia, and Davis (2004) found that individuals with full PTSD, when compared to those with partial PTSD, showed greater impairment in terms of work days lost, greater interference with work or daily activities, spend less time on interpersonal communication, and increased conflicts or tensions because of their personal reactions to the trauma. Norris, Murphy, Baker, and Perilla (2003) found that functional impairment was the best single predictor of the duration of PTSD symptoms (more than one year vs. less than one year). Stein, McQuaid, Pedrelli, Lenox, and McCahill (2000) compared primary care patients with PTSD to patients with no mental disorders. The results showed greater impairment in work, and home and social life in the PTSD group.

Based on data from the National Vietnam Veterans Readjustment Study, Kulka et al. (1990) found that both male and female veterans with PTSD, compared to those without PTSD, reported poorer physical health, a greater need of medical services, and greater work impairment. The PTSD veterans were also less likely to be married compared to those without PTSD, and those who had married experienced more marital problems and a higher rate of divorce. The PTSD veterans also reported a higher rate of arrests, vagrancy or homelessness, as well as a much higher rate of acts of violence in the previous year. Using data from the National Comorbidity Survey, Whisman (1999) found that those patients

diagnosed with PTSD were between three and six times more likely to divorce than those without PTSD. In this study, PTSD was the second most likely diagnosis associated with marital problems.

Several studies of Vietnam veterans showed that the severity of aggressive behaviour was associated with the severity of PTSD symptoms (Byrne & Riggs, 1996; Glenn et al., 2002; Zatzick, Marmar, et al., 1997). Riggs, Byrne, Weathers, and Litz (1998) found that Vietnam veterans with PTSD showed more anxiety related to intimacy than those without PTSD, and Cook, Riggs, Thompson, Coyne, and Sheikh (2004) reported that World War II ex-prisoners of war experienced poorer adjustment and communication with their partners and more difficulties with intimacy, when compared to ex-prisoners of war without PTSD.

In a relatively rare study of female Vietnam veterans, those with PTSD reported a higher incidence of not working or poor physical health, or both, compared to those without PTSD (Zatzick, Weiss, et al., 1997). In studies of women with histories of intimate partner violence, Stein and Kennedy (2001) and Laffaye, Kennedy, and Stein (2003) found that those with PTSD reported poorer occupational and overall functioning, and poorer physical functioning, vitality, social functioning, as well as role limitations, than those without PTSD.

2.6 EPIDEMIOLOGY AND PREVALENCE

Since 1980 most PTSD research has been carried out with Vietnam combat veterans or specific samples of the population that have been exposed to disasters. Only a relatively small number of studies have been conducted which provide estimates of the prevalence of PTSD in the general population or in different subgroups (Breslau, 2001a). Surveys of the general US population had indicated that PTSD affects about 1 in every 12 adults at some stage. This is approximately 15% to 24% of those exposed to some kind of traumatic event (Breslau et al., 1991; Kessler et al., 1995). Reported rates of PTSD in general community samples vary widely across the world, from as low as 1.3% in Germany to 37.4% in Algeria (De Jong et al., 2001; Kessler et al., 1995; Perkonigg, Kessler, & Storz, 2000). Community based studies reveal a lifetime prevalence of PTSD ranging from 1% to 14% (DSM-IV, APA, 1994) or, according to Kaplan and Sadock (1998), from 1% to 3% of the general USA population, although an additional 5% to 15% may experience sub-clinical forms of the disorder (McFarlane, 2002). Among high-risk groups, whose members experienced traumatic episodes, the life-

time prevalence rates vary from 5% to 75% (Kaplan & Sadock, 1998). A life-time prevalence of 10.4% for women and 5.0% for men in the USA was found by the National Comorbidity Survey (Kessler et al., 1995).

2.6.1 Trauma populations

The prevalence of PTSD in the wake of national disasters created by terrorism has been catapulted into the limelight by the September 11 attacks in New York. In a study of 560 US adults, assessed three to five days after the September 11 terrorist attacks in New York, Schuster et al. (2001) reported that 44% of the sample had experienced one or more PTSD symptoms. In a larger national study by Schlenger et al. (2002) one to two months after the attack, the prevalence of probable PTSD in the New York metropolitan area was 11.2%, which is much higher than the overall national estimated prevalence of 4.3% (Resnick, Galea, Kilpatrick, & Vlahov, 2004). In a study by Silver, Holman, McIntosh, Poulin, and Gil-Rivas (2002) nine days to six months after the September 11 attacks, participants reported an average of almost 5 symptoms of acute PTSD-related stress at the initial assessment, although only 2% of the sample reported having experienced direct exposure, while 96% of the sample reported no losses related to the attacks. A series of epidemiological studies were conducted primarily on the population in New York City and the surrounding metropolitan area and included assessment of exposure variables, prior history factors, peri-event reactions, post-event factors and assessment of probable PTSD, major depression, and changes in drug, alcohol, and cigarette use (Ahern et al., 2002; Boscarino, Galea, Ahern, Resnick, & Vlahov, 2002; Boscarino, Galea, Ahern, Resnick, & Vlahov, 2003; Galea et al., 2002; Galea et al., 2003; Vlahov et al., 2002). The major finding from the initial report by Galea et al. (2002) was that at five to eight weeks following the attacks, the prevalence of current probable PTSD related to the terrorist attacks was 7.5% in Manhattan. As noted by Schlenger et al. (2002), this prevalence falls within the estimated prevalence of PTSD (11,2%) that was found in the New York metropolitan area at a similar point following the attacks. These figures are quite similar to the prevalence of PTSD of 34% observed in victims of the Oklahoma City bombing (North et al., 1999).

For natural disasters, a prospective study of 78 survivors of the 1988 earthquake in Armenia, by Goenjian, Steinberg and Najarian (2000) reported PTSD rates of 86.7% at 1.5 years and 73.3% at 4.5 years following the earthquake. Lower rates of 33.2% at nine months and 14.4% at two years following

an earthquake in Australia were reported by Lewin, Carr, and Webster (1998), while PTSD rates of 22.7% in a sample of 66 earthquake victims in China and 23% in a sample of 56 survivors in India were reported (Sharan et al., 1996; Wang, Gao, & Shinfuku, 2000).

A prospective study by Karamustafalioglu et al., (2006) examined 464 adult survivors of the 1999 earthquake in Turkey. Assessments for PTSD symptoms were done in three consecutive surveys within twenty months following the earthquake. This represented the biggest sample ever studied and followed up for nearly two years after an earthquake. The prevalence of subjects who met PTSD criteria on one survey or more was 45.7%. (Prevalence at each of the three surveys was as follows: 30.2% after 1 to 3 months, 26.9% after 6 to 10 months, and 10.6% after 18 to 20 months following the trauma). Participants who met the criteria during all 3 surveys were 5.2 of the entire sample. Thus the prevalence of a chronic course among the PTSD patients was 11.3%. The prevalence among females (49.8%) was higher than in males (39.9%). Prevalence was also significantly higher among participants with an education level lower than junior high school (48.3%) compared to subjects with junior high school education and above (34.8%). Delayed onset of PTSD during the first year was shown by 2.6% of the sample and an additional 1.5% showed delayed onset during the second year following the trauma.

Several studies found prevalence rates for PTSD of 70% and above after rape and sexual violence (McFarlane, 2002). Breslau et al. (1998) reported that the prevalence of PTSD following rape was nearly twice as high in women as in men. In the 1996 Detroit Area Survey of Trauma (Breslau et al., 1998) the conditional risk of PTSD associated with sexual assault was 35.7% in women versus 6.0% in men.

In samples of women, differential patterns of risk for PTSD based on type of trauma (e.g., rape vs physical assault) have been documented. Resnick et al. (1993) found that female victims of physical or sexual assault are four to five times more likely to develop PTSD than female victims of noncriminal trauma. Three prospective studies investigated the occurrence of PTSD in assault victims compared to rape victims (Foa, 1995; Riggs, Rothbaum, & Foa, 1995; Rothbaum, Foa, & Murdock, 1992). Across these studies, lower rates of PTSD were observed in victims of nonsexual assault compared to victims of rape.

In a large longitudinal study of community residing male veterans of World War II and the Korean War, it was found that those exposed to moderate or heavy combat have a 13.3 times greater risk of PTSD decades later (Spiro, Schnurr, & Aldwin, 1994). About 30% of Vietnam veterans experienced PTSD, and an additional 25% experienced sub-clinical forms of the disorder (Kaplan & Sadock, 1998). The National Vietnam Veterans Readjustment Survey showed that 8.5% of female theater veterans suffered from war-related PTSD while an additional 7.2% showed partial symptoms of the disorder (Kulka et al., 1990).

In a study by Coffey et al. (2006) a prevalence rate of 43% for PTSD was found after motor vehicle accidents in the USA. No differences were found between male and female survivors in terms of prevalence of PTSD or severity of PTSD symptoms as measured by the CAPS. Norris (1992) examined the frequency and impact of 10 potentially traumatic events in a large multisite epidemiological study and found motor vehicle accidents to be a leading cause of PTSD, exceeded only by sexual and physical assault. Extrapolating from the rates of trauma and PTSD, Norris estimated that motor vehicle accidents could account for 28 cases of PTSD for every 1 000 adults in the United States. A study by Blanchard and Hickling (1997) of 158 survivors of road traffic accidents found that 39% were diagnosed with PTSD one to four months after the event. In this study, Blanchard and Hickling came to the conclusion that motor vehicle accidents are one of the most common traumatic events in the USA with over three million people injured each year, 8% to 40% of whom will suffer from PTSD in the first post-accident year. Mayou, Bryant, and Duthie (1993) interviewed 188 emergency room patients who had experienced motor vehicle accidents in Oxford, UK. Fourteen (8.0%) met DSM-III-R criteria for PTSD at three months, and 13 (7.6%) met PTSD criteria at twelve months after the accidents. After a five-year follow-up period, 9 cases of PTSD (8.1%) and 10 other individuals with 'minor' PTSD were found. Ehlers, Mayou, and Bryant (1998) assessed 1148 motor vehicle accident survivors showing prevalence rates of 23% and 17% for PTSD at three months and one year follow-up respectively.

A large community survey of 2493 adults in the USA showed the prevalence of PTSD to be 3.5% in individuals exposed to physical attack (Helzer, Robins, & McEvoy, 1987). Amick-McMullen, Kilpatrick, and Resnick (1991) conducted a random-digit-dial survey of a nationally representative sample of 12,500 men and women, aged 18 or older, in the USA. Their findings indicated that 23% of

homicide survivors developed full PTSD and 50% developed one or more of the symptoms of PTSD. This study also demonstrated that individuals do not have to witness a homicide directly to develop PTSD. Although only 6% of the survivors of homicide witnessed the homicide, 19% of survivors developed PTSD. Murphy et al. (1999) found that two years after the death of a child, 21% of mothers and 14% of fathers met diagnostic criteria for PTSD, compared to an estimated lifetime prevalence of 7.8% of PTSD nationally (Kessler et al., 1995).

As to PTSD in the security forces, a study conducted in Johannesburg found that 49% of active members of an Internal Stability Unit of the South African Police Services (SAPS) met criteria for PTSD (Kopel & Friedman, 1997). Applications for retirement from the South African security forces on medical grounds increased alarmingly in later years (Emsley & Coetzer, 1996), and PTSD is one of the most common diagnoses among these individuals. Emsley, Seedat, and Stein (2003) also conducted a study with 124 members of the South African Police Services and Correctional Services who were declared permanently medically disabled due to PTSD, and found a prevalence rate of 18% for PTSD.

2.6.2 Other groups

Although women are exposed to proportionally fewer traumatic events in their lifetime than men, they have a higher lifetime risk of PTSD (Resnick et al., 1993). The 1996 Detroit Area Survey documented a nearly two-fold higher lifetime rate of PTSD in women (18.3%) than in men (10.2%) (Breslau et al., 1998). An epidemiological study in Australia, with a sample of 10 641 participants, documented the twelve-month prevalence of PTSD to be higher in women (3.8%) than in men (2.0%) (Rosenman, 2002). In all the studies that included both males and females, exposure to trauma is lower in females than in males. However, a 2:1 ratio of female-to-male lifetime prevalence of PTSD is typically reported. (Breslau. 2001a; Breslau, et al., 1991; Kessler et al., 1995).

Adolescence is commonly recognized as a stressful period. Apart from the vulnerability to stress attributed to developmental changes, it is often exacerbated by increased levels of divorce and single-parent families, increasing rates of poverty, homelessness, drug abuse, family violence, child abuse, and exposure to violent crime. In a study by Berton and Stabb (1996) of 97 high school juniors who were exposed to violence, 25% presented with clinical PTSD.

A few studies have reported rates of PTSD found in medical settings with special characteristics. The following percentages were found for inner-city young adults: 50% (Leskin, Ruzek, Friedman, & Gusman, 1999), a poor rural African town: 20% (Carey, Stein, Zungu-Dirwayi, & Seedat, 2003), the Gaza-strip: 29% (Afana, Dalgard, Bjertness, Grunfeld, & Hauff, 2002), and patients seeking alternative medical treatment in the U.K. and the U.S.A.: 6% (Davidson et al., 1998).

A study in a South African township primary health care clinic on the prevalence of trauma exposure and PTSD (Carey et al., 2003) showed a current diagnosis of PTSD in 19.9% of the sample. A lifetime diagnosis of PTSD was made for 45.5% of women and 42,3% of the men.

Relatively little is known about the extent and consequences of traumatic exposure in older adults (Cook, 2001). Though some studies showed symptom stability over decades (Engdahl, Harkness, Eberly, Page, & Bielinski, 1993), more recent findings suggested that PTSD symptoms are highest after exposure, decline for years, and increase in later life. Only a few studies have been undertaken on the prevalence and effects on older individuals of several types of trauma, such as criminal victimization or elder abuse, neglect, or exploitation (McCabe & Gregory, 1998; Pillemer & Finkelhor, 1988). To a large extent the psychological consequences of abuse, most notably PTSD, in older adults remain unstudied (Comijs, Penninx, Knipscheer, & Van Tilburg, 1999).

Very few studies on the prevalence of PTSD in children are available. Prevalence and incidence rates from studies of at-risk children have varied widely, ranging from 0% to 100% (Pynoos, Frederick, & Nader, 1987; Schwarz & Kowalski, 1991; Yule, Udwin, & Murdock, 1990). Recent findings suggested an incidence of PTSD of approximately 20% in children who experienced road traffic accidents, and an incidence rate of 10% to 12% in children who had been admitted to hospital casualty wards in the UK following common childhood mishaps such as falling off walls and other high places (Heptinstall, 1996; Yule, 2000).

2.7 MEMORY AND POSTTRAUMATIC STRESS DISORDER.

Re-experiencing symptoms refer to repeated *involuntary intrusive memories*, involving intense sensory and visual memories of the event, at times accompanied by extreme physiological and psychological distress, and which may be brought on by real or symbolic stimuli, or may be spontaneous recurrences. These memories may present with a dissociative quality which is characteristic of flashbacks and dreams. Several authors (Ehlers et al., 2004; McFarlane, 2002; Van der Kolk & Van der Hart, 1991) consider intrusive re-experiencing as a core symptom of PTSD, signifying PTSD as a disorder of memory, in which the traumatic experience is not normally integrated. This had resulted in a strong research focus on the role of memory in PTSD.

Theorists agree that re-experiencing symptoms are the result of the way in which trauma memories are encoded, organised in memory, and retrieved (Brewin, Dalgleish, & Joseph, 1996; Conway & Pleydell-Pearce, 2000; Ehlers & Clark, 2000; Foa & Rothbaum, 1998; Foa, Steketee, & Rothbaum, 1989; Keane, Zimmerling, & Caddell, 1985; Van der Kolk & Fisler, 1995). The exact nature of the core features of trauma memories has however been the object of considerable debate (Ehlers et al., 2004). The qualities of intrusive re-experiencing can be examined from different angles, the first being the question of *thoughts versus sensation.* In the early literature on PTSD it was not uncommon to describe intrusive memories as intrusive thoughts (Ehlers et al., 2004). Since then research has suggested that intrusive (unwanted, spontaneously triggered) memories consist mainly of relatively brief *sensory* fragments of the actual traumatic experience (Ehlers & Steil, 1995; Mellman & Davis, 1985; Van der Kolk & Fisler, 1995). Regardless of the type of trauma, research has shown visual sensations to be the most common type of intrusive re-experiencing, followed by other sensory impressions such as bodily sensations, smells, sounds and tastes. It is not uncommon for the intrusive memories to consist of several sensory components (Ehlers & Steil, 1995; Ehlers et al., 2002; Hackmann, Ehlers, Speckens, & Clark, 2004). Intrusive memories should be distinguished from other non-memory cognitions which may also be experienced intrusively, but which are functionally different (Ehlers & Clark, 2000; Joseph, Williams, & Yule, 1997). These non-memory cognitions, which according to Reynolds and Brewin (1998, 1999) may even be more frequent than memories or flashbacks, may include evaluative thoughts and questions, or reasonings on how one's life has been ruined by the trauma (Hackmann et al., 2004; Murray, Ehlers, & Mayou, 2002)

An additional factor is the *lack of time perspective*. Memories of specific autobiographical events are usually classified as episodic memories. This concept is used to describe a memory system that makes it possible to aquire and retrieve information about specific experiences that occurred at a particular time and place. Retrieval from episodic memory is unique because it involves the sense or experience of the self in the past – autonoetic awareness (Ehlers et al., 2004). One of the defining features of episodic memory, i.e. the awareness that the content of the memory is something from the past, appears to be lacking from intrusive re-experiencing symptoms in PTSD. In an intrusive dissociative flashback the person literally appears to relive the experience, losing all awareness of his/her present surroundings. The sensory impressions, rather than being aspects of memories from a past episode, are re-experienced as if they are happening right now. Hackmann et al. (2004) described these intrusive memories as appearing to the patient as if they were happening in the 'here and now'. The sense of 'nowness' may not be restricted to flashback experiences, but may also apply to briefer intrusive memories that do not involve loss of awareness of present surroundings. The vivid nature of the intrusive memories has also been pointed out by Brewin et al. (1996). The accompanying emotions are also the same as those original emotions experienced at the time of the trauma (Brewin et al., 1996; Ehlers & Clark, 2000; Foa & Rothbaum, 1998).

Van der Kolk and Van der Hart (1991) pointed out that intrusive memories are relatively resistant to change and show a typical *lack of context*. Ehlers and Clark (2000) noted that PTSD patients often re-experience their original sensory impressions and emotions in spite of the fact that they later acquire information which contradicts the original impression. Patients may even experience two contradicting intrusive flashbacks without any change in the contents of either of the two.

Research by Ehlers and Clark (2000) showed that these memories do not appear to be random fragments but that they mainly represent stimuli that signalled the onset of the trauma or the moments with the largest emotional impact. The intrusive memories can therefore be understood as stimuli that, through *temporal* association with the traumatic event, acquired the status of warning signals: stimuli that, if encountered again, would indicate impending danger (Ehlers et al., 2002). This might explain why intrusive memories induce a sense of serious current threat. Previous research has shown that central elements of highly emotional experiences are remembered best (Christianson, 1992), and on this basis it could have been expected that trauma victims would have intrusive memories of the most

distressing moments of their ordeal, rather than for example the face of an assailant just *before* the actual assault. The 'warning' stimuli seem to consist of markers of the situational context in which the trauma occurred, and markers of location may be early warning signals that can be spotted from far away and therefore can be avoided in future (Ehlers et al., 2004). A notable finding by Hackmann et al. (2004) was that patients experienced a small number of involuntary intrusive memories of a traumatic event, and that these occurred in a very repetitive way.

A wide range of triggers of involuntary re-experiencing have been reported (Brewin et al., 1996; Ehlers & Clark, 2000; Foa, Rothbaum, & Kozak, 1989). Many of the trigger stimuli are cues without a strong or meaningful relationship to the traumatic event, but may instead simply be cues that were temporally associated with the traumatic event (Charney, Deutch, Krystal, Southwick, & Davis, 1993; Keane et al., 1985). These could for example be physical cues similar to those present just before or during the trauma.

Most trauma survivors remember the essence of what happened but show confusion about the exact temporal order of the events, and are often unable to access some details. The question remains why persons with PTSD have persistent re-experiencing symptoms, and which aspects of the trauma memories explain them.

Different aspects of what victims recall of the experienced trauma may contribute to the difficulty often experienced in appraisal of the traumatic event. Confusion about the time course of events, problems in accessing important details of the event, and problematic recall resulting from encoding errors at the time of the event may all influence the appraisal. Confusion about the temporal order of an event may crucially affect its real meaning as well as its implications for the future (Ehlers et al., 2004).

To explain re-experiencing symptoms, Ehlers and Clark (2000) built on research on *non-trauma autobiographical memories.* Despite an abundance of retrieval cues, individuals are as a rule not flooded by involuntary memories in their day-to-day lives. According to Conway and Pleydell-Pearce (2000) the reason for this is that autobiographical events are normally elaborated and incorporated into an autobiographical memory knowledge base. This elaboration *enhances* the ease of intentional retrieval through higher-order meaning-based retrieval strategies, and *inhibits* cued retrieval through

direct triggering by stimuli associated with the event (Conway & Playdell-Pearce, 2000; Markowitsch, 1995). Consequently, when an autobiographical memory enters consciousness, it comprises both specific information about the event as well as context information. In PTSD, trauma memories are not fully elaborated in this way (Brewin et al., 1996; Ehlers & Clark, 2000; Foa, Steketee, et al., 1989; Rachman, 1980), and they are not adequately integrated into their context in time, place and relevant previous information. This explains the absence of context in time and the absence of links to subsequent information. As the inhibitory effect of elaboration is lacking, it also contributes to the triggering of intrusive memories by matching cues.

A view gaining in popularity is that forgetting trauma needs to be explained by mechanisms different from those of the ordinary memory process. Growing interest has for instance been focused on the ways in which trauma seems to alter a number of physical systems. A number of studies traced links between neuro-anatomical and neuro-chemical correlates of trauma and their impact on memory (Bremner, 1999; Bremner, Southwick, & Charney, 1999). Research has suggested that the link between extreme and/or chronic stress and impaired memory performance occurs through the release of the stress hormone cortisone which may have a deleterious effect on the hippocampus (Bremner, Scott, et al., 1993; De Bellis, 2001). Studies of PTSD patients have repeatedly shown deficits in neuro-psychological performance (Golier & Yehuda, 2002). According to Nixon, Nishith, and Resick (2004) a relatively constant finding is that of impaired performance on verbal learning as well as short-term and delayed recall memory tasks in civilian PTSD patients (Jenkins et al., 1998), combat survivors with PTSD (Vasterling et al., 2002), and Holocaust survisors with PTSD (Golier et al., 2002). A number of studies, however, did not demonstrate such deficits (e.g., Koenen et al., 2001; Stein, Kennedy, & Twamley, 2002).

According to Neylan et al. (2004) decreased performance on neuro-cognitive tasks may be of particular relevance to PTSD because several studies have documented decreases in hippocampal volume (Bremner et al., 1995; Bremner et al., 1997; Gilbertson et al., 2002; Gurvits et al., 1996; Stein et al., 1997) and reduced concentrations of the neuronal marker *N*-acetal aspartate (NAA) (Freeman, Cardwell, Karson, & Komoroski, 1998; Schuff et al., 2001). In a sample of combat veterans, Bremner et al. (1995) found that hippocampal atrophy in PTSD was associated with decreased function in

explicit memory. However, Stein et al. (1997) did not find this association in a study with women who had a history of childhood sexual assault.

Ehlers and Clark (2000) suggested that two other memory processes are involved in the easy triggering of re-experiencing symptoms: the wide range of triggers, and the affect-without-recollection phenomenon in PTSD. They identified these two processes as perceptual priming (enhanced ability to identify objects as a result of prior encounter) and associative learning (Charney et al., 1993; Keane et al., 1985;). These two processes underlie expectations about what stimuli the person will encounter (priming) and what will happen next (associative learning). The combination makes it likely that the individual will notice external (visual or auditory cues) or internal (posture, feelings, arousal) stimuli that are trauma reminders, and respond to them with automatically triggered re-experiencing symptoms (Ehlers et al., 2004). Ehlers and Clark (2000) concluded that PTSD sufferers have strong perceptual priming for stimuli they encountered shortly before and during the trauma. Due to the processing advantage and reduced perceptual threshold for these stimuli, cues associated with the trauma that directly trigger memories of the event are more likely to be noticed because the inhibitory effect of elaboration/integration into context, is lacking. Because implicit memory traces are not well discriminated from other memory traces (Baddeley, 1997), vague physical similarity would be sufficient for the perception of stimuli as being similar to those present during the trauma (poor stimulus discrimination) and for the triggering of intrusive flashbacks, even if the context in which the stimulus is observed differs greatly. Within this framework, re-experiencing symptoms can be seen as resulting from processes that warn the individual of impending danger. Perceptual priming sets up the expectation that the individual may again encounter a stimulus configuration similar to the one encountered just before or at the onset of the trauma or at the onset of its worst moments. Associative learning informs the individual what will likely happen next and trigger the corresponding emotional responses to activate a behavioural response (Ehlers et al., 2004).

Psychological factors may differentiate traumatic memories from others. Ward and Carrol (1997), Safer, Christianson, Autry, and Osterlund (1998) and Mechanic, Resick, and Griffin (1998) discussed ways in which trauma may lead to differences in the cognitive processing of events, while Van der Kolk and Fisler (1995) presented preliminary findings suggesting that traumatic memories may be stored in sensory-motor form rather than narrative form, therefore making them less accessible by

means of conventional memory retrieval strategies. There are several obstacles to the collection of valid trauma histories (Krinsley, Gallagher, Weathers, Kutter, & Kaloupek, 2003). These obstacles or challenges include (a) labeling and issues of self-definition, such as a woman not calling forced sex "rape" because she knew her assailant; (b) reporting reluctance attached to shame and stigma (Della Femina, Yeager, & Lewis, 1990, Kilpatrick, 1983; Mollica & Caspi-Yavin, 1991). Inconsistent reporting has occurred, especially increased reporting of traumatic events during repeated assessments (Arnow et al., 1999; Goodman, Corcoran, Turner, Yuan, & Green, 1998; Leserman, Drossman, & Li, 1995; Martin, Anderson, Romans, Mullen, & O'Shea, 1993; Roemer, Litz, Orsillo, Ehlich, & Friedman, 1998; Southwick, Morgan, Nicolaou, & Charney, 1997; Wyshak, 1994). These increases have been explained as underreporting at the initial assessment (Leserman et al., 1995), and additional lower magnitude stressor events being reported at later assessments (Goodman et al., 1998; Martin et al., 1993). Evidence that increased reporting of traumatic events was accompanied by increased reporting of PTSD symptoms, came from two studies (Roemer et al., 1998; Southwick et al., 1997). This effect may be similar to state-dependent memory retrieval as described by Bower (1981). These findings are consistent with the view that personal assessment of trauma history is a reactive process that can affect memory retrieval and reporting (Krinsley et al., 2003). Memory consistency was also found to be lower, although substantial when evaluated in relation to narrower, self-identified 'worst' traumatic events (Krinsley et al., 2003).

Cahill (1997) and Cahill and McGaugh (1995) reported laboratory findings demonstrating the ability of emotional arousal to enhance memory of stressful events. A study by Krinsley et al. (2003) showed that directly experienced events were reported more consistently than events experienced from the perspective of a witness.

When PTSD patients initially relive the trauma in treatment, they appear to retrieve it in a disjointed order and in separate parts and not as segments of an integrated memory. Ehlers et al. (2004) observed that when PTSD patients remember a particularly distressing segment of the trauma (a 'hotspot') they do not access other subsequent and relevant information that actually corrected the impressions they had or predictions (including a 'felt sense') they made at the time.

In a study of survivors of an oilrig disaster Holen (1991) found that dissociation during the trauma was a distinctive predictor of PTSD. Similar results were found by Koopman et al. (1994) in survivors of the Oakland Hills firestorm. The ability of peri-traumatic dissociation to predict posttrauma pathology have been demonstrated in studies of female Vietnam veterans (Tichenor et al., 1996), emergency workers (Marmar et al., 1999), and hospitalized trauma victims in Jerusalem (Shalev et al., 1996). Krystal, Southwick and Charney (1995) suggested that peri-traumatic dissociation may lead to bizarre and distorted memories reflecting altered perception at the time the memory was formed. They quoted examples suggesting that changes in sensory domains are accompanied by changes in the critical features of the trauma context. Other theorists (Gelinas, 1983; Terr, 1991) supported this 'dissociative encoding style'. Foa and Kozak (1986) and Foa and Riggs (1993) suggested that persistent emotional disturbances, such as PTSD, may be indicative of inadequate processing of the trauma memory, and proposed that the recovery process must involve the organizing and streamlining of these memories (i.e., the organization/articulation hypothesis). Peri-traumatic dissociation may produce disorganized and fragmented memories that resist streamlining and organizing. Through analisysis of trauma narratives, Amir, Stafford, Freshman, and Foa (1998) found that the degree of articulation in the narratives of recent assault victims was inversely related to the severity of PTSD three months later. Foa, Molnar, and Cashman (1995) also used trauma narratives to examine changes between the first and last recounting of traumatic memories during exposure therapy. They found that an increase in organized thoughts was related to a decrease in depression, while a decrease in fragmentation was related to a reduction in PTSD. These results suggested that disorganization and fragmentation of memories due to peritraumatic dissociation, are related to subsequent post-trauma pathology.

Holmes et al. (2005) proposed two qualitatively distinct forms of dissociation: detachment and compartmentalization. According to DePrince, Chu, and Visvanathan (2006) detachment includes experiences of disconnection from the self or environment, such as depersonalization, derealization, and out-of-body experiences. Compartmentalization includes dissociative amnesia and some unexplained neurological symptoms, such as conversion paralysis.

Much of the literature on PTSD and dissociation focused on dissociation at the time of the traumatic experience (per-traumatic dissociation) as a predictor of PTSD. A meta-analysis of 68 studies by Ozer, Best, Lipsey, and Weiss (2003) revealed a medium contributing effect of peri-traumatic dissociation to later PTSD symptoms. In spite of the limitations of retrospective reports, studies have demonstrated

relationships between peri-traumatic dissociation and later PTSD symptoms (Shalev et al., 1996). Dissociation probably serves adaptive and maladaptive functions at the same time, depending on the context (DePrince et al., 2006). Van der Hart, Nijenhuis, and Steele (2005) argued that structural dissociation serves a defensive function merely to the extent that the person lacks integrative capacity regarding the trauma and that, as integrative capacity increases, the usefulness of the structural dissociation as defense mechanism decreases.

Gershuny, Cloitre, and Otto (2003) reported that the relationship between peri-traumatic dissociation and later PTSD symptomatology was mediated by fears of death and loss of control during the trauma, which are central cognitive components of panic. These findings raised the possibility that peri-traumatic dissociation may be related to panic and not primarily to pathological dissociation. In a follow-up study Gershuny, Najvitz, Wood, and Hepner (2004) found that dissociation mediated links between trauma and later psychopathology, including PTSD symptoms. Feeny, Zoellner, Fitzgibbons, and Foa (2000), in a study of 160 female vicrtims of sexual assault, distinguished dissociation from emotional numbing and depression in predicting PTSD. This data provided evidence that emotional numbing and dissociation are separate constructs and that numbing rather than dissociation is predictive of later PTSD.

2.8 POSTTRAUMATIC STRESS DISORDER IN CULTURAL CONTEXT

Although the contemporary concept of PTSD is largely a product of Euro-American history and culture, epidemiological research showed that traumatic stress can be found among a variety of populations with different cultures and political and religious systems (Hinton et al., 1993; Kroll et al., 1989; Mollica, Wyshak, & Lavelle, 1987; Westermeyer, Bouafuely, Neider, & Callies, 1989). There is, however, a growing literature on ethno-cultural aspects of mental disorders in general and PTSD in particular (Chemtob, 1996; Marsella, Friedman, Gerrity, & Scurfield, 1996). However, there is little methodologically sound research on transcultural aspects of traumatic stress and PTSD (Marsella, Friedman, & Spain, 1996).

Research on PTSD in different cultures raised questions such as: Is the interpretation of trauma the same in different cultures? Do individuals from different cultures react differently to trauma? Do different cultures attach different types of stigma to certain types of trauma, e.g., rape or sexual assault? How do different cultural groups support (or reject) victims of trauma? The answers to these

and other questions may determine whether the diagnostic nomenclature is appropriate to divergent cultures, and whether the terminology and diagnoses of PTSD, as known in Western cultures, can be transposed to non-Western cultures.

The concept of suffering and illness takes on different meanings in different cultures. Anthropologists have documented how, in non-Western societies, misfortune and illness are attributed to the intrusion of evil spirits, the magic of sorcerers or witches, the breach of taboos, the loss of one's soul, or the intrusion of an object. These five major traditional explanations of illness and suffering can be found around the world (Last, 1993). Attempts to explain non-Western therapeutic strategies with the vocabulary of Western psychology remain controversial. Translation of therapies based on local cultural concepts into the language of psychology has been described as complicated, imperfect, impossible, or even inappropriate (Cantlie, 1994; Kakar, 1985).

In all cultures the ways in which suffering and illness are presented publicly play an important role in the moral fabric of a community and bring about a recognition of specific sources of morality. With the concept of PTSD in mind one must try to draw the line between this Euro-American description of suffering and a locus of moral authority. In the original cultures the bewitched and possessed were threatened by evil forces coming from outside the self, over which one had very little control and for which one did not feel morally responsible. Today, the locus of moral responsibility for PTSD victims is still shifted away from the self, but the prevailing moral order has become secular. Witches, sorcerers, and priests are no longer involved. Now, in Western-orientated societies, the secular institutions of the state and civil society create the moral order (Argenti-Pillen, 2000). In the aftermath of extreme events, such as traumatic experiences, people now tend to borrow rapidly from other, presently available worldviews to repair their impoverished and eroded cultural systems of meaning, morality, and dignity (Nordstrom, 1992).

It is against the background of this cultural void or crossover, that the Euro-American concept of PTSD must be evaluated. Some authors have concluded that all humans are similar with respect to their capacity to experience and express a number of fundamental emotions, such as joy, fear, anger, sadness, disgust, shame and guilt (Scherer & Wallbott, 1994). Russell (1994) questioned this fundamental hypothesis of universality, while Lutz (1988) saw emotions as cultural constructs. Matsumoto (1989) argued that fear is expressed and perceived universally, but the expression of other

emotions may be modified by cultural influences. Stamm and Friedman (2000) believed that all humans have the capacity to experience and express fear, helplessness or horror, when exposed to traumatic stress, and argued further that PTSD intrusion (Criterion B) and arousal (Criterion D) symptoms may be universal posttraumatic indications of the psycho-biological evolutionary process, whereas expressions of avoidance/numbing (Criterion C) symptoms may be determined more by cultural than by universal factors (Friedman & Marsella, 1996).

Draguns (1994) maintained that mental illness is a mixture of universal and cultural-specific factors. In line with this perception, the DSM-IV (APA, 2000) recommends that ethno-cultural factors should be carefully considered in any diagnostic assessment. Such factors include the individual's cultural identity, cultural explanations of the individual's illness, cultural factors related to the psychosocial environment and levels of functioning, cultural elements of the relationship between the individual and the clinician, and overall cultural assessment for diagnosis and care.

The individualism–collectivism dichotomy (Keats, Munro, & Mann, 1989) represents another way to characterize differences in culture. People from traditional cultures are often collectivists who perceive the self as part of a larger whole (family, community, or tribe). They are primarily concerned with the effects of their decisions on others. They share material and non-material resources, and are willing to accept the opinions of others. A good example of collectivism is the Zulu concept of 'ubuntu' ("I am because we are"). In contrast, individualists are motivated by their personal preferences, needs, and rights, placing a higher priority on personal than group goals. Rosenthal and Feldman (1992) stated that a person's position on the individualism–collectivism spectrum has important implications for the assessment and treatment of PTSD and other psychiatric syndromes. Western mental health clinicians generally focus on what Marsella et al. (1996) called 'personal uniqueness'. However, where the family or a small family group is a more appropriate unit for treatment, the clinician may have to address a more culturally traditional 'we-self' rather than the traditional Western 'I-self' for therapy to be relevant, accessible and successful (Roland, 1996).

Two major components of post-traumatic distress identified in non-Western people are somatization and dissociation (Jenkins, 1991; Kirmayer, 1996). Dissociation has been identified as a major component of posttraumatic distress in Turkish survivors of childhood sexual abuse (Sar, Yargic, & Tutkun, 1996), Japanese women who had a history of sexual abuse (Berger, Saito, Ono, & Tezuka, 1994), and Cambodian refugees (Carlson & Rosser-Hogan, 1991, 1993). Yet, dissociation is the focus

of only one criterium system (DSM-IV A3: psychogenic amnesia), and an individual can meet the diagnostic criteria for PTSD without any evidence of dissociation (Stamm & Friedman, 2000). Somatization is not included in the PTSD diagnostic criteria, yet Marsella et al. (1996) pointed out that somatic symptoms may serve as an idiom of posttraumatic distress for cultures or subcultures that deny or re-interpret psychological dysfunction. This will not only influence the diagnosis, but also the treatment of PTSD in non-Western cultures. It was found that among Ugandan victims of government counter-insurgency forces, somatic problems were the most overt manifestations of distress, rather than other symptoms typically associated with trauma (Giller, Bracken, & Kabaganda, 1991). Eagle (2002) also pointed out that, given the integrative worldview of African culture in which the psyche-soma or body-mind splits are absent, it is not surprising that many African people present with somatic symptoms such as back and chest pains or feelings of faintness or dizziness after experiencing a traumatic event.

There is growing empirical evidence that people from traditional cultures experience somatic distress following extremely stressful events (Hough, Canino, Abueg, & Gusman, 1996; Jenkins, 1991; Kirmayer, 1996; Robin, Chester, & Goldman, 1996). There is also evidence that PTSD may be a risk factor for medical illnesses (Friedman & Schnurr, 1995). Consequently, physical symptoms are often an important component of post-exposure distress in traditional cultures. Among Asian and Central American refugees, there is an important association of physical health complaints and psychological symptoms, including post-traumatic distress (Palinkas, 1995). Among indigenous people in North America, it is generally believed that illnes can occur as a result of misfortune (Joe, 1994), and Kirmayer (1996) conceptualized traumatic stress as a socio-political and psycho-physiological experience that has an explanation and a narrative theme with cultural and sociopolitical variations.

Although numerous studies have been conducted on the prevalence of trauma exposure and resultant PTSD, the extent to which these findings are generalizable across diverse cultures and societies is unclear. Much of what is known about violence comes from research in countries with Anglo-European traditions, such as the USA. Very little is known about reactions to violence in countries such as Mexico, whose culture and histories are very different from that of the United States. Before conducting an epidemiological study on the prevalence of PTSD in Mexico, Baker et al. (2005) undertook preliminary research to determine whether PTSD was a relevant construct for Mexican trauma survivors. Results from qualitative interviews determined that Mexican respondents mentioned

14 of the 17 specific PTSD criterion symptoms with very little or no prompting (Norris, Weishaar, et al, 2001). In another quantitative study conducted with samples from the United States and Mexico, a four-factor measurement model representing the accepted multi-criterion conceptualization of PTSD fit the data of the US and Mexican samples equally well (Norris, Perilla, & Murphy, 2001). Both studies implied that PTSD is a relevant and measurable construct in Mexico.

In their subsequent study Baker et al. (2005) found that a large percentage of Mexicans had experienced violence at least once during their lives, the most frequent types of violence being physical assault and threat by weapon, with slightly lower, but not trivial, prevalence rates of sexual assault and molestation. Almost all forms of violence were associated with a moderate to high probability of developing PTSD, but victims of sexual and domestic violence were found to be particularly vulnerable. The differences which were found between the sexes and between types of violence, largely conformed to findings from previous studies in the United States (Breslau, et al. 1998; Kessler, et al., 1995; Sorenson & Siegel, 1992) and Canada (Stein et al., 1997), suggesting that there may be universal patterns in the sex distribution of violence cross-culturally. The data of this study also suggested that female participants may have been underreporting violence and this finding is consistent with previous studies with women of Mexican ancestry, some of whom were US born and others Mexican born (Sorenson & Telles, 1991). Baker et al. (2005) argued that Mexican women born in Mexico may be more traditional than those born in the USA and, therefore, more hesitant to discuss unwanted sexual contact. This was in line with the findings of Lira and Koss (1999) and may reflect on cultural aspects.

A further finding by Baker et al. (2005) was that nearly a quarter of exposed women reported symptoms consistent with PTSD. This rate was more than five times higher than that of the male participants. One explanation for this may be that cognitions related to trauma, such as helplessness and emotional distress, may be more dissonant with men's self-concepts than with women's. This dissonance may be even greater in cultures with more traditional views of men and women. Traditional gender role socialization may therefore cause men in Mexico to suppress symptom experience more than men in the United States (Norris, Perilla, Ibanez, & Murphy, 2001). Conversely, poverty, discrimintion, and oppression have been seen to be related to women's capacity to cope with traumatic stressors (Wolfe & Kimerling, 1997). These socio-economic and socio-cultural explanations may be especially relevant for Mexican women because so many live in poverty, and all live in a culture that

fosters traditional views of men and women (Vasquez-Nuttal, Romero-Garcia, & De Leon, 1987). Therefore, women in Mexico may be more likely to experience and report symptoms after a stressful event than women in the USA. This was partially borne out in one of the only studies that included both immigrant and US-born women of Mexican descent in the same sample (Holman, Silver, & Waitzkin, 2000), which reported that Mexican immigrants reported lower rates of trauma and domestic violence than US-born Latinos did. Heilemann, Kury and Lee (2005), in a study with women of Mexican descent in the USA, stressed that while the variable of generation suggests that longer and more complex exposure to the United States from the time of birth through childhood and/or adolecence increases vulnerability to trauma and/or PTSD, generation alone cannot provide insight into factors such as cultural identity, values related to social or cultural practices, and culturally or socially embedded beliefs that influence daily life among women of Mexican descent. They further pointed out that, although results of their study demonstrated that exposure to general and sexual trauma increased with generation or time spent in the United States, especially in childhood/adolescence, for women of Mexican descent living in the United States, their convenience sample did not represent all women of Mexican descent in the United States, and their findings cannot be generalized to the larger population of Latina women.

South Africa consists of a diversity of cultures. Although most of the Black cultures have major overlapping characteristics, there are also vast differences in basic cultural characteristics. The differences between these cultures on the one hand and the basically Western culture of the white population from European descent raise questions about the applicability of both the diagnosis and treatment of PTSD. The appropriateness of applying nosological systems developed in Western countries (e.g., the DSM-IV-TR and ICD-10) to conceptualize the psychological reactions to trauma in a culturally diverse society such as South Africa, has not been systematically examined.

Research on survivors of torture, both in South Africa and elsewhere in the world, indicated that such experiences are likely to result in serious psychological sequelae with long-term after-effects (Foster, Davis, & Sandler, 1987; Pillay, 2000; Simpson, 1993; Somnier & Genefke, 1986). Torture, as was used for decades by the South African government to suppress Black opposition to its Apartheid policies, meets the criterium of involving 'actual or threatened death or serious injury' or being 'a threat to the physical integrity of a person' which, according to the DSM-IV-TR (APA, 2000), is the first requirement for a diagnosis of PTSD. Furthermore, Kagee and Naidoo (2004) pointed out that a

diagnosis of PTSD has been widely used in South Africa to conceptualize the effects of torture from a psycho-diagnostic perspective. They argued that a focus on the psychiatric symptomatology of torture survivors may ignore the psychological distress experienced by them (Bouwer & Stein, 1998; Sarraj, Punamaki, Salmi, & Summerfield, 1996). Whereas victims of torture in different contexts previously may have seen their experiences in religious, legal, or ideological terms, the focus has recently been mainly psychological, reflecting a 'Western trauma discourse' (Summerfield, 1999). As a result, psychological terminology and terms such as stress, trauma, distress and depression have become part of African languages in many cultural contexts, even where such terms might not have existed previously, e.g., in isiXhosa, a South African black language which has no equivalent for 'depression'. Many accepted models of psychopathology have been critisized for their inaccurate depictions of the needs and experiences of patients in developing countries (Bulhan, 1985; Naidoo, 1996). These criticisms ring true especially when these models have been applied to societies in the midst of, or with a history of political and social unrest (Bracken, Giller, & Summerfield, 1995; Summerfield, 1999). The assumption therefore developed that human rights violations, such as torture, not only cause distress and suffering, but also create psychiatric disturbance, which in turn may victimize and pathologize survivors by framing them as potential psychiatric cases. Therefore, by typifying distress and suffering following a traumatic event as a psychiatric condition, clinicians and researchers unwittingly convey an expectation of continued psychopathology in the aftermath of torture (Kagee & Naidoo, 2004). Existing theories on response to trauma have been tested on samples drawn mainly from Western cultures. Little is therefore known as to how these theoretical formulations account for the experience of South African torture victims, given the unique cultural, political and historical context which will most likely determine the nature and extent of psychological sequelae in this population (Kagee & Naidoo, 2004). The nature of psychological distress which torture survivors in South Africa experienced, is likely to be quite different from that of trauma victims living in comparatively peaceful, economically prosperous and stable countries. Distress among Black South Africans is probably less individualistically constructed and more intimately tied with perceptions of family, community, and social well-being (Swartz, 1998). Consequently, a focus only on overt symptoms elicited in a diagnostic interview or checklist, without obtaining information about the personal meanings, beliefs, and cognitions that survivors may attribute to the experience, represents a uni-dimensional and decontextualized understanding of the experience (Kagee & Naidoo, 2004).

Various pretrauma characteristics such as social and family support (Davidson et al., 1991), spirituality and religious faith, socio-economic status, and education level (Shalev et al., 1996), have been identified in other populations as buffers against the development and maintenance of psychological disturbances following trauma. There is also a strong probability that among political activists, the strength of their commitment to the liberation movement in South Africa may have acted as a buffer to the development of psychological disturbance following their abuse in prison (Dawes, 1990; Punamaki & Suleiman, 1990).

The experience of abuse or torture for political reasons is seldom the only etiological factor in the development of the trauma response. The meaning ascribed to the experience may rather be influenced by proximal and distal factors in the person's experience and personal history. Such factors focus attention on the complexity, resilience, and dynamism of individuals that are often overlooked by a strict focus on overt psychiatric reactions to traumatic events. It is therefore argued that in much of the literature on survivors of torture and abuse, effective and successful engagement with life stressors is often overlooked and focus is concentrated on the endorsement of psychological symptoms (De Jong et al., 2001; Reppesgaard, 1997; Somasundaram, 2001). This does not imply a denial of trauma symptoms, but calls attention to the fact that a nosological emphasis may be inappropriate in conceptualizing the distress of abuse and torture survivors (Kagee & Naidoo, 2004). In addition, some symptoms often serve adaptive functions and help the person to survive (Simpson, 1993). In situations of severe threat and danger, symptoms such as hyperarousal, hypervigilance, enhanced auditory acuity, and restlessness are adaptive rather than pathological (Basoglu & Mineka, 1992). Certain symptoms of psychiatric disturbance, e.g., recurring dreams and nightmares, as specified and normed on Western samples, may have alternative meanings and significance among non-western respondents (Bracken et al., 1995), including Black South African detention survivors (Kagee & Naidoo, 2004). The disparity between functionality and elevated scores on diagnostic checklists, suggested that calls for the routine screening of survivors of human rights violations in South Africa (e.g., Kaminer, Stein, Mbanga, & Zungu-Dirwayi, 2001), based on apparently high prevalence estimates of psychiatric disturbamce among this population, may be misplaced (Kagee & Naidoo, 2004). In a survey of 20 South African respondents who were detained and tortured for political reasons, Kagee (2004) reported that although the main concerns expressed were unrelated to traumatization, participants also indicated that they experienced symptoms of posttraumatic stress. This data suggested that although a too great focus on traumatic responses may be misplaced, it remains important to consider the possibility that former detainees may exhibit symptoms of this nature. Consequently, critique of the trauma discourse as a

Western phenomenon need to be tempered with evidence of the lived reality of psychological sequelae experienced by this population (Kagee, 2004).

Suffering and distress are experienced in a social context and as such are shaped by the meanings and understandings that people apply to these events (Summerfield, 1999). These meanings and understandings are determined by the specific cultural beliefs and background of the society of which an individual forms part. In studies of male Palestinian torture survivors (Sarraj et al., 1996) and of Turkish activists who had been tortured (Paker, Paker, & Yuksel, 1992), secondary economic factors were found to outweigh traditionally Western-accepted criteria in the development of torture related stress, and political activism was found to constitute a buffer between the traumatic experience and the risk for post-torture psychopathology.

Kagee (2004, 2005) conducted two qualitative studies with samples of 20 and 140 Xhosa-speaking South Africans who had been detained for political reasons and who had been tortured on at least one occasion. Participants were recruited by means of snowball sampling (Struwig & Stead, 2001). The results of the studies showed that the major concerns of the participants were somatic problems, economic marginalization, non-clinical emotional distress, and dissatisfaction with the present political dispensation in South Africa. Because responses were not confined to a predetermined structure as would be imposed by a battery of checklists or a structured interview, the demand characteristics of the interviews were minimized. The fact that participants voluntarily expressed symptoms of PTSD suggested that it is likely to be a real concern for individuals who have suffered abuse in detention. Even though symtoms of traumatization were not formally assessed, their expression in this sample suggested that PTSD, as a nosological entity, may indeed be an emic phenomenon in the South African context. Thus PTSD symptoms may not only be rooted in a Western trauma discourse as Summerfield (1999), and Bracken et al. (1995) have suggested. Similar conclusions were reached by Foster et al. (1987), Pillay (2000), Simpson (1993), and Somnier and Genefke (1986).

Some researchers suggested that social, cultural, and political influences are embedded into interventions, therefore making these interventions difficult to use in different settings. (Bracken et al., 1995; Rousseau, Drapeau, & Platt, 1999). Eisenbruch (1991) argued that what is described in the DSM-IV as pathology, may in fact, in some cultures, be a normal response to trauma that needs no professional support. This view holds that it may be presumptuous to extrapolate from Western cultural

concepts to cultures that very often view well-being in terms of social cohesion rather than in terms of one's own personal state. Trauma victims from many cultures may experience apparently adverse psychological states but continue to function at a high level.

Although the role of gender in the prevalence of PTSD has been stressed, very little research on PTSD has taken into account the differing gender roles and gender images in some non-Western cultures and how this factor might influence the reactions (physically, psychologically, and in day-to-day living) of trauma victims. Foa, Steketee, et al. (1989) suggested that gender differences in response to traumatic events may relate to differences in the meaning of a traumatic event. It can be reasoned that the 'meaning' of a traumatic event may differ from one culture to another, and the event could therefore elicit different reactions in different cultures.

Issues such as family structure, time orientation, and social and occupational commitments have often been overlooked by health-care professionals, and may lead to the failure of treatment interventions previously well accepted in other countries (Norris & Alegria, 2005). In Western culture, most PTSD therapy has been managed on a one-to-one basis. In Asian cultures it was found that any intervention based on an individual approach must always be put into the context of the family and community structure, culture, religion, and socio-economics (Boehnlein, 1987). Any psychological intervention in these cultures should be designed primarily to ensure the restoration of families and then help healing and rehabilitation to occur within the restored family unit.

2.9 NEUROBIOLOGY

An early proposal for a neuro-psychological explanation of intrusive memories and associated psycho-physiological arousal in PTSD, came from Kolb (1987). He suggested that PTSD results from excessive traumatic stimulation that overwhelms the capacity to process information efficiently, and argued that such stimulus overload occurs when the capacity to process information signalling threat to life overwhelms the cortical processes concerned with perceptual discrimination and effective adaptive responses for survival.

Based on data from clinical and animal studies, indications are that there are multiple memory systems with quite distinct anatomical localisations and organisation (Nadel & Moscovitch, 1998; Squire,

1992). It was shown that the cerebellum, neocortex, and amygdala all store different types of information, while the hippocampus activates explicit memory, encompassing the retrieval of specific episodes and their contextual framework (Nadel & Moscovitch, 1998). Furthermore, Baddeley, Bueno, and Cahill (2000) and McGaugh (2004) showed that the hippocampus suppresses the fear response in the amygdala when medium stress conditions arise, but when conditions of extreme stress prevail, this pathway is blocked with a resulting response of exaggerated fear. Studies in which functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) were used, showed changes in hippocampal volume in patients suffering from PTSD. An MRI study of 26 Vietnam veterans with PTSD found an 8% reduction in the volume of the right hippocampus, compared to matched control subjects (Bremner et al., 1995). A second study used quantitative volumetric MRI techniques to compare hippocampal volumes in seven Vietnam veterans suffering from PTSD with those of seven veterans without PTSD, and eight normal control subjects (Gurvits et al., 1996). In the veterans with PTSD both the right and left hippocampal volumes were significantly lower than in the two control groups. These differences remained significant after adjustment for whole brain volume, age, and lifetime alcohol consumption. Studies by Bremner et al. (1997) and Stein et al. (1997) also reported a reduction in hippocampal volume in adult patients with PTSD related to childhood sexual or physical abuse, compared to control subjects. Kitayama, Vaccarino, and Kuther (2005) conducted a meta-analysis of nine studies measuring hippocampal volume in PTSD and found a significant reduction in both right and left hippocampal volume in 133 adults with PTSD compared to 148 healthy control subjects. These findings suggested that PTSD may be associated with damage to the hippocampus, resulting in deficient explicit memory functioning. However, Gilbertson et al. (2002) warned that it is important to recognise that the relationship between PTSD and hippocampal volume may not be causal, but that hippocampal damage may possibly be present before the onset of PTSD, and that this may perhaps in some way be a predisposing condition for the development of PTSD.

Several studies measured activation of different brain areas in PTSD patients following exposure to different kinds of trauma-related or neutral stimuli (Bremner et al., 1999; Kosslyn, Alpert, & Thomson, 1993; Rauch, Van der Kolk, & Fisler 1996; Shin, Kosslyn, & McNally, 1997). The results suggested that PTSD is associated with an increased allostatic load, with prolonged activation of the hypothalamic-pituatary-adrenal axis. A decrease in hippocampal volume might precede or follow this increased allostatic load, resulting in the impairment of explicit memory and perhaps also loss of restraint of fear responses mediated by the amygdala and other components of the limbic system. The

damage to the hippocampus might mean that the individual is unable to form structured contextual memories of the trauma, while de-activation of Broca's area might prevent the individual from developing verbal representations of the trauma (Foa et al., 2006).

From the research to date it is clear that the diverse cortical processes involved in perceptual discrimination and adaptive survival responses, as well as structural abnormalities of the hippocampus which play a crucial role in memory and learning, may all contribute to an individual's responses to percieved danger. Multiple memory systems, organised in distinct anatomical localisations, paired with the preceding or following decrease in hippocampal volume, hamper the formation of explicit and structured memory of the initial trauma. Finally, inability to form a clear verbal account of a trauma may result from functional de-activation of Broca's area in the brain.

2.10 NEUROCHEMICAL SYSTEMS

By studying animals and humans who have adapted well to highly adverse conditions, researchers have recently begun to identify a neurochemical profile of resilient individuals which may help in predicting who will develop psychiatric symptoms in response to traumatic stress, as opposed to who will rebound rapidly or may even derive benefit from their challenging experiences (Southwick, Morgan, Vythilingham, Krystal, & Charney, 2003).

Charney (2004), as well as Charney et al. (1993), found that exposure to traumatic stressors activates arousal responses mediated by the serotonergic and noradrenergic systems which leads to changes in several other neurotransmitter and neuroendocrine systems. During dangerous situations the sympathetic nervous system releases epinephrine and norepinephrine in order to protect the organism. According to Southwick et al. (2003) the magnitude of sympathetic nervous system responses to stress and danger varies from one person to the next. Some people have an unusually robust sympathetic nervous system response to stress and in essence 'over-react'. Unchecked persistent sympathetic nervous system hyper-responsiveness may contribute to chronic anxiety, hypervigilance, fear, intrusive memories, and increased risk for hypertension and cardiovascular disease. Southwick et al. (1999) reported such responses in individuals diagnosed with PTSD. Morgan, Wang, Mason, et al. (2000) and Morgan, Wang, Southwick, et al. (2000) found that, in contrast, individuals with high psychological resilience maintain sympathetic nervous system activation within the range of adaptive elevation,

which is high enough to respond to danger, but not so high as to lead to incapacity, fear and anxiety. A series of studies reviewed by Dienstbier (1989) showed that high chronic levels of epinphrine may be associated with chronic feelings of stress. However, enhanced performance and emotional stability has been associated with a consistent pattern of relatively low baseline epinephrine and strong bursts or spikes in epinephrine during stressful and challenging situations, followed by rapid returns to baseline level. A particular neurochemical which acts to maintain sympathetic nervous system activity within an optimal range or window, is neuropeptide Y. It is an amino acid that is released with norepinephrine when the sympathetic nervous system is strongly activated (Southwick et al., 1999). One of the important actions of neuropeptide Y is to inhibit the continued release of norepinephrine in order to prevent the sympathetic nervous system from 'overshooting'. Preliminary studies in highly resilient Special Forces soldiers have shown that high levels of neuropeptide Y during extreme training stress are associated with higher performance levels (Morgan, Wang, Mason et al., 2000; Morgan, Wang, Southwick et al., 2000). The robust increases in norepinephrine are held back in these soldiers by equally robust increases in neuropeptide Y. Among traumatized combat veterans with chronic PTSD, however, resting and stress-induced levels of neuropeptide Y have been reported as low, compared to controls (Rasmussen et al., 2000). When the sympathetic nervous system is provoked or stressed in veterans with PTSD, they also experience an increase in norepinephrine but the accompanying release of neuropeptide Y appears to be insufficient to hold the rising levels of norepinephrine in check. It is likely that rapid increases in norepinephrine contribute to exaggerated increases in heart rate, blood pressure, respiratory rate, anxiety, panic, vigilance, and even intrusive combat-related memories (Southwick et al., 1999). Neuropeptide Y therefore appears to be a neurobiological resilience factor that helps to maintain sympathetic nervous system reactivity at an optimal level. Heilig, Koob, Ekman and Britton (1994) showed that neuropeptide Y has anxiolytic effects in animals, and Husum and Mathe (2002) demonstrated that antidepressant drugs increased neuropeptide Y in depressed patients with low levels of neuropeptide Y.

Galanin, a peptide, is involved in pain control, cardiovascular regulation, food intake, neuro-endocrine control, memory and learning, as well as anxiety. A high percentage of noradrenergic neurons in the locus coeruleus co-expresses galanin. Galanin is preferentially released under conditions of high norepinephrine activity. It reduces the firing rate of the locus coeruleus. Bing, Moller, Engel, Soderpalm and Heilig (1993) and Moller, Sommer, Thorsell and Heilig (1999) demonstrated that central administration of galanin modulates anxiety behaviour in rats and, when it is injected directly

into the amygdala, it blocks the anxiogenic effects of stress, which is associated with increased norepinephrine release in the amygdala. Galanin, like neuropeptide Y, therefore appears to modulate the behavioural effects of stress-induced increases of norepinephrine, with the overall behavioural effects of norepinephrine hyperactivity depending on the balance between norepinephrine, neuropeptide Y, and galanin.

The hypothalamic-pituatary-adrenal axis, like the sympathetic nervous system, plays a central role in the stress response. Stress stimulates the release of corticotrophin-releasing hormone which in turn activates the adreno-corticotrophic hormone. Adreno-corticotrophic hormone in turn increases the release of cortisol and dehydro-epiandrosterone. Cortisol then mobilises energy stores and increases arousal, vigilance, selective attention, and consolidation of memory. At the same time, cortisol inhibits growth, contains the immune response, and has regulatory effects on brain regions that are important to the stress response (Yehuda, 2003). Chronically elevated corticotrophin-releasing hormone in the cerebrospinal fluid has been linked with both major depression and PTSD in humans. Extreme elevations of cortisol in animals have been shown to have toxic effects on the body and brain. These effects include neural degeneration of the hippocampus, which is involved in memory and learning (Bremner, Narayan, et al., 1999). Dehydro-epiandrosterone, which is secreted in conjunction with cortisol, helps to modulate the effects of cortisol. Under conditions of extreme stress, this modulation may serve a protective role (Kimonides, Khatibi, Svendsen, Sofroniew, & Herbert, 1998). There are indications that the capacity to restrain initial and chronic corticotrophin-releasing hormone responses to stress may be associated with psycho-biological resilience, and that dehydro-epiandrosterone may limit the toxic effects of excess in stress-induced cortisol. Regulation of the relative contribution of corticotrophin-releasing hormone 1 and corticotrophin-releasing hormone 2 receptors may also prove to be important in modulating physiological and psychological responses to stress (Bale et al., 2002).

Serotonin, dopamine, benzodiazepine receptor binding, testosterone, and estrogen are other likely neurobiological mediators of stress resilience (Caspi et al., 2003; Hariri et al., 2002; Morgan, Wang, Mason, et al., 2000; Morgan, Wang, Southwick, et al., 2000). Variations of the serotonin transporter gene have been associated with abnormal levels of anxiety, with a propensity to become conditioned to fear, to variations in brain responses to emotional stimuli, and with the likelihood of developing depression in response to life stress (Caspi et al., 2003; Hariri et al., 2002). Studies of chronically stressed animals as well as of humans with excessive fear and anxiety-related behaviours and

symptoms, have demonstrated cortical alterations in benzodiazepine receptor binding. Gamma-aminobutyric acid benzodiazepine receptor density and function may play an important role in stress resilience and vulnerability (Nutt & Malizia, 2001). Komesaroff, Esler and Sudhir (1999) suggested that while estrogen appears to have a beneficial effect in the short term by blunting hypothalamic-pituatary-adrenal axis and noradrenergic responses to stress, there is the possibility however, that long-term elevations of estrogen caused by chronic stress, may enhance hypothalamic-pituatary-adrenal axis responses. A further suggestion is that increased sensitivity to stress in women may partly be related to differences in stress-related gonadal steroid levels and function.

Although these potential mediators of vulnerability to stress and resilience may individually have modest or at times even insignificant effects on health, the additive or combined effect on dysregulations in multiple neurobiological systems may be quite substantial (Friedman, 2002). McEwen and Stellar (1993) described allostatic load as a cumulative measure of physiologic dysregulation in multiple systems Seeman, McEwen, Rowe, and Singer (2001) conducted a study measuring ten biological markers of physiologic regulation, (including serum dehydro-epiandrosterone level, average systolic blood pressure, and 12-hour overnight urinary excretion of epinephrine) and found that none of the markers individually predicted a decline in health status. However, when all ten markers were summarised together in a measure of allostatic load, they successfully predicted health outcome, including new cardiovascular episodes and cognitive decline. If these findings are taken as a guideline, it may imply that resilient individuals may be those with relatively high stress-induced neuropeptide Y, galanin, dehydro-epiandrosterone, and testosterone, as well as relatively low corticotrophin-releasing hormone and low hypothalamic-pituatary-adrenal axis and locus coeruleus / norepinephrine activation. On the other hand, vulnerable individuals would be more likely to achieve relatively high stress-induced increases in estrogen, dopamine, and hypothalamic-pituatary-adrenal axis and locus coeruleus / norepinephrine activation, as well as relatively low stress-induced neuropeptide Y, galanin, dehydro-epiandrosterone, and testosterone (Southwick et al., 2003).

The neural pathways in the brain which regulate reward, motivation, learning responses to fear, memory, and adaptive social behaviour, also play a central role in mediating stress vulnerability, probably through their relationship to specific character traits. Resilient individuals may possibly be able to maintain a positive self-concept, optimism and hopefulness when they are exposed to chronic stress, abuse and neglect, because their neural reward and motivation pathways keep functioning well

even under adverse circumstances. Similarly, other behaviours associated with resilience, such as the ability to bond with a group of individuals, to attract and to use support from others, and to display altruistic behaviour, may be enhanced by optimal functioning of neural reward and motivation pathways (Masten & Coatsworth, 1998). Furthermore, functional interactions among glutamate, N-methyl D-aspertate receptors, dopamine, and dopamine receptors have a great influence on optimal functioning of reward circuits. Environmentally and/or genetically induced abnormalities in these functional interactions could facilitate vulnerability or resistance to anhedonia and hopelessness when having to face stress (Lenox, Gould, & Manji, 2002).

Neural pathways involved in fear conditioning, consolidation of memory, reconsolidation of memory, and extinction, have all been implicated in the pathophysiology of PTSD (Bremner et al., 1999). These regions are likely to be involved in stress vulnerability and resilience. The hypothesis has been posed that stress-resilient individuals, when compared to stress-vulnerable counterparts, are less likely to over-consolidate emotional memories and less likely to over-generalize specific conditioned stimuli to a larger context. They are also more likely to display a greater capacity to reorganize existing memories and to extinguish traumatic memories (Southwick et al., 2003).

It has been estimated that inherited factors contribute up to 32% of the variance of PTSD symptoms above and beyond the contribution of trauma severity (True et al., 1993). It is very likely that genetic variability in neurotransmitters, hormones, neural circuits, and neuropeptides may play a critical role in stress vulnerability and stress resiliance (Southwick et al., 2003).

Southwick et al. (2003) hypothesized that taking neuropeptide Y, particularly by individuals who do not naturally release sufficient amounts, may boost physiological resilience during times of stress. Similarly, the prescription of other potential neurobiological mediators of resilience, such as dehydro-epiandrosterone or testosterone, might improve hardiness in individuals. They argued that some forms of psychotherapy may serve to bolster extinction of the fear-conditioned memories and cortical inhibition of limbic hyper-responsivity so commonly seen in individuals with anxiety disorders.

Both medication and psychotherapy for PTSD may reverse the structural and functional changes in the affected systems, subsequently normalizing the responses to stress. This hypothesis was validated in a study where 11 PTSD patients were assessed by means of single photon emission computed

tomography (SPECT) scans before and after treatment with a selective serotonin reuptake inhibitor (SSRI) (Seedat et al., 2004). Significant deactivation of the left medial temporal cortex was observed following SSRI treatment. There was a significant correlation between reductions in PTSD symptoms and activation of the left paracingulate region (medial prefrontal cortex). These findings suggested that SSRI treatment may eliminate learned responses of fear because it reverses the abnormal regulation of amygdala activity by the medial prefrontal cortex as seen in PTSD (Seedat et al., 2004; Rauch, Shin, & Whalen, 1998).

Although the magnitude of sympathetic nervous system responses to stress and danger varies from one individual to the next, it is apparent that recent research has prompted the identification of a neurochemical profile of resilience which could facilitate future predictions about which individuals may develop psychiatric symptoms after trauma. Sympathetic nervous system hyper-responsiveness was identified in individuals with PTSD. Certain neurochemicals, such as neuropeptide Y, have been identified as acting to maintain a stable sympathetic nervous system. The peptide Galanin has furthermore been shown to modulate the behavioural effects of other neuro-substance increases induced by stress. Other likely neurobiological mediators of stress resilience have been identified as serotonin, dopamine, bensodiazapine receptor binding, testosterone and estrogen. Based on these and other research findings, specific neural pathways have been implicated in the pathophysiology of PTSD. Furthermore it has been shown that both psychotherapy and medication for PTSD may reverse the structural and functional changes in the affected systems, subsequently normalising the response to stress.

2.11 CONCLUSION

Although the concept of a trauma-related disturbance has existed for over a century, the recognition of PTSD as a formal disorder and its official categorization in the diagnostic nomenclature occurred only in 1980, when it was first introduced in the third edition of the DSM (APA, 1980). Official recognition of PTSD as a formal disorder led to a surge of research on PTSD, particularly in areas such as assessment, prevalence, memory, neurobiology and neurochemical systems, risk and protective factors, cultural influences, and treatment. This resulted in a refinement of diagnostic criteria, new theoretical formulations for understanding the disorder, and new treatment procedures.

However, despite these developments, a need for further reasearch in particular areas remains. For example:

- There is still no concensus on the prevalence of PTSD in general community samples or even in specific trauma populations.
- Although progress has been made in identifying possible risk and protective factors for the development of PTSD, further research is needed particularly to establish the predictive power of combinations of risk or protective factors already identified.
- The diagnosis and treatment of PTSD remain controversial, particularly in non-Western cultural contexts, as the bulk of research on PTSD emanated from the USA and Europe. There is a need for more research on how cultural variables impact on the experience of trauma and the treatment of PTSD.
- The concept of memory and especially trauma memory needs further clarification in order to understand the origin and role of intrusive memories in PTSD.

3. TREATMENT OF POSTTRAUMATIC STRESS DISORDER

3.1 INTRODUCTION

A review of the litterature showed that even before PTSD was officially recognized as a psychiatric disorder in 1980, trauma-ralated distress was treated by means of different psychotherapeutic approaches and modalities. Since its official recognition as a psychiatric disorder by the American Psychiatric Association (APA, 1980), the emphasis has largely shifted to studies investigating the effectiveness of psychotherapeutic procedures in the treatment of PTSD, particularly from a cognitive-behavioural perspective.

Several modalities of psychotherapy for the treatment of PTSD have been described in the literature, including psychodynamic therapy (Andrews, 1991; Brom et al., 1989; Horowitz, 1976), hypnotherapy (Brom et al., 1989), eye-movement desensitisation and reprocessing (Boudewyns, Stwertka, Hyer, Albrecht, & Sperr 1993; Jensen, 1994; Pitman et al., 1996; Renfrey, & Spates, 1994; Rothbaum, 1995; Silver, Brooks, & Obenchain, 1995; Vaughan et al., 1994; Wilson, Becker, & Tinker, 1995), systematic desensitization (Brom et al., 1989; Frank et al., 1988; Wolpe, 1958) and exposure (Foa et al., 2005). The objectives of most of these treatments are to create a realistic interpretation of the trauma, to provide an opportunity to react to the trauma memories, to overcome the cognitive and thought avoidance associated with PTSD, to achieve a sense of mastery over the intrusive recalls, and to modulate the patient's affect.

3.2 OVERVIEW OF PSYCHOLOGICAL TREATMENTS

3.2.1 Psychodynamic Treatment

Psychodynamic principles, such as denial, abreaction, and catharsis have often been included in crisis intervention for individuals exposed to trauma (Burgess & Holmstrom, 1974b; Evans, 1978; Fox & Scherl, 1972). However, the psychodynamic theories of trauma and treatment procedures for PTSD have not been widely tested in controlled outcome studies, and most of the available studies are critisized for their methodological shortcomings (Foa & Meadows, 1997).

Horowitz's (1976) theory of trauma represents an example of the psychodynamic approach to PTSD. Based on the concepts of denial, abreaction, catharsis, and the stages of recovering from trauma he developed a treatment for PTSD, which aims at resolving the intraphysic conflict arising from a traumatic experience, rather than the resolution of specific PTSD symptoms such as intrusive thoughts or flashbacks (Foa & Rothbaum, 1998). The traumatized individual must reconcile the traumatic event and its meaning with his or her concept of self and the world (Horowitz, 1976). According to Horowitz a patient's response to trauma varies between the two phases of denial and intrusive symptoms. Avoidance, denial and emotional numbness are the result of defensive overcontrol. When this overcontrol fails, this phase gives way to intrusive thoughts, flashbacks, or nightmares due to failed defense mechanisms. The nature of therapy depends on the phase in which the patient presents, and the primary goal of psychodynamic treatment is the integration of the traumatic experience by means of therapeutic re-experiencing in a supportive environment (Horowitz, 1976). Insight into the conscious and unconscious meaning of symptoms may help the patient master the trauma and restore functioning. Apart from developing a therapeutic alliance and handling transference and counter-transference issues, the therapist has to recognise the patient's history, as well as his/her existential interpretation of the traumatic moment (Horowitz, 1976). The therapist must consider the person's self-image, his strategies for coping, as well as his defence mechanisms against intrapsychic and interpersonal threats. The emphasis is on helping the patient work through the trauma experience by means of gradual re-exposure to the traumatic event and reinterpretation of its meaning (Sherman,1998). Although Horowitz adheres to a different theoretical viewpoint, his treatment includes components similar to those of the cognitive-behavioural treatments. For example, his concepts of 'dosing' of the traumatic experience and of 'encouragement of expression' are similar to exposure techniques.

As indicated, not many treatment outcome studies for PTSD from the psychodynamic perspective have been reported, while available studies yielded conflicting results. Some studies have suggested that psychodynamic treatment may be useful in treating PTSD (Lindy et al., 1983; Scarvalone et al., 1995). Lindy et al. (1983) treated 28 victims of the Beverley Hills Supper Club fire with individual sessions of short-tern psychodynamic psychotherapy. Survivors, defined as those who were present at the scene, rescue workers, relatives of the deceased, and those who identified the bodies, were assessed for trauma-related diagnoses using the DSM-III criteria. Of the initial 30 participants, two did not have any trauma-related diagnoses, and only nine met criteria for PTSD, while the rest received various trauma-related diagnoses (e.g., adjustment disorder) (Foa & Meadows, 1997). Three measures were used to

assess treatment outcome: a self-report symptom checklist (Derogatis, 1983), a therapist-rated target symptoms measure, and an independent global rating of impairment severity. Treatment was manualized and therapists trained and regularly supervised. Although no control group or random assignment were used, survivors who did not request psychotherapy, but agreed to paricipate in the study, served as an untreated comparison group. The treated group revealed only subclinical symptoms of PTSD two years after the trauma, while the non-treated comparison group failed to improve at the same rate. The results suggested that psychodynamic treatment may be useful with trauma victims without significant trauma-related pathology.

Using a quasi-experimental design, Scarvalone et al. (1995) compared interpersonal process group therapy (IPGT), based on psychodynamic principles and procedures, with a waiting-list control condition in a sample of 43 female childhood sexual abuse survivors. A history of abuse was the only specified inclusion criterion for this study. The results indicated that the treatment group improved on a number of measures. Pre-treatment percentages of women meeting PTSD criteria were 91% for the IPGT group and 85% for the control group. At post-treatment, only 39% of the IPGT group and 83% of the controls still met PTSD criteria. On some measures, e.g., a self-report measure of intrusion, the IPGT group showed more symptom reduction than the controls, while on other measures, e.g., for depression and dissociation, both groups showed equal symptom reduction. Due to the lack of blind assessors, it could not be determined to what extent the expectancy for improvement factor was responsible for these positive findings.

On the other hand, Grigsby (1987) found psychodynamic therapy ineffective in treating a traumatized Vietnam veteran. Other inconclusive and/or negative results were reported by Bart (1975), Cryer and Beutler (1980) and Roth, Dye, and Lebowitz (1988), but unfortunately full reports of these studies are not available.

In general, the few existing outcome studies of psychodynamic treatment for PTSD are characterized by methodological shortcomings, such as lack of controls, inadequate assessment of outcomes, and vaguely described treatments. This limints any conclusions on the effectiveness of psychodynamic therapy in the treatment of PTSD. There is consequently a need for more research in this regard.

3.2.2 Group Therapy

Group psychotherapy is described as a treatment in which carefully selected individuals who suffer from the same disorder meet in a group guided by a trained therapist and help one another effect healing. By using a variety of technical procedures and theoretical constructs, the leader directs members' interactions to bring about changes. Compared to individual therapies, two of the main strengths of group therapy are the opportunity for immediate feedback from a patient's peers, and a chance for both patient and therapist to observe a patient's psychological, emotional, and behavioural responses to a variety of people, who elicit a variety of transferences (Kaplan & Sadock, 1998).

Foy et al. (2000) argued that group therapy is particularly appropriate for combat veterans with trauma-related distress. Similarly Walker and Nash (1981) suggested that group therapy is especially indicated in more chronic cases of PTSD.

Depending on its theoretical perspective, group approaches may be classified as 'supportive', 'psychodynamic', or 'cognitive-behavioral'.

Supportive group programs are usually to a large extent unstructured, and are intended to provide supportive interaction between members suffering from the same disorder (at times also including family members). It focuses on members' daily functioning and the group provides guidance in this respect. According to Kaplan and Sadock (1998) supportive groups are particularly indicated for psychotic and anxiety disorders, such as PTSD. Communication centres primarily around environmental factors and positive transference, and intragroup dependence are encouraged. The therapist's aim is to strengthen existing defenses, sometimes to the point of actively giving advice. Socialization outside the group is encouraged so as to improve adaptation to the environment.

No literature on the application of supportive group therapy for PTSD, or its effectiveness, could be identified.

Although psychodynamic groups have been used in the treatment of psychiatric disorders, there is a relative scarcity of litterature on this topic. One exception is a detailed report by Zaidi (1994) on a time-limited psychodynamic pilot group developed for treating adult male abuse survivors with PTSD

in an inpatient setting. Although other psychotherapeutic techniques were incorporated in this treatment program, the treatment concentrated on identifying underlying themes and giving meaning to these themes. For this reason Zaidi (1994) presented it as a psychodynamic group program.

Five male inpatients participated in the pilot group. Group meetings of 90 minutes each were scheduled twice a week for five weeks.

The first session focused on a psycho-educational presentation of prevalence rates and descriptions of various forms of abuse, designed to address the stigmatization associated with abuse, to mitigate self-blame, and to lay the foundation for reframing survivor behaviours as constructive adaptations to adverse experiences. Group members completed a self-drawing and a drawing of a member of the opposite sex. These drawings were used to explore self-concepts, including body image and affect, as well as perceptions of members of the oposit sex.

Session 2 consisted of members completing simple genograms which they then shared with the group, followed by discussions of family relationships, cultural background, family roles, expectations, and cross-generational patterns. Patients were then instructed in relaxation techniques, including a graduated approach of deep breathing, progressive muscle relaxation, and guided imagery.

Sessions 3 to 5 were devoted to sharing abuse histories by means of a 'time line' of key childhood experiences which the group members were asked to complete prior to session 3. Sharing abuse histories early in the group process further minimized stigmatization, eliminated secrecy, and reduced the anticipatory anxiety of disclosure. This segment of the procedure allowed ample discussion of abusive incidents, cathartic affective expression, cognitive reworking of the events, and examination of adaptive and maladaptive survivor behavioural patterns.

During session 6 members read aloud their letters to an abused child, which they had been asked to write between sessions. The letters were then discussed in the group. This exercise was used to access feelings of empathy for 'the child within' and to heighten awareness that the child victim is not culpable (Davis, 1990).

A similar exercise was to write a letter to an abuser, while letters were then discussed in session 7. These letters, written to explore often avoided feelings of anger, helped members to express intense

anger in a directed and nonviolent manner. (Similar excercises assigned intermittently throughout the remainder of treatment focused on learning to respond to the 'inner voice', developing awareness of internalized messages from childhood and how such messages affect the self-concept as well as perceptions of how one is viewed by others, attending to thoughts and attitudes about sex). Group members were also provided with written information regarding typical cognitive distortions (Burns, 1980), and were encouraged to attend to these distortions in their own and other group members' perceptions of events.

In session 8 the art therapy technique of collage (Gutierrez, Zaidi, & Lanktree, 1990) was used as a vehicle for discussion of male-female relationships. Paricipants were given large blank papers, scissors, glue, and a wide variety of magazines. They were instructed to divide the page in half, to write 'MEN' on one side and 'WOMEN' on the other and to affix photographs and/or words reflecting their concept of 'what it means to be a man/woman' under each relevant heading. Group discussion followed during which members had to explain the reasons for their selections and underlying themes. Discussion was then guided to questions of intimacy, sexuality, and relationships with women. With one exception members of the group depicted women as happy, attractive, and carefree, whereas men were depicted as solemn, angry, and burdened with troubles of the world.

Session 9 included a psycho-educational presentation of appropriate disciplinary tactics. Members were given handouts describing a rationale for 'discipline' versus 'punishment' and were instructed in basic parenting principles as well as specific techniques, such as 'time out'. This provided the basis for a group discussion and problem-solving. In addition, group members participated in role-play exercises designed to improve communication with their own children by decreasing the ambiguity of commands, and clarifying the connection between failure to comply and specific consequences.

In session 10 members recapped what they had learned, identified issues for further exploration, and discussed plans for follow-up treatment. Members were given referrals to therapists experienced in work with adult survivors of childhood abuse so that treatment could continue on an out-patient basis, if needed.

Although participants appraised the treatment program as very positive, no outcome data was published and no conclusions could be drawn on the efficacy of the treatment.

Cognitive-behavioural group therapy is a well established modality of cognitive-behavioural treatment, and has empirically been shown to be effective when compared to individual cognitive-behavioural interventions (Najavits & Garber, 1989). An example of such a group program was designed by Najavits, Weiss, and Liese (1996) for treating women with PTSD combined with substance use disorder. Substance use disorders and PTSD co-occur at a relatively high rate, often associated with a more severe course than would be expected from either disorder alone (Brady, Killeen, Saladin, Dansky, & Becker, 1994; Brown, Recupero, & Stout, 1995; Miller, Downs, & Testa, 1993; Najavits et al., 1996).

The contents of each weekly session were focused on a specific CBT skill, and the total number of sessions were divided into five units: an individual pre-group interview, an individual HIV risk counseling session, an introductory group unit to provide patients with basic education on their disorders (2 sessions), behavioural skills to prevent drug use and to control PTSD symptoms (7 sessions), cognitive restructuring, with particular attention to maladaptive thoughts associated with PTSD (6 sessions), relationship and communication skills, and emphasis on betrayal and mistrust that are associated with PTSD (Herman, 1992), (6 sessions), and a review/termination unit, focusing on processing termination of the group, ways to replace the support the group has provided, and cues by which to assess one's progress in recovery from PTSD/substance use disorders (3 sessions).

Group treatments for trauma survivors incorporate either 'covering' or 'uncovering' methods. Supportive groups represent a 'covering' approach, in which the emphasis is placed on addressing current life issues. Psychodynamic and cognitive-behavioural groups represent 'uncovering' approaches, and are designed to address members' specific traumatic experiences and memories directly (Foy et al., 2000). The task of the group facilitator is not to interact therapeutically with a group member, but to engineer an environment in which interaction between members form a therapeutic environment in which healing can be accomplished. The variety of procedures used by the facilitator to accomplish the task of shaping the group process, may include giving direct instruction, modeling, questioning, and selectively encouraging specific types of content and interactions while at the same time discouraging others. The actual therapy occurs in interaction between the members of the group.

3.2.3 Hypnotherapy

Hypnosis is a complex mental phenomenon defined as a state of heightened focal concentration and receptivity to the suggestions of another person who brings about the condition by focusing the person's attention on a monotonous routine (Kaplan & Sadock, 1998). Unlike sleep, in which typical electroencephalogram (EEG) changes are evident, hypnosis has no known psychophysiological basis (Kaplan & Sadock, 1998).

The use of hypnosis in trauma-related disorders goes back to the work of Freud, who used hypnosis to produce the abreaction and catharsis which he deemed necessary for resolving a psychic conflict (Foa & Rothbaum, 1998). Since then, hypnosis has been used for the treatment of trauma victims, firstly because hypnotic phenomena such as dissociation are common in coping with trauma and its sequelae, and secondly, because hypnosis may facilitate the recall of traumatic events which were encoded in a state of dissociation, and which are therefore not available to concious recall (Spiegel, 1989).

A number of case reports were published on the use of hypnosis in the treatment of PTSD (Jiranek, 1993; Kingsbury, 1988; Leung, 1994; MacHovec, 1983; Peebles, 1989; Spiegel, 1988, 1989), covering traumas such as combat, rape, and industrial accidents. Most of these reports, however, lack methodological precision as the symptoms targeted for treatment and the diagnostic criteria used were not specified. Only three reports (Peebles, 1989; Leung, 1994; MacHovec, 1983) gave a detailed account of how hypnotherapy was conducted. Peebles (1989) described the use of hypnosis for the treatment of PTSD in a young woman who gained consciousness three times while under anesthesia. Upon awakening from the surgery, the patient reported having gained consciousness while under anesthesia, registering operating room conversation, feeling pain as an incision was made and later when she was stitched up. She could not signal the operating room personnel of her awareness. Subsequently she rapidly developed symptoms of PTSD, initially treated with minor tranquilizers and sedatives for symptom alleviation, but without effect. She had recurrent flashbacks, which were particularly severe when she tried to sleep. The act of falling asleep was similar to losing consciousness under anesthesia and it triggered flashbacks of the surgical experience. Her ability to concentrate was severely impaired. The patient arrived for her first session in a state of extreme panic. The therapist acknowledged her panic and assured her that nothing would go out of control. The therapist spoke slowly, in low, soothing and steady tones, beginning to establish a relationship before

trance induction. This was necessary to prevent the hypnosis from becoming symbolic of a surgical procedure and then causing retraumatization. Trance induction was brief.

When the patient returned for the second session, her symptoms were worse. She had barely slept and her headaches were worse. She could only remember a small section of the memory from the first session. The rest of the recaptured memory was like a "glass wall" through which she could only see cloudy and vague images. From this session onwards, the patient was asked, at the beginning of each session, to verbally rehearse out of trance, the bits of memory recall now at her command. To counteract retraumatization, the pace of recall during the following sessions were limited to smaller bits of memory. Next, to soften her re-experiencing of being awake in surgery, she was instructed to 'administer her own anesthetizing medication' through a self-regulating deepening of her trance state. Finally, to counteract her terror-filled experience of being paralyzed in surgery, unable to signal others, she was instructed to demonstrate to herself that she was no longer paralyzed by moving her arm to touch her stomach. Each of these techniques were aimed at mastery, and to regain a sense of control, and to help her sharpen her discrimination between past and current reality. Having the patient relive the traumatic experience, only this time with a guiding other person present in the reliving, changed the emotional tone of what was originally experienced. Specific suggestions were repeatedly made regarding remembering the presence of the therapist whenever the traumatic memory came back. In this way, internalization of the therapist as a new part of memory, and as a new way through which to process the trauma memory, was fostered. During the following sessions the ego-mastery, memory consolidation, and object-relations techniques were interwoven with the continuing abreactive work. After seven sessions of hypnosis the patient's symptoms, headaches, body tremors and nausea disappeared one by one as she gained insight into what they represented.

The only controlled study on the effectiveness of hypnotherapy for treating PTSD was published by Brom et al. (1989), in which hypnosis was compared with desensitization, psychodynamic therapy, and a waiting-list control condition. Participants included 112 survivors of a variety of traumas, all meeting the diagnostic criteria for PTSD. All three treatment conditions produced superior improvement compared to the waiting-list condition. There were, however, no differences across the three treatments. According to Foa and Rothbaum (1998) inspection of the pre- and post-treatment means indicated that mean improvement on the Revised Impact of Event Scale was 29% for psychodynamic therapy, 34% for hypnotherapy, and 41% for desensitization, compared to about 10% for the waiting

list group. They pointed out that despite the limitations of the study, its results suggested that hypnotherapy, as well as desensitization and psychodynamic therapy, may alleviate post-trauma suffering to a certain extent.

3.2.4 Eye Movement Desensitization and Reprocessing

Building on Wolpe's (1958) technique of systematic desensitization, Shapiro (1989) modified this treatment by replacing progressive muscle relaxation with induced eye movements as the reciprocal inhibitor of distress. This intervention was initially designed as treatment for traumatic memories, and called Eye Movement Desensitization (EMD). The procedure entails that, after a traumatic target memory was identified, the therapist would ask the patient to articulate a self-referent negative cognition associated with the memory, (e.g., "I am shameful") as well as a positive cognition (e.g., "I am honorable") to replace the negative one. The therapist then moves his finger to and fro in front of the patient's eyes, instructing the patient to track his finger visually while concentrating on the distressing memory. After each group of ten to twelve eye movements, the therapist would ask the patient to give a rating of his distress as well as the strength of his belief in the positive cognition. This procedure would be repeated until the patient's distress subsides and his/her belief in the positive cognition is increased.

Shapiro (1989) maintained that a single 50-minute session of EMD was 100% successful in abolishing the distress associated with a traumatic memory in survivors of combat, childhood emotional or sexual abuse, and rape. She explained these impressive results by hypothesizing that the crucial component of the EMD procedure is the repeated eye-movements while the memory is maintained in awareness (Shapiro, 1989). At a later stage Shapiro reconceptualized EMD in terms of 'accelerated information-processing', renaming it Eye Movement Desensitization and Reprocessing (EMDR) (Shapiro, 1995). According to McNally (1999) the basis of the shift from EMD to EMDR appears to be more conceptual than procedural, as the EMDR intervention is very similar to the original EMD.

Several studies have since investigated the efficacy of EMDR in treating trauma survivors. Three kinds of randomized, controlled trials were reported, namely comparisons with waiting-list controls, comparisons with other treatment modalities, and dismantling studies for investigating the active ingredients of EMDR (McNally, 1999).

Testing the efficacy of EMDR against a waiting-list control condition, Wilson et al. (1995) and Wilson, Becker and Tinker (1997) reported significantly better results with patients treated with EMDR compared to the controls. However, although all the patients in this study were seeking therapy for posttraumatic stress, nearly two-thirds did not qualify for a PTSD diagnosis on entering the study. Rothbaum (1997) also reported favourable results with EMDR for rape survivors, all of whom met the diagnostic criteria for PTSD. However, McNally (1999) stressed that, although comparison of an intervention against a no treatment control condition is common in psychotherapy research, particularly for new treatments, such an approach does not exclude the possibility that whatever benefits are achieved, may merely be the consequence of non-specific factors common to all psychotherapies. Similarly Forbes, Creamer, and Rycroft (1994) argued that, consistent with the above possibility, therapeutic change following EMDR may be related to suggestibility in patients with PTSD.

Some randomized trials compared EMDR with other treatment interventions. Jensen (1994) reported no differences between the effects of EMDR and 'treatment-as-usual', which consisted of short debriefing sessions after combat, followed by either psychodynamic, cognitive or behavioural therapy sessions at a later stage, in combat veterans with PTSD. Marcus, Marquis, and Sakai (1997) however, found EMDR more effective than any of these types of therapy. As the treatments with which EMDR were contrasted were ecologically valid but unstandardized, it is virtually impossible to judge whether they were delivered appropriately. In a study of civilians with PTSD after different traumas, Vaughan, et al. (1994) found no differences between the effects of EMDR and applied relaxation. Scheck, Schaeffer, and Gillette (1998) found EMDR to be superior to Rogerian person-centred therapy in a group of young female survivors of sexual and physical abuse. Carlson, Chemtob, Rusnak, Hedlund, and Muraoka (1998) found EMDR to be more effective than relaxation training and bio-feedback in the treatment of veterans with combat-related PTSD. That three out of five of these comparative studies indicated EMDR to be more effective than Rogerian active listening, relaxation, etc., may indicate that EMDR does contain an active ingredient not shared by these other interventions (McNally, 1999). However, the results of these studies do not imply that relaxation or Rogerian active listening, for example, are effective as PTSD treatments.

Only one controlled study directly compared EMDR with a cognitive-behavioural intervention. Devilly, Spence, and Rapee (1998) randomly allocated civilian PTSD patients to either EMDR or a cognitive-behavioural treatment group, based on the treatment approach of Foa (Foa & Rothbaum, 1998). Both EMDR and CBT significantly reduced PTSD symptoms, but CBT was significantly more effective and also better tolerated by patients than EMDR. At follow-up, patients treated with CBT continued to improve, while EMDR patients started to show relapse.

Distinguishing it from other imaginal desensitization approaches, Shapiro (1989) argued that eye movement was the crucial component of EMDR and responsible for the apparently powerful effects of EMDR. Researchers (Boudewyns & Hyer, 1996; Devilly et al., 1998; Gosselin & Matthews, 1995; Pitman et al., 1996; Renfrey & Spates, 1994; Wilson, Silver, Covi, & Foster, 1996) have since compared the standard EMDR procedure to EMDR without eye movement (e.g., patients focused their eyes without moving them or the therapist just tapped the patient's fingers). In only one of these studies (Wilson et al., 1996) the effects of EMDR differed from the effects of EMDR without eye movements. Lohr, Tolin, and Lilienfeld (1998) however, critisized this study on statistical and methodological grounds. Overall, the available studies do not support the hypothesis that eye movement is the crucial component of EMDR. There are two explanations for these results. According to the first view, what is effective in EMDR (i.e., imaginal exposure) is not new, and what is new (i.e., eye movements) is not effective. A meta-analysis by Van Etten and Taylor (1998) implied that EMDR produced similar effects to those produced by conventional behavioural and cognitive-behaviour therapies for PTSD. According to the second view, studies comparing EMDR with other control manipulations (e.g., finger tapping) were actually only comparing two versions of EMDR because all "dual stimulation" procedures basically serve the same purpose as eye movements (Shapiro, 1994). In addition, Shapiro (1995) also indicated "forced fixation" (presumably of the eyes) as another variant of EMDR.

The arguments by EMDR proponents that researchers who reported negative results for EMDR were inadequately trained or that they did not strictly adhere to the EMDR protocol (Greenwald, 1996), have been refuted in a review by Lohr et al. (1998). They reported that in 15 out of 16 EMDR outcome studies therapists had been trained in accredited EMDR workshops. Neither is there any convincing evidence, according to Pitman et al. (1996), that fidelity to protocol predicts outcome in EMDR treatment. As emphasized by Rosen and Lohr (1997), the burden of proof rests on the advocates of

EMDR to demonstrate that eye movements in itself really have therapeutic value. Considering the above-mentioned results and conclusions from studies such as those of Van Etten and Taylor (1998), Lohr et al. (1998), Pitman et al. (1996) and Devilly et al. (1998), EMDR cannot at this stage conclusively be credited with proven efficacy in the treatment of PTSD.

3.2.5 Cognitive-behaviour therapy

Various techniques and procedures for the treatment of PTSD are used within the broad framework of cognitive-behaviour therapy. These include cognitive restructuring, cognitive reprocessing therapy, stress inoculation training, systematic desensitization, and exposure treatment.

3.2.5.1 Cognitive therapy and cognitive restructuring

Beck's Cognitive Therapy, originally developed for the treatment of depression (Beck, Rush, Shaw, & Emery, 1979), and Ellis's Rational-Emotive Behaviour Therapy (Ellis, 1962), are both examples of cognitive restructuring therapies based on the information processing paradigm. It is hypothesized that the way in which an individual gives meaning to his world and structures events, influence his or her emotions and behaviours and not the event per se. Biased information-processing is considered to be at the core of all emotional disturbance.

According to Beck's cognitive theory of psychopathology, the central pathological process involved in anxiety disorders is hyperactive cognitive patterns or self-schemas relevant to danger which are continually structuring external or internal experiences as signs of danger or threat (Beck, Emery, & Greenberg, 1985). Beck et al. (1985) used the concept of a mode to signify a superordinate organizing principle which, when dominant, determines the type of schema activated at a given time. In the anxiety disorders the vulnerability mode is hypothesized to dominate, involving the activation of self-schemas that are hypersensitive to threat cues, thus introducing a systematic bias into information-processing. Central to Beck's theory is the hypothesis that individuals suffering from different anxiety disorders, process information differently. It is, for example, hypothesized that the self-schemas of individuals with panic disorder are hypersensitive to information associated with physical threat, e.g., changes in heart rate, sweating, while individuals with social phobia are overly concerned with social threat, especially a vulnerability to negative evaluation by others (Beck et al., 1985). Numerous

studies offer support for these hypotheses (e.g., Dodge, Hope, Heimberg, & Becker, 1988; Hope, Rapee, Heimberg, & Dombeck, 1990; Malvone, Bruch, & Heimberg, 1993; Mattia, Heimberg, & Hope, 1993; McNally, Kaspi, Riemann, & Zeittin, 1990; Salkovskis & Clark, 1990; Van Niekerk, Möller, & Nortje, 1993).

Information-processing theories of PTSD apply Lang's notion of fear structures as a network in memory (Lang, 1977, 1979). Foa, Steketee, et al. (1989), for example, proposed that the fear structure present in PTSD consists of mental representations of trauma-related stimili, information about cognitive, behavioural and physiological responses to the event, and meanings associated with the stimulus and response elements. Activation of this fear structure will lead individuals with PTSD to evidence a range of cognitive biases, such as attentional bias to potential threats, intrusive thoughts of the trauma (memory bias), and exaggerated beliefs about trauma-related issues. In particular, Foa, Steketee, et al. (1989) proposed that PTSD is associated with an overestimation of the likelihood of negative events occuring (probability bias), and an overestimation of the adverse consequences of such events (cost bias). According to Foa, Steketee, et al. (1989) the fear structure in PTSD is more pervasive regarding stimuli, responses and meanings that connate danger, compared to other anxiety disorders in which it is more circumscribed. This predicts that biases in the judgement of risk extend to a range of threatening situations including those previously perceived as benign.

Ehlers and Steil (1995) argued that the idiosyncratic meaning of intrusive symptoms is important in the maintenance of PTSD. Dysfunctional meanings concern both the occurrence of intrusions (e.g., 'The fact that I have these uncontrollable memories means that I am going crazy') and their content (i.e. the traumatic event and its sequelae). Examples of the latter are 'My life is ruined', 'It was my fault', and 'It will happen again'. The authors suggested that there are two pathways by which dysfunctional meanings maintain posttraumatic intrusions and other PTSD symptoms. These pathways are assumed to determine the degree of distress, and thus arousal, caused by intrusions, and also to determine the extent of cognitive and behavioural avoidance (Steil & Ehlers, 2000).

The proposed '*distress*' pathway leads to short-term maintenance of arousal and re-experiencing symptoms:

- The distress caused by the negative meaning of the intrusions is likely to be accompanied by physical symptoms such as heightened arousal, sleep problems, and poor concentration.
- Physical arousal in turn may act as an internal trigger for the occurrence of intrusions.
- Furthermore, intense distress and arousal can confirm the negative meaning the intrusions have for the patient (e.g., 'I am incompetent').

The '*avoidance*' pathway leads to the maintenance of posttraumatic intrusions in the short and long term:

- The dysfunctional meaning of the intrusions motivates the patient to engage in behavioural and cognitive avoidance strategies intended to bring the intrusions to an end.
- In a vicious circle effect, the increase in upset and arousal which is connected to the occurrence of intrusions may contribute to the motivation to avoid these experiences.
- Cognitive strategies like thought suppression and rumination actually lead to increased intrusion frequencies.
- The patient may find the paradoxical effects of cognitive avoidance strategies very alarming and these strategies may therefore contribute to the level of ditress caused by posttraumatic intrusions ('Although I try very hard to get rid of these memories, they keep coming back!'). Furthermore, these strategies might directly be related to symptoms of heightened arousal like difficulties in sleeping or concentrating.
- Avoidance of reminders of the trauma prevents reduction in distress/arousal (habituation).
- It prevents change in meaning of the intrusions and the trauma, and therefore maintains the intrusions in the long term.

In two studies with 159 and 138 individuals respectively, all of whom had experienced a motor vehicle accident, Steil and Ehlers (2000) found that whether or not posttraumatic intrusions are experienced as distressing depends on their idiosyncratic meaning for the person. Both studies consistently showed substantial correlations between the dysfunctional meanings assigned to the intrusions and the distress caused by them. These correlations remained significant when intrusion frequency, accident severity, and general anxiety-related catastrophic conditions were partialed out. If the occurrence or content of

intrusions are interpreted as indicating insanity, incompetence, permanent negative change or future danger, the person experiences distress. If, on the other hand, intrusions are seen as a normal part of recovery and processing of the trauma, distress is less likely (Steil & Ehlers, 2000). Both these studies confirmed the hypothesis that the dysfunctional meaning of posttraumatic intrusions predicted coping strategies that have been found to maintain intrusive cognitive phenomena, namely avoidance of reminders, thought suppression, rumination, and distraction. All of these avoidance strategies are likely to prevent full emotional processing of traumatic experiences and/or change in maladaptive cognitions. The results of both studies also confirmed that the combination of intrusion frequency, dysfunctional meaning, and avoidance measures explained substantially more variance in PTSD severity than the combination of intrusion frequency and measures of accident severity (Steil & Ehlers, 2000).

The objective of cognitive restructuring is to modify a patient's dysfunctional automatic thoughts and underlying schemas. This is done in steps in which patients are taught to identify their dysfunctional thoughts, to challenge those evaluated as inaccurate or unhelpful, and finally, to replace them with more logical or beneficial thoughts. In the case of trauma survivors, attention is particularly focused on their appraisal of safety/danger, trust, and their views of themselves (Rothbaum, Meadows, Resick, & Foy, 2000). Cognitive restructuring entails a didactic or educational component in which the patient is presented with the rationale of cognitive restructuring in order to understand his symptoms in terms of faulty information-processing. It also incorporates cognitive and behavioural procedures as well as homework assignments to assist the patient in identifying and changing his/her dysfunctional automatic thoughts and underlying schemas.

Frank et al. (1988) reported Cognitive Restructuring as effective in treating post-rape symptoms. However, methodological shortcomings such as the lack of a control group, make it difficult to draw definite conclusions about the efficacy of cognitive restructuring from this study. Comparing cognitive restructuring, prolonged exposure, and a combination of cognitive restructuring and prolonged exposure, Marks et al. (1998) found that all three treatment modalities were effective in reducing PTSD symptoms. Rieckert and Möller (2000), in a study of adult survivors of childhood sexual abuse, found cognitive restructuring, based on Rational-Emotive Behaviour Therapy, to be effective in reducing symptoms of anxiety, depression, guilt, and anger.

3.2.5.2 Cognitive processing therapy

Cognitive Processing Therapy (CPT) was developed by Resick and Schnicke (1992) specifically for the treatment of rape victims. It is based on the information-processing paradigm and entails a combination of cognitive restructuring and exposure. McCann and Pearlman (1990) as well as McCann, Sakheim, and Abrahamson (1988) suggested that the trauma of rape leads to disruption of existing schemas, specifically in the areas of safety, trust, power, respect and intimacy. Cognitive restructuring is used to target each of these areas in designated sessions. In addition to cognitive restructuring, CPT also includes exposure in which the patient gives a detailed written description of the rape and then reads it back.

In a quasi-experimental, uncontrolled and unstructured study by Resick and Schnicke (1992) 19 rape victims were treated with this procedure. It consisted of 12 weekly sessions of 90 minutes each. At the first session, an information-processing formulation for PTSD was presented and as homework participants were requested to write about the trauma's meaning for them. During the second session, participants were taught to identify and differentiate feelings from thoughts, and were given A-B-C sheets to complete as homework so that they could become aware of the connection between self-statements and emotions. During the next two sessions, which constituted the exposure componenet of CPT, participants were asked to write an account of the rape, and they were encouraged to experience all their relevant emotions fully while writing and reading over the account. During the fifth session, they were taught to identify and challenge their own maladaptive beliefs. They were given a list of questions to ask themselves about these beliefs. In the sixth session, each participant was given a 'challenging beliefs' worksheet, which is a more elaborate version of the A-B-C sheet and which incorporates a list of challenging questions and analysis of faulty thinking patterns. During the seventh session the areas of beliefs most likely to have been affected by the rape, namely safety, trust, power, esteem, and intimacy, were introduced. These were discussed at the hand of how prior positive beliefs could be disrupted, or prior negative beliefs confirmed by rape. Suggestions for possible solutions (more adaptive self-statements) were also included. The themes were presented sequentially and analyzed, one per week (sessions 7 – 11). The therapists and group members helped each other confront specific therapeutic blockages. At the 11th session, participants were again asked to write about the meaning of the traumatic event, without referring to their first assignment. During the final

session beliefs regarding intimacy and participants' essays and goals for the future were discussed (Resick & Schnicke, 1992).

Resick and Schnicke (1992) concluded that CPT yielded better results than individual treatment programs. However, these conclusions are questioned due to the methodological shortcomings of the study. A later, controlled study by Resick et al. (1998), in which sexual assault survivors were randomly assigned to either CPT, exposure, or a minimal attention waiting group, showed that both exposure and CPT significantly reduced PTSD symptoms.

3.2.5.3 Stress Inoculation Training

In contrast to exposure therapy (Foa & Kozak, 1986) and cognitive restructuring (Beck et al., 1985) which aim at correcting the cognitive mechanisms underlying pathological anxiety, the objective of anxiety management programs such as stress inoculation training is to empower the patient with coping skills by which the anxiety can be better managed.

Stress inocculation training (SIT) was originally developed by Meichenbaum (1975) for treating anxiety and consists of eight to twenty sessions, normally twice a week, each session lasting 90 minutes.

The program consists of three phases:

- An educational phase (sessions 1 and 2) which includes an explanation of the treatment program as well as education about the nature of fear and anxiety.

- A skill-building phase during which the patient learns a set of coping skills to manage the identified fear responses across the three response channels (physiological, cognitive and behavioural). Skills training includes muscle relaxation, breathing control, thought stopping, cognitive restructuring, guided self-dialogue (during which the therapist helps the patient to focus on self-statements, to identify negative self-statements and to replace it with positive coping statements), role playing, and covert modeling which consists of imaginal roleplay. During this imaginal roleplay the therapist identifies an anxiety-provoking image A, the patient then imagines A, verbalizes the images and introduces successful coping statements. The process is repeated by the patient. Each session begins with a ten minute review of homework

assignments. A new coping skill is then taught using a non-assault related situation at first, followed by an assault-related situation.

- An application phase during which the patient learns step-by-step how to apply these coping skills in daily situations which provoke anxiety. These steps include assessing the probability of the feared event, managing avoidance behaviour with thought-stopping and relaxation, controlling self-criticism with guided self-dialogue and cognitive restructuring, engaging in the feared behaviour using problem-solving skills and skills learned via roleplaying and covert modeling, and reinforcing self for using skills in feared situations.

The efficacy of SIT with female rape victims was examined in two uncontrolled studies by Kilpatrick et al. (1982) and Veronen and Kilpatrick (1982). In the Kilpatrick et al. (1982) study, one of the goals was to assess which treatment rape victims preferred. Patients were allowed to choose among three treatments offered: SIT, SD, and a peer counseling condition. No patients chose the SD treatment, 70% chose the SIT and 30% chose peer counselling. Both these reports focused on post-rape sequelae of fear, intrusions, and avoidance, rather than on the full syndrome of PTSD. The SIT treatment was spelled out in considerable detail and was delivered by peer counsellors who had experience in counseling rape victims and who received specific training in SIT. The Veronen and Kilpatrick (1982) study came to the conclusion that SIT was effective in reducing rape-related fear and anxiety, as well as avoidance, general tension, and depression. Most of these gains were maintained at three months follow-up. Although the lack of a control group precluded definitive conclusions about the efficacy of SIT, this study suggested that SIT can effectively reduce rape-related psychopathology (Foa & Meadows, 1997).

The first controlled study examining the efficacy of SIT in ameliorating post-rape psychological problems was conducted by Resick et al. (1988). Rape victims were assigned to four treatment conditions: SIT, assertiveness training, supportive psychotherapy, and a naturally occurring waiting-list. Assignment to treatment was determined by openings in the next available treatment. Target symptoms were not clearly specified and inclusion criteria were described only as having been raped at least three months before participation in the study, absence of incest history, absence of severe competing psychopathology, and problems with rape-related fear and anxiety. Several self-report measures were included, including the revised Impact of Event Scale. A structured interview which

was not explicitly described in the report, was also used at the initial evaluation (Foa & Meadows, 1997). At post-treatment, all three procedures produced improvement in fear and anxiety, with no improvement in the waiting-list condition. On the revised Impact of Event Scale, SIT produced a 27% reduction of fear and anxiety compared to an increase of 14% in the waiting-list controls.

In another comparative study, Foa et al. (1991) randomly assigned female victims of sexual or nonsexual assault with PTSD to one of three treatment conditions: prolonged exposure (PE), SIT, or supportive counselling. A wait list control group was included. Treatment targets were PTSD symptoms, which were assessed at pre- and post-treatment and at follow-up, using interviews and self-report measures, with the interviews conducted by trained clinicians who were blind to treatment condition. All treatments included nine sessions of 90 minutes each conducted over five weeks and were based on detailed treatment manuals. The same therapists conducted all three treatments to avoid therapist bias, and they were supervised throughout the study. This study therefore fulfilled all gold standard criteria, with the exception of a required minimum severity threshold and details about ongoing interrater reliability assessments (Foa & Meadows, 1997). Immediately following treatment, both PE and SIT patients showed improvement on all three clusters of PTSD symptoms. Patients receiving supportive counselling or who were on the waiting-list improved on the arousal symptoms, but not on the avoidance or re-experiencing symptoms. At follow-up, PE appeared to be the most successful on all measures of psychopathology, as 55% of women in the PE treatment no longer met the diagnosis of PTSD, compared to 50% of the SIT group and 45% of the supportive counselling group.

In a second study Foa (1995) compared PE, SIT, a combination of PE and SIT, and a waiting-list control condition. All gold standards were met in this study. All three treatments showed significant improvement in terms of PTSD and depressive symptoms at post-treatment, while the control group did not improve. These treatment effects were maintained at six months follow-up. During an examination of patients who achieved good end-state functioning as defined by criterion scores on PTSD symptoms as well as measures of depression and anxiety, 21% of patients in the SIT group, 46% of patients in the PE group, and 32% of patients in the combined PE/SIT group achieved this goal at post-treatment. At six months follow-up, 75% of patients in the PE group, 68% of patients in the SIT group, and 50% of the patients in the PE/SIT group lost the PTSD diagnosis, while all waiting-list patients still retained the diagnosis.

3.2.5.4 Systematic Desensitization

Systematic desensitization (SD) was developed by Wolpe (1958) for the tratment of anxiety, particularly for phobic anxiety. It is based on a conditioning paradigm, and consists of two components: relaxation training and the presentation of a hierarchy of anxiety-inducing stimuli. The principle of reciprocal inhibition entails that imaginal (or in vivo) exposure to the feared stimuli is paired with relaxation, in a graded hierarchical fashion. According to this principle, the increasing level of relaxation paired with the fear stimuli, will reciprocally diminish the anxiety.

Studies by Schindler (1980) and Wolff (1977), conducted before the official recognition of PTSD, obviously did not use standardized measurements of PTSD, nor were independent blind evaluations utilized. Manualized treatment protocols and adherence ratings were not reported either. In addition, some of the patients included were recent assault victims, at least some of whom would be expected to recover naturally (Foa et al., 1995). This uncontrolled factor therefore inflated treatment results (Foa & Meadows, 1997). After the recognition of PTSD as a diagnostic chategory, Frank et al. (1988) conducted an uncontrolled study on the effectiveness of SD in treating PTSD. Although participants in the these three studies showed improvement in terms of post-trauma symptoms, the methodological problems of these studies rendered the results inconclusive.

In a controlled study by Brom et al. (1989), 112 trauma victims were treated with either hypnosis, SD, or psychodynamic therapy. A waiting-list control group was included. Patients included in the study suffered from a variety of traumas, although the majority did not directly experience a trauma, but had lost a loved one as result of trauma. All the patients reportedly met the criteria for PTSD, although the assessment method was not reported explicitly. Assessment included two pre-treatment interviews, but no post-treatment interviews. Outcome evaluation was based solely on standardized self-report measures, thereby introducing possible expectancy bias. Treatments were carried out by trained and supervised therapists, but no adherence ratings were obtained. Therapists provided the treatment in which they individually specialized, which thus increased their faith and competence in the treatment but also introduced a possible confound of therapist effects that cannot be separated from treatment differences. Several other biases also occurred. Patients on the waiting-list received unspecified treatment outside the research setting, and the number of sessions this group had, varied across treatments. All three treatment conditions produced superior improvement compared to the waiting-

list condition, but no difference between the three treatments were observed. Psychodynamic therapy decreased avoidance more than intrusion symptoms, while hypnosis and SD reflected the reverse pattern. Inspection of the means indicated improvement on the revised Impact of Event Scale was 29% for psychodynamic therapy, 34% for hypnotherapy, and 41% for SD.

3.2.5.5 Exposure Treatment

An overview of the litterature on the psychological treatment of PTSD showed prolonged exposure to probably be the most widely used and extensively researched procedure for the treatment of PTSD from a cognitive-behavioural perspective. A variety of theories to explain the effect of exposure treatment were also developed. As exposue treatment is the focus of the present study, it will be discussed in detail in Chapter 4.

3.2.6 Psychological Debriefing

Psychological debriefing does not constitute a treatment procedure for PTSD, but entails interventions to prevent the development of negative psychological sequelae, such as PTSD, following exposure to trauma. The term denotes a brief preventative procedure that is presented shortly after a traumatic event.

Psychological debriefing incorporates a variety of interventions. The crisis intervention model aims at assisting the patient in re-establishing rational problem-solving (Bisson, McFarlane, & Rose, 2000). Psycho-education entails giving trauma victims a psychological map to better understand their reactions so as to help them contain their distress and to institute a series of self-regulatory processes (Bisson et al., 2000).

Probably the most important variation of psychological debriefing is Critical Incident Stress Debriefing (CISD), which was first described by Mitchell (1983) as an individual or group intervention for ambulance personnel following exposure to traumatic situations in their work. It is a semi-structured intervention consisting of seven phases (Mitchell, 1983):

- A fact phase: Victims describe their role in the event and provide factual accounts of what happened.
- A thought phase: Victims are encouraged to put words to their first thoughts during the event.
- A reaction phase: Survivors are helped to move from a cognitive to an emotional level and to express the powerful emotions attached to their experience.
- A symptoms phase: Survivors are encouraged to explore the thoughts, feelings and behaviours they experienced during the event.
- A teaching phase: Survivors are educated about stress reactions and how to cope with it.
- A re-entry phase: During this phase facilitators encourage survivors to continue with counselling and provide them with a list of relevant resources available in their area.
- A closure phase: During this phase facilitators answer any final questions survivors may have and bring the CISD session to a close.

Mitchell (1983) initially commented that a follow-up CISD might be necessary several weeks or even months after a critical incident to deal with unresolved issues if these are present.

Dyregrov (1989) developed a debriefing procedure based on Mitchell's model consisting of the following phases:

- The introduction, explaining the purpose of the procedure.
- Discussion of the patient's expectations of the procedure, as well as the facts of what happened during the trauma.
- Discussion of the victims' thoughts and impressions in order to put his/her reactions into perspective, as well as to help with the integration of the traumatic experience.
- Discussion of the victim's emotional reactions, where the debriefer attempts to aid the release of the relevant emotions.
- Normalization, where the debriefer attempts to facilitate the acceptance of the emotional reactions which have been expressed.
- Future planning/coping, where the debriefer focuses on ways to manage the victim's symptoms which may arise, and to attempt to mobilize the patient's internal support mechanisms.
- Disengagement, a stage in which other topics are discussed and guidance is given regarding the need for further help and counseling if it should be necessary.

Adaptations of the Mitchell/Dyregrov model of CISD were developed by Lee, Slade, and Lygo (1996), Hobbs, Mayou, Harrison, and Warlock (1996), and Irving and Long (2001).

Raphael (1986) described a less structured psychological debriefing procedure,which still had much in common with the Mitchell model and which was designed as a group intervention for secondary rather than primary victims. Armstrong, O'Callahan, and Marmar (1991) designed the Multiple Stressor Debriefing Model, containing elements from the other debriefing procedures, for use with American Red Cross personnel.

Despite the fact that psychological debriefing is commonly used following trauma, Rose and Bisson (1998) and Bisson et al. (2000) pointed out that adequately controlled outcome studies for these interventions are scarce and warned that these procedures have become generally accepted without significant scientific evidence for their effectiveness.

Rose and Bisson (1998) conducted a review of the six outcome studies available at that stage, which included the following: Bisson, Jenkins, Alexander, and Bannister (1997) conducted a randomised controlled trial of PD for 130 hospitalised victims of acute burn trauma and compared it with a control group receiving standard hospital care. The results showed that the intervention group fared worse on all measures at 13 months. Bunn and Clarke (1979) conducted a randomised controlled study of 30 relatives of hospitalised, seriously ill or injured patients. At assessment immediately after the debriefing sessions, the intervention group was reported to be less anxious than the standard hospital care control group. Bordow and Porritt (1979) reported a study with 70 hospitalised motor vehicle accident victims. A standard hospital care group was included, as well as a group who received extended psychiatric care. At three to four months follow-up, the PD group fared better than those who received no intervention, but worse than the group who received extended care. Hobbs et al. (1996) reported a study with 106 hospitalised motor vehicle accident victims. At four months follow-up the debriefing intervention group fared worse than the standard hospital care control group on some measures. It is unclear whether the higher injury severity score in the PD group was controlled for. Lee et al. (1996) compared 60 hospitalised women who received debriefing following early miscarriage, with a standard hospital care control group. At four months follow-up there was no discernable difference between the two groups. Hobbs and Adshead (1996) conducted a study with 63 hospitalised survivors of motor vehicle accidents. A standard hospital care control group was included.

At one week, and one and three months follow-up no differences were found between the groups. Participants, who demonstrated high trait anxiety and high scores on the BDI on entry into the PD group, were significantly better at follow-up. It must be noted that those who bcame 'unusually distressed' during counselling were dismissed. Based on this review Rose and Bisson(1998) concluded that two studies found negative effects, two demonstrated no overall effect and two revealed some evidence for a positive outcome.

Miller-Burke, Attridge, and Fass (1999) surveyed 141 bank employees, involved in 42 different bank robberies, after receiving CISD. CISD was rated as 'helpful' by 78% of the employees. In a similar study of duty-related stress amongst police officers, Robinson, Sigman, and Wilson (1997) found that 63% of respondents stated that CISD was beneficial. Typically respondents reported a greater cognitive and emotional understanding of the event itself and felt reassured that their reactions were normal. Respondents also felt that the process prepared them for possible future reactions and they believed they were more in control in terms of coping. Leonard and Alison (1999) investigated appraisal and coping behaviours as well as symptom and expectation outcomes following CISD. Two groups of 30 Australian police officers involved in shooting incidents participated in the study. One group received CISD and the other group acted as controls. The CISD group demonstrated a significant reduction in anger levels and greater use of specified coping strategies compared to the controls.

Irving and Long (2001) conducted a study with three women who had experienced divergent traumatic incidents. A case study approach was used and initial contact took place 24 hours following the traumatic experience. Critical Incident Stress Debriefing was administered and at six months following the traumatic experience the women were interviewed again. The participants concluded that the debriefing intervention provided a safe forum for them to explore their needs, process their experiences and create constructive narratives.

In a review article on the effectiveness of psychological debriefing, Arendt and Elklit (2001) came to the conclusion that in general, debriefing does not prevent psychiatric disorders or mitigate the effects of traumatic stress, even though participants may find the intervention helpful. However Bisson et al. (2000) argued that there are many potentially important variables that have not been adequately and systematically addressed by existing studies, including the time lapse between the trauma and the PD, the nature of the trauma, the experience of facilitators, and the nature of the PD.

Rose, Bisson and Wessely (2003) extended the review by Rose and Bisson (1998) by adding four more studies to the original six. These studies were conducted by Conlon, Fahy, and Conroy (1999), Lavender, and Walkinshaw (1998), Rose, Brewin, Andrews, and Kirk (1999) and Small, Lumley, Donohue, Potter, and Walderstrom (2000). In all these studies, participants were randonly allocated to an early single-session debriefing intervention or a no intervention condition. The authors concluded that these studies have methodological shortcomings including absence of blind assessors, small sample sizes, and variation in the techniques used. The studies provided little evidence that an early psychological intervention prevents psychopathology following trauma (Rose et al., 2003).

There is growing concern that PD and CISD may potentially be harmful. Rose et al. (2003) raised the possibility that these interventions may lead to an increase in psychological distress by creating re-exposure to the traumatic event. As PD involves intense imaginal exposure to the traumatic event within a short time of the incident, this may serve as a further trauma, exacerbating symptoms without helping emotional pocessing. Another criticism against psychological debriefing is that it may medicalise normal distress. It may increase the expectancy of developing psychological symptoms in individuals, who might not normally have developed them. The finding has constantly been that no matter how severe the trauma, not every victim develops a formal long-term psychiatric disorder. By its increasing awareness of psychological distress, PD/CISD may paradoxically induce distress in victims, who would not otherwise have developed it. The danger also exists that PD may be seen as a substitute for the traditional support given by friends and family. Additional criticism links the negative outcome of debriefing with the association between heightened arousal in the early post-trauma phase and the greater likelihood of long-term psychopathology (Bryant & Harvey, 2000; Shalev, 2001). Because verbalising of the trauma in debriefing may override a natural inclination of the highly distressed to avoid reminders of the trauma, habituation to the evoked distress may not occur. Therefore, overriding dissociation and avoidance soon after trauma via debriefing may be detrimental to some individuals, particularly those with heightened arousal (Ruzek & Watson, 2001). In addition, PD is based on the principle that there is a uniform and, to a certain extent, predictable pattern of reactions to trauma. It also assumes that discussing the trauma is therapeutic, and that attempting to deny it is not. This is not necessarily true in every case. Attempting to forget or distance oneself may for some individuals be an adaptive response which will not necessarily lead to psychological symptoms or PTSD. An intervention like PD or CISD may thus interfere with adaptive defence mechanisms such as denial.

3.3 PHARMACOTHERAPY

It has been argued that, given the number of psychobiological systems that appear to be involved and/or altered in PTSD, it may well be that PTSD is not a unitary psychobiological abnormality, but that there may be a number of possible mechanisms through which this disorder might evolve. Another proposed possibility is that there are different psychobiological subtypes of a common posttraumatic stress disorder (Friedman, Davidson, Mellman & Southwick, 2000). Rasmussen and Charney (1997) concluded that because of this complexity, there is no single animal model that is applicable to PTSD.

Psychobiological abnormalities found in PTSD involve specific neurotransmitter, neurohormonal and neuroendocrine systems. This knowledge is relevant to understanding why certain drugs might be effective in treating PTSD smptoms, and it might guide the development of future drugs designed specifically for use in PTSD (Friedman et al., 2000).

In studies on pharmacotherapy for PTSD the antidepressant class of drugs were initially used. Most have been shown to provide at least some relief of PTSD symptoms. Tricyclic anti-depressants and selective serotonin reuptake inhibitors (SSRIs) seem to be effective for PTSD, but the effects of other drugs are equivocal. The benefits of tricyclic antidepressants have been modest, but the benefits of SSRIs have been more impressive (Foa & Rothbaum, 1998).

3.3.1 Conceptual basis for medication in PTSD

Two conceptual frames of reference support the use of medication in PTSD. Firstly, Sargent and Slater (1940) advocated the use of medication in an abreactive context to help uncover dissociated or repressed material which presumably could lead to a variety of symptoms. Hogben and Cornfield (1981) also supported this model, which primarily derived from immediate intervention with patients suffering from acute PTSD. Their observations that pharmacotherapy aids the psychotherapeutically driven recovery process by promoting more powerful abreactive affects, suggest that this model should be applicable to chronic PTSD as well.

The second model, also by Sargent and Slater (1940), seemingly stands in contrast to the first model by suggesting that chronic PTSD responds less successfully to abreactive approaches. According to this view, antidepressant drugs suppress symptoms disrupting the lives of PTSD sufferers, and allow them to restore their normal, effective coping mechanisms. This model presents medications as largely being suppressive of symptoms rather than aiding expression of affect (Foa & Rothbaum, 1998).

3.3.2 Biological basis for medication in PTSD

According to Foa and Rothbaum (1998) pharmacotherapy in PTSD is based on the neurobiological evidence that uncontrolled life-threatening trauma leaves an imprint on, amongst others, the opiate and other neuropeptidergic systems, the hypothalamic-pituary-adrenal axis, and the autonomic nervous system. This results in dysregulation of multiple neurochemical systems. Although evidence is building to support a unique constellation of biological abnormalities in PTSD, data from phenomenological, neurobiological, and treatment response studies suggest that PTSD shares components with dissociative states (Bremner et al, 1992), panic attacks (Mellman & Davis, 1985), and obsessions. Part of the defining criteria for PTSD are dissociative phenomena (eg., flashbacks, feelings of detachment, psychogenic amnesia), as well as obsessive phenomena (eg., intrusive recollections, attempts at violence). Evidence is emerging for a spectrum of disorders (including various affective and anxiety disorders) that are responsive to antidepressants (Hudson & Pope, 1990). Tricyclic antidepressants (amitriptaline, imipramine), monoamine oxidase (MAO) inhibitors (phenelzine) and SSRIs (fluoxitine) have all been reported to be beneficial at some level in the treatment of PTSD.

There is also growing evidence that the neurotransmitter serotonin is important in PTSD. It is important to recognize that serotonin antagonists, such as trazodone and mianserin, can reverse conditioned avoidance responses mediated serotonergically. SSRI drugs, such as fluoxitine, are effective in the strengthening of impulse control (connected with serotonin levels) which is often lacking in PTSD and may affect modulation.

Following an overview of current research findings, Friedman et al. (2000, p. 85) concluded: "Adrenergic Hyperreactivity appears to be associated with hyperarousal, reexperiencing, panic/anxiety symptoms, and probably associated with dissociation and rage/aggression. Adrenergic mechanisms also play a key role in processing traumatic memories. An alpha-2 adrenergic agonist such as

chlonidine or a beta-adrenergic such as propranolol might be expected to attenuate this abonormality. Tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs) also reduce adrenergic activity through more indirect mechanisms. As shown in studies on treatment of panic disorder, the antiadrenergic effects of both TCAs and MAOIs can be clinically significant." In addition, Friedman et al. (2000) suggested that further consideration of the unique pathophysiology of PTSD would suggest that drugs acting on key mechanisms of the human stress response, itself, might have potential usefulness in PTSD treatment. This could include currently experimental drugs that reduce the actions of CRF, such as CRF antagonists, neuropeptide Y agonists, or drugs that enhance the actions of neuropeptide Y (Friedman et al., 2000).

3.4 CONCLUSION

Based on the preceding review of the literature on the psychological treatment of PTSD, the following conclusions can be drawn.

- Different psychotherapeutic approaches and procedures are used to treat PTSD, including psychodynamic therapy, group therapy, hypnotherapy, eye movement desensitization and reprocessing, and cognitive-behaviour therapy.
- However, very few studies on the effectiveness of psychodynamic therapy for PTSD or studies, which compared its effectiveness to that of other types of interventions, are available, placing a question mark behind its appropriateness for the treatment of PTSD. The same applies to the use of hypnotherapy in treating PTSD.
- Despite more available studies investigating the effectiveness of EMDR as treatment procedure for PTSD, EMDR cannot at this stage conclusively be credited with proven efficacy in the treatment of PTSD.
- The literature revealed a strong emphasis within the cognitive-behavioural framework on outcome studies for PTSD. Different cognitive-behavioural treatment procedures are described, including cognitive restructuring, cognitive processing therapy, stress inoculation training, systematic desensitization, and prolonged exposure. Although outcome studes are reported for all these procedures, there seems to be a need for further investigation of cognitive restructuring and cognitive processing therapy in the treatment of PTSD. Existing well controlled studies offer support for the effectiveness of stress inoculation training and prolonged exposure as treatment procedures for PTSD.

- The effectiveness of psychological debriefing as a preventative procedure following trauma has not been established. In addition, there is growing concern that psychological debriefing may be potentially harmful as the imaginal exposure to the traumatic event, characteristic of this procedure, may lead to retraumatization.

4. EXPOSURE TREATMENT FOR PTSD

A review of the treatment outcome literature showed exposure to be the most researched psychological treatment for PTSD. Its effectiveness has been investigated for a broad spectrum of trauma populations, including rape victims, survivors of motor vehicle accidents, war veterans, survivors of terrorist attacks, and survivors of natural disasters (Shalev, Yehuda, & McFarlane, 2000). Consequently, based on the available research data, exposure treatment is currently considered the treatment of choice for PTSD.

This chapter provides an outline of the theories underlying exposure treatment, as well as a discussion of outcome studies on exposure.

4.1 COGNITIVE-BEHAVIOURAL THEORIES OF PTSD

There are two broad categories of cognitive-behavioural theories of PTSD: the social-cognitive theories and the information-processing theories. The social-cognitive theories (e.g., Horowitz, 1986; Janoff-Bulman, 1985, 1992) focus on the integration of a traumatic experience into a person's pre-existing view of the world. Janoff-Bulman's theory of adaptation to trauma (Janoff-Bulman, 1985; 1992; Janoff-Bulman & Frieze, 1983) is based on the notion that people hold basic, unchallenged assumptions about themselves and the world they live in. Beginning in early childhood, these assumptions developed out of the person's interactions with the world and other people (Du Plessis, 1998). The concept of an 'Assumptive world' is used to refer to this strongly held set of beliefs and assumptions about the world and the self which is confidently maintained and used as a means of recognizing, planning and acting. These assumptions or schemas represent abstracted knowledge structures, stored in memory, and involve a rich network of information about a given domain, capable of guiding subsequent perception and appraisal (Du Plessis, 1998). This "Assumptive world" theory states that individuals hold three core assumptions, namely about the benevolence of the world, the meaning of the world to them, and the worthiness of the self. According to Janoff-Bulman (1992), the category of benevolence includes the benevolence of the impersonal world, as well as the benevolence of people. The basic belief in the benevolence of the world rests on the belief that events are orderly and comprehensible. Because traumatic events are extremely salient and disturbing, victims are

forcefully confronted with a catastrophic upheaval of their conceptual system and consequently experience the loss of old, firmly held and positive views of the world and themselves (Janoff-Bulman, 1992). When this new information associated with a traumatic experience is integrated into the existing knowledge structures, successful information-processing has occured. When such integration does not take place, it may result in PTSD.

The Information-processing theories focus more specifically on trauma-related threat, on how trauma-related information is represented in the cognitive system, and on how it is subsequently processed. These theories, underlying exposure treatment for PTSD, include Rachman's Emotional Processing theory, Ehlers and Clark's Cognitive theory, Brewin, Dalgleish and Joseph's Dual Representation theory, and Foa's Emotional Processing theory.

4.1.1 Rachman's Emotional Processing Theory

Rachman (2001) described emotional processing as a process through which emotionally traumatic events are absorbed, evaluated and integrated so that other, normal and undisturbing experiences and behaviours can proceed without disruption. Dysfunctional emotional processing is an unsatisfactory process whereby traumatic experiences are not absorbed effectively and which forms the basis for PTSD. In his view the criterion for unsatisfactory emotional processing is the intrusive re-experiencing in PTSD. Rachman described four main factors which lead to unsatisfactory processing: state factors (such as dysphoria, illness and fatique); maladaptive cognitions (a tendency to catastrophize and use cognitive biases such as an inflated sense of responsibility); personality factors (a sense of incompetence, high levels of neuroticism, and extreme introversion); and stimulus factors (sudden and intense stimuli, unfamiliarity, signals of danger, irregularity of stimuli, and large chunks of stimuli).

This theory suggested a way to measure the severity of PTSD by measuring the extent and impact of intrusive re-experiencing, but it does not succeed in explaining the other criteria for PTSD such as hyper-arousal and avoidance. It also fails to explain why different individuals, exhibiting the same factors, may display different symptoms and intensities of symptoms. In addition, there is no research support for Rachman's Emotional Processing Theory.

4.1.2 Ehlers and Clark's Cognitive Theory

According to Ehlers and Clark (2000), anxiety is based on the emotional appraisal of an *impending* threat in the present time. Anxiety in PTSD, on the other hand, is based on *memories* of trauma which was experienced *in the past.* The initial anxiety-provoking stimulus is therefore not present at the time of the current onset of anxiety in PTSD. Ehlers and Clark (2000) suggested that the PTSD patient is unable to see the trauma as a time limited situation which does not necessarily have adverse consequences for the future, but processes the information of the traumatic event in a manner that does make it a threat to him/her in the present.

The authors proposed a two-stage process model to explain why a sense of current threat may develop in one trauma victim and not in another. The proposed stages are related to (a) individual differences in the appraisal of the trauma and its sequelae, and (b) individual differences in the nature of the memory of the event and its link to the victim's other autobiographical memories. Ehlers and Clark (2000) argued that, in the first stage, individuals have characteristic idiosyncratic negative appraisals of the trauma and its possible after-effects, and this give rise to the current feeling of threat. This experienced threat could be internal (e.g., I am a bad person...) or external (e.g., The world is a dangerous place...). These negative appraisals may present in the form of either over-generalizations from one situation to another, in the process changing a previously safe situation into a potentially threatening situation, or in the form of exaggeration, greatly increasing the imagined likelihood of further trauma as well as the intensity of the remembered feelings and actions of the victim during the trauma. The victim may interpret flashbacks, lack of concentration, irritability, and mood swings (which are normal short-term reactions to trauma) as indications that he/she has permanently changed for the worst. Furthermore, victims' memories of the event underlie their appraisals of their emotional responses during or after the event, such as shame, guilt, fear, and anger. These negative appraisals of emotions may serve to exacerbate the inability to see the relevant trauma as a time-limited event (Ehlers & Clark, 2000). The second stage of this model relates to the memory of the trauma and its re-experiencing in a voluntary or involuntary manner. Ehlers and Clark (2000) maintained that unwanted intrusion and/or the problematic voluntary retrieval of some memories of the trauma are due to the way the trauma is processed in the victim's memory. The trauma is unsuccessfully placed in the correct context of time and space, because it is not successfully incorporated into the person's autobiographical memory base.

They suggested that difficulty with intentional recall can be accounted for by this poor semantic routing, and unwanted intrusions can be accounted for by the lack of time context that has arisen in the basic memory. They further argued that strong associations between stimuli and responses can increase the likelihood of intrusions and emotional responses. Strong perceptual priming to traumatic details of the event, as well as biased recall of these details, can be a further activator of re-experiencing from apparently cue-less situations.

4.1.3 Brewin, Dalgleish and Joseph's Dual Representation Theory

Brewin (1989) originally proposed a theory involving dual representations of the experienced trauma in memory. Brewin et al. (1996) expanded upon this basis to evolve the Dual Representation Theory. The first representation of the trauma consists of the conscious experiences of the trauma, which can be recalled and edited at will by the individual. The second representation encompasses the unconscious experiences of the trauma which cannot be recalled at will by the patient. The conscious, detailed representation of the trauma in memory is called Verbally Accessible Memory (VAM), and can be retrieved from the autobiographical memory at any time by the individual. These are memories with a complete personal context of past, present and future (Brewin & Holmes, 2003). These memories will include information about the meaning of the event, emotional and physiological information, as well as situational information. They are reasonably detailed but can be highly selective because high levels of anxiety during the trauma increase its attentional selectivity and lowers short-term capacity. The unconscious representation of the trauma in memory is called Situational Accessible Memory (SAM) and contains information of a lower level of processing of aspects that were too quick to be recognised by the VAM. These could include bodily changes such as heart rate or an increased rate of respiration, and they can not be retrieved at will (Brewin & Holmes, 2003). The SAM can only be accessed when in a context similar to that of the traumatic event. Brewin et al. (1996) described this context as either internal (thinking about the trauma) or external (hearing about a similar event). The SAM is much larger and less selective, and the meanings of the memories may be different than in the VAM.

According to Brewin et al. (1996) the process of emotional processing after trauma, the aim of which is to reduce negative affect and to restore a sense of safety and control, and which may have a negative or a positive outcome, can be explained through the use of VAM and SAM. The SAM provides the victim with sensory and physiological feedback of the event (known as flashbacks), bringing with it the

relevant emotional arousal given to it by the VAM. The VAM then attempts to accommodate this feedback or information by giving it meaning, cause, and blame where needed, as well as editing the autobiographical memory where needed (Brewin, 1989). This process then changes the content of the SAM so that its information does not automatically reactivate flashbacks. Brewin and Holmes (2003) stressed that this theory typifies PTSD as a hybrid disorder which potentially includes two pathological processes. The first process includes the resolution of negative beliefs and their emotions, which can be accounted for in the VAM, and the second process is the management of the flashbacks, which is the domain of the SAM. Recovery from trauma is dependant on the outcome of both these processes.

4.1.4 Foa's Emotional Processing Theory

Emotional Processing Theory, developed by Foa and Kozak (1986), is based on Lang's bio-informational theory of fear (Lang, 1977, 1979). This theory represents fear in memory as a network of structures made up of associated stimulus, response, and meaning elements, designed as a program to escape or avoid threatening danger (Rauch & Foa, 2006). A gun may, for instance, be included in the fear structure as a stimulus element. This would in turn be connected to various behavioural and physiological response elements, such as running away, hiding, sweating, heart palpitations, etc. It would also be connected to various meaning elements, such as 'I am going to die'. Whenever one or more of the fear stucture elements are matched by a stimulus in the environment, it is activated and the activation spreads throughout the whole fear network. This network includes stimulus information about the traumatic event, information about cognitive, behavioural, and physiological reactions to the trauma, and interoceptive information which forms a link between these stimulus and response elements (Brewin et al., 1996). Activation of this fear network by triggering stimuli (i.e. reminders of the trauma) causes information in the network to enter consciousness (the intrusion symptoms of PTSD). Attempts to avoid and suppress such activation lead to the avoidance symptoms of the disorder.

Foa and Kozak (1986) stated that pathological fear structures, which lie at the basis of the anxiety disorders, differ from normal fear structures in the sense that they involve excessive response elements as well as resistance to modification, and that reality is not represented accurately by the association among the different fear elements. In their application of the emotional processing theory to PTSD, Foa and Rothbaum (1998) proposed that the fear structure of PTSD includes excessive stimulus and

response elements as well as pathological meaning elements. A motor vehicle accident survivor may accurately associate driving fast with danger, but he may inaccurately associate red cars with danger because he was hit by a red car. In reality, however, red cars are no more dangerous than any other colour car. Similarly, the rape survivor who was raped at a dam may afterwards see all dams as dangerous and may completely avoid dams. Both these trauma survivors may have problematic meaning elements in their fear structures, such as 'I should have been able to prevent the trauma', or 'I am incompetent to handle stress'. Emotional Processing Theory, as applied to PTSD (Foa & Riggs, 1993; Foa & Rothbaum, 1998), postulates that the fear structures of trauma survivors with PTSD include two basic dysfunctional cognitions underlying the development and maintenance of PTSD. The first dysfunctional cognition is that the world is totally dangerous (e.g., It is dangerous to be alone). The second dysfunctional cognition is that oneself is totally helpless and incompetent (e.g., I can't handle any stress and my PTSD symptoms mean that I am going completely crazy).

Successful resolution of the trauma can only be accomplished by integrating the information in the fear structure with the existing memory structures. This integration firstly requires the activation of the fear network so that it becomes accessible for modification, and secondly, it requires the availability of information that is incompatable with the fear network so that the overall memory structure can be modified. However, the unpredictability and uncontrollability of the traumatic event make it difficult to assimilate, into existing memory, structures in which the world is controllable and predictable (Foa & Kozak, 1986; Foa & Riggs, 1993; Foa, Steketee, et al., 1989; Foa, Zinbarg, & Rothbaum, 1992). Furthermore, factors like the severity of the trauma disrupt the cognitive processes of attention and memory at the time of the event and this leads to the formation of a fragmented and disjointed fear network which is consequently difficult to integrate with existing organized structures. Effective psychosocial intervention will therefore require modification of the pathological elements of the fear structure. Foa and Kozak proposed two specifically necessary conditions for the therapeutic modification of a fear structure: (a) the fear structure must be activated, and (b) information which is incompatible with the elements of the fear structure must be presented and integrated into the fear structure to replace the pathological elements with realistic elements.

According to Foa and Cahill (2001) the processes operating in the natural recovery from trauma, are similar to those occurring in the successful treatment of PTSD. After traumatic experiences survivors view the world as extremely dangerous and themselves as incompetent to cope with stress. This

reflects fear structures that include stimulus elements which have become associated with a meaning of danger, even though they are objectively non-threatening, as well as responses that were probably adaptive at the time of the trauma and associated with the meaning of incompetence. Most survivors gradually find that these pathological elements are corrected during daily activities that reveal them to be ungrounded. Traumatized individuals who systematically avoid trauma-related thoughts and activities do not have the opportunity to incorporate reality-based, disconfirming information and therefore they maintain the pathological elements. In this chain of reactions chronic PTSD develops, feeds itself, and is maintained.

Effective treatment involves engaging the patient with the daily activities which he or she is continuously avoiding as well as with the traumatic memory, and in this way disconfirming the pathological elements of the fear structure. Exposure therapy therefore involves systematic, repeated confrontation with the traumatic memories (imaginal exposure) and with avoided trauma-related situations (in vivo exposure). According to the Emotional Processing theory, these exposures present patients with information which disconfirms the pathological elements of the fear structure, thereby ameliorating the PTSD symptoms. Foa and Rauch (2004), for example, showed that greater reduction in thoughts of incompetence and the dangerous nature of the world, following prolonged exposure therapy, is associated with greater reduction of PTSD symptoms.

4.2 EXPOSURE TREATMENT

4.2.1 Characteristics of Effective Exposure Treatment

Although there has been considerable debate on the exact nature of the core features of trauma memories (Ehlers et al., 2004), the preceding theories argued that the re-experiencing symptoms in PTSD are the result of the way in which trauma is encoded and organized in memory, and retrieved (Brewin et al., 1996; Ehlers & Clark, 2000; Foa & Rothbaum, 1998). Exposure treatment for PTSD involves systematic and repeated confrontation with the traumatic memories and with avoided trauma-related situations. This allows the fear structure in PTSD to be activated, and information, which is incorporated with the elements of the fear structure, to be presented and integrated into the fear structure.

According to Marks (1987) it is now generally accepted that, for the treatment of anxiety to be effective, it must contain an element of exposure. However, it may be argued that all psychotherapies, irrespective of theoretical framework, entails some form or variation of exposure. For example, in Rogerian psychotherapy the patient is expected to talk about his or her fears and anxieties in a supportive and non-threatening environment, thereby exposing the patient to what is feared. Systematic desensitization entails gradual exposure, while flooding/implosion encompasses maximum exposure. Modelling and social skills training also have an exposure ingredient.

The question arises as to what are the characteristics or modalities of effective exposure. Marks (1987) reviewed the outcome literature on exposure treatment for phobic and obsessive-compulsive disorders, and concluded that the following constitute the modalities of effective exposure treatment.

- Live exposure yields better results than fantasy exposure. Fantasy exposure is worth considering when the real stimuli evoking fear are not readily accessible for live exposure, but live exposure is still needed even after there has been complete habituation to fantasy exposure.
- Self-exposure is effective and yields results similar to therapist-aided exposure in fantasy and/or in vivo, particularly if an instruction manual for self-exposure is added.
- Longer duration of fantasy exposure (sessions longer than 50 minutes) yields better results.
- Massed sessions are better than spaced sessions.
- Improvement during exposure treatment seems to be enhanced by blocking escape and avoidance in both overt behaviour and covert thought.
- Both slow and rapid exposure works, but rapid exposure seems to work faster.
- Relaxation training does not enhance exposure outcome.
- Neither very low nor very high arousal enhances exposure. What does seem important, is maintenance of attention to fear cues (engagement) until discomfort dies down. Attention or engagement can be regarded as a minimum level of arousal for exposure to be effective.
- Contact with fear cues during exposure has to be emotional as well as physical. Exposure then becomes functional, letting the emotional information access central representations of fear so that they can be activated, processed, and habituated.
- Cognitive restructuring does not add to the value of exposure treatment.

4.2.2 Foa's Exposure Treatment for PTSD.

Variations in exposure treatment for PTSD (e.g., Cooper & Clum, 1989; Foa & Rothbaum, 1998; Keane et al., 1989; Richards et al., 1994) are largely determined by the extent to which they adhere to the modalities of effective exposure identified by Marks (1987). The manualized exposure treatment program for PTSD, developed by Foa and her colleagues, meets all of these criteria (except that it incorporates breathing retraining), and will now be described as it is used in the current study.

Foa and Rothbaum (1998) identified a range of mechanisms involved in the improvement of PTSD symptoms. Firstly, repeated *imaginal* reliving of the trauma is seen to promote habituation and reduce anxiety associated with the trauma memory, and also to correct the erroneous idea that anxiety is going to last forever unless avoidance or escape is practiced continuously. Secondly, deliberate confrontation with the feared memory blocks negative reinforcement associated with fear reduction following cognitive avoidance of trauma-related thoughts and feelings. Thirdly, reliving the trauma experience in a therapeutic and supportive setting incorporates safety information into the trauma memory, and helps the patient to realize that remembering the trauma is not dangerous. Fourthly, focusing on the trauma memory for a prolonged period helps the patient to differentiate the traumatic event from non-traumatic events. This serves to establish the trauma as a specific occurrance rather than a representation of a dangerous world and of an incompetent self. Fifthly, the process of imaginal reliving helps to change the meaning of PTSD symptoms from one of personal incompetence to one of courage and mastery. Sixthly, prolonged, repeated reliving of the traumatic event presents the opportunity to focus on details which underlie negative evaluations of the self, and to modify them (Foa, Keane, & Friedman, 2000).

As indicated, the practical exposure treatment developed by Foa and her colleagues, typically involves imaginal exposure, in which the patient recalls the traumatic memories in the therapists office. A rape victim, for example, is asked to go back in her mind to the trauma of the rape itself and to relive it in her mind. She is instructed to close her eyes and to describe the rape out loud in the present tense, as if it is happening right now. This narrative may be audio-taped, and the patient is requested to use the tape to practice imaginal exposure at home daily. Although this reliving procedure is usually painful

to the patient, it becomes less painful the more exposure is repeated and is essential for the emotional processing of the trauma. An additional motivation for repeating this reliving procedure is that many trauma victims regard the process of remembering the trauma as dangerous, and therefore avoid thinking or processing it. Imaginal reliving serves to dispel or disconfirm this mistaken belief of danger, and thus helps to reduce the associated PTSD symptoms.

Imaginal Exposure consists of the following:

- Present the rationale for imaginal exposure to the patient.
- Be alert to the patient's anxiety and give him/her re-assurance.
- Explain that the session will be audio-taped so that the patient can listen to it as homework.
- Ask the patient to close his/her eyes, and to recall the trauma vividly, speaking in the present tense.
- In the first and second exposure sessions, allow the patient to approach memories gradually. In the third session, begin asking directive questions.
- Every ten minutes, ask the patient for a rating of his/her anxiety level at that time on a scale from 0 to 100 (Subjective Units of Discomfort Scale; SUDS) and record it on the Therapist Imaginal Exposure Recording Form.
- Continue for 30 to 60 minutes.
- After exposure, allow time in the session for the patient to calm down. Use the breathing retraining method if necessary. Encourage the patient to talk about his/her reactions to reliving the assault.
- Be available to talk to the patient by telephone between sessions.

The alternative form of exposure, *in vivo* exposure or real-life exposure, involves actually confronting realistically safe places, situations, or objects, which may be reminders of the trauma. The confrontation is repeated until the reminders do not trigger strong emotions any more.

Foa and her colleagues use both imaginal exposure and in vivo exposure in their treatment protocol. The imaginal exposure forms part of the agenda of each treatment session, whereas the in vivo exposure is used as homework exercises.

Foa and Rothbaum (1998) describe the following steps for teaching this method:

- Present the rationale for *in vivo* exposure to the patient.
- Give concrete examples of habituation.
- Introduce the subjective units of discomfort (SUD) scale.
- Construct a hierarchy of avoided situations with a SUD rating for each.
- Work with the patient to develop homework assignments based on this hierarchy.
- Instruct patient in *in vivo* procedure:
    - Patient begins with situations that evoke moderate anxiety levels (e.g., SUD =50).
    - Patient puts herself/himself into an anxiety-provoking (but realistically safe) situation.
    - Patient records time and initial SUDs rating on the *In Vivo* Exposure Homework Recording Form.
    - Patient must remain in situation for 30 to 40 minutes or until anxiety decreases by at least 50%
    - Patient records endpoint SUD rating for the situation.

The therapeutic process should begin with a rationale being presented to the patient. Then, giving examples of the habituation process should serve to motivate the patient to practice *in vivo* exposure. Thereafter, the SUD scale is introduced in preparation for constructing a list of situations which the patient experiences as distressing. If the patient has difficulty in coming up with situations he/she is avoiding, a suggestion may be given from a prescribed list of situations which are typically avoided by assault victims, if this is applicable. Thereafter the process is systematically continued in order of the prescribed steps, as set out.

It is typical for patients to feel threatened by the prospect of imaginal exposure and of reliving of the trauma. The therapist should continuously reassure the patient e.g., 'You are doing fine. Stay with the image; I know this is difficult, but you are doing a good job; Stay with the image. You are safe here.'

During the third and subsequent imaginal exposure sessions the therapist should start identifying those aspects of reliving the trauma that produce the most anxiety for the patient. These memories are

referred to as 'hot spots'. They will need repeated exposure in order for habituation to occur and for exposure to be successful. During the last few sessions SUD ratings during imaginal exposure should range from 20 to 30, and finally between 15 and 20. At this point 'hot spots' should be peaks of anxiety with SUDs ratings over 20. These parts of the memory are then reviewed in repetitive manner (up to six or seven times) during a single session.

In these later sessions imaginal exposure should be conducted for 30 to 45 minutes with SUD scores recorded every 10 minutes. During the recollections eliciting intense distress (i.e. the 'hot spots') the patient can be encouraged to 'slow down'. In order to facilitate confrontation with fear-evoking cues during imaginal exposure, the patient may be asked questions such as 'What are you feeling?, Are you experiencing any physical reactions? Describe them. Where do you feel that in your body?'.

Problems may arise during imaginal exposure. A patient may have difficulty expressing his/her feelings, or the patient may be afraid to cry because she fears she may never be able to stop. Some patients may feel inhibited in the therapists office, worrying that other people outside the office might be able to hear them if they become emotional. Others have just become so successful in cutting theselves off from their feelings that they find it difficult to let go. In all these instances the therapist should reassure them and proceed with the review of the memory of the trauma. A patient may also engage in avoidant behaviour in order to maintain control. The patient becoming quiet or turning her head left and right, may be attempts to avoid certain parts of the memory. The patient should then be encouraged to confront the memory through questions like 'What's happening now?'. Another effective method is to suggest to the patient to use the 'slow motion technique' in her imagination to view the trauma. The patient should constantly be encouraged and reassured that whatever he/she remembers, it will not change what has happened. The patient should also repeatedly be assured that the tharapist will help him/her to handle the relived trauma memories. Foa and Rothbaum (1998) quoted one of their patients as describing each session of imaginal exposure therapy as 'peeling a layer off of an onion, and after a few sessions you get to the stinky part in the middle, and then it does not stink any more' (pp. 168).

After each session of reliving, the patient should be encouraged to sit in the waiting room for a few minutes to unwind. The therapist should praise the patient for showing courage. When homework is assigned, the therapist should discuss whether there is a private spot at home where the patient can

listen to the tape of the session. The patient should be reminded to record his/her anxiety levels and any comments every time he/she practices imaginal exposure at home.

Patients usually receive between 10 and 15 sessions in a program. In the final session, the benefit that has been received from the therapy is discussed, as well as the possible need for further additional treatment relating to other issues (Foa, 2006).

Some criticism emerged against exposure therapy:

- Some clinicians argued that encouraging trauma survivors to relive their trauma in imagination, is cruel and revictimizing. In reality, successful exposure treatment eventually *reduces* the survivor's intrusive memories and diminishes painful affect associated with those memories. It must also be remembered that the pain is already there. It is not created by the therapist, who must access the emotions to assist the patient in emotionally processing the memory so that it can become less painful.

- A second myth is that patients are reluctant or unwilling to participate in exposure treatment. On the contrary, the majority of patients have been willing to participate once the procedure had been explained. There is also not a higher dropout rate from exposure therapy than from other treatments (Foa et al., 1991).

- Another myth is that exposure therapy 'forces' the trauma survivor to recall the painful memory. However, to understand avoidance is to understand PTSD. The trauma memories intrude into consciousness because they have not been adequately processed. The survivors avoid them because they are painful, thereby preventing them to be processed. In order for trauma survivors to regain control over the traumatic memories, this vicious circle has to be broken. Exposure therapy is a very efficient and effective way to assist the suvivor to confront traumatic memories. The rationale and treatment is explained to the survivors, who can then make an informed choice about their treatment. The therapist is actually helping the patients to do what they could not do alone.

- It is sometimes argued that exposure does not allow trauma survivors to recover 'at their own pace'. These patients have not recovered when they were left to their own pace! An essential component of effective exposure therapy is the therapist's sensitivity to individual differences in speed of habituation and response to anxiety-provoking situations. At the same time, it is essential that the patients' exposures are long enough and frequent enough to allow adequate processing (Rothbaum & Schwartz, 2002).

4.3. RESEARCH ON EXPOSURE TREATMENT FOR PTSD

4.3.1 Gold Standards

The methodology of psychotherapy outcome studies has advanced considerably over the past years. Consequently, assessment and methodological strategies that were acceptable in earlier studies are no longer considered sufficiently rigorous. Foa and Meadows (1997) described a set of essential methodological criteria for treatment outcome studies, referred to as "Gold Standards" for treatment outcome research.

These methodological criteria, as described by Foa and Meadows (1997) and Foa et al. (2000), are listed below in order to allow an assessment of existing exposure outcome studies in terms of these criteria in the next section (4.2.2.).

- *Clearly defined target symptoms.* The mere experience of a trauma is not an indication for treatment in and of itself. Significant trauma-related symptoms, such as the presence of PTSD or other common reactions to trauma (such as depression), should be present to justify treatment. Whatever the target symptom or syndrome, it should be defined clearly so that appropriate measures can be employed to assess improvement. In addition to ascertaining diagnostic status, it is also important to specify a threshold of symptom severity as an inclusion criterion for treatment. According to Foa and Meadows (1997) including individuals with mixed PTSD symptoms may muddle treatment findings for two reasons: Firstly, it is more difficult to detect improvement in such individuals, Secondly, they are likely to exibit very

mild symptoms following treatment, simply because of their relatively low initial psychopathology. These two scenarios may lead to opposite biases, the first, minimizing treatment efficacy and the second inflating its effects. A related issue to target symptoms is the importance of delineating inclusion and exclusion criteria. Delineation of inclusion/exclusion criteria can be of assistance both in examining predictors of outcome and in evaluating the efficacy of the treatment and its generalizability beyond the studied sample. If a treatment is effective regardless of sample differences, it proves more robust.

- *Reliable and valid measures.* Once target symptoms have been identified and the population defined, measures with good psychometric properties should be employed. For studies targeting a particular diagnosis, assessment should include instruments designed to yield diagnosis as well as instruments that assess symptom severity.

- *Use of blind evaluators.* Early studies of treatment of traumatized individuals relied primarily on therapist and patient reports to evaluate treatment efficacy and thus introduced expectancy and demand biases into the evaluation. The use of blind evaluators is a current requirement for a credible treatment outcome study. Two procedures are involved in keeping an evaluator blind. First, the evaluator should not be the same person conducting the treatment. Second, patients should be trained not to reveal their treatment status during the evaluation so as not to bias the evaluator's ratings.

- *Assessor training.* The reliability and validity of an assessment depends largely on the skills of the evaluator; thus, training of assessors is critical and a minimum standard of evaluator competency should be specified. This includes demonstrating interrater reliability and calibrating assessment procedures over the course of the study to prevent evaluator drift.

- *Manualized, replicable, specific treatment programs.* It is also important that the treatment chosen is designed to address the target problem defined by the inclusion criteria. For example, if PTSD is the disorder targeted for treatment, employing a treatment developed for depression may not be appropriate despite the high prevalence of depression in patients with PTSD. Detailed treatment manuals are of utmost importance in evaluating treatment efficacy because

they help to ensure consistent and standardized treatment delivery across patients and across therapists, and afford replicability of the treatment to determine generalizability.

- *Unbiased assignment to treatment.* To eliminate an important source of bias, neither patients nor therapists should be allowed to choose the patient's treatment condition. Instead, patients should be assigned randomly to treatment condition, or assigned via a stratified sampling approach. This helps to ensure that observed differences or similarities among treatments are due to the techniques employed rather than to extraneous factors.

- *Treatment adherence.* The final component of a well-controlled study is the use of treatment adherence ratings. These ratings inform as to whether the treatments were carried out as planned, and whether components of one treatment condition drifted into another (Foa et al., 2000, pp. 10 – 11).

4.3.2 Outcome Studies on Exposure Treatment for PTSD

Exposure treatments, all involving the common feature of having patients confront their fears, vary on the dimentions of exposure (imaginal vs in vivo), exposure duration (short vs long), and arousal level during exposure (low vs high). Systematic desensitization, for example, is at the extreme of imaginal, brief and minimally arousing exposure, and in vivo at the other extreme on each dimention (Foa & Meadows, 1997). Some of the earliest studies of exposure treatments for PTSD incorporated systematic desensitization (e.g., Frank et al., 1988; Schindler, 1980). Although these studies reported some improvement in posttrauma symptoms, methodological limitations rendered these results inconclusive. One exception was the study by Brom et al. (1989). In this study, patients in the desensitization condition showed a mean improvement of 41% on the Impact of Events Scale, which was higher than the other treatments examined, although the difference did not reach statistical significance (Foa & Meadows, 1997).

Some earlier case reports and uncontrolled studies also examined implosive therapy or flooding, characterized by high arousal levels during exposure, as treatment for PTSD (e.g., Fairbank & Keane, 1982; Johnson, Gilmore, & Shenovy, 1982; Keane & Kaloupek, 1982), with promising results. Most

of these early treatments, however, included additional procedures such as anger control and relaxation training.

One of the earliest controlled studies of prolonged exposure was conducted with 16 combat veterans with PTSD symptoms, and compared standard psychosocial treatment and pharmacological treatment, with and without the adjunct of imaginal flooding (Cooper & Clum, 1989). The imaginal flooding sessions were flexible regarding number and duration of sessions, (six to fourteen sessions) but otherwise reasonably standardized. Patients who received imaginal flooding showed a greater reduction of some symptoms, such as nightmares, as well as less anxiety during a behavioural avoidance test, compared to those who received only standard treatment. The results showed that the addition of an exposure component to standard therapeutic treatment led to greater reduction of PTSD and associated symptoms. This study did not use blind evaluators.

Keane et al. (1989) conducted a controlled study of 24 Vietnam war veterans with combat-related PTSD, randomly assigned to a treatment or a waiting-list condition. The treatment program included relaxation training, practice in non-traumatic imagery, and flooding (imaginal exposure). The results indicated that flooding was associated with a decrease in anxiety and depressive symptoms, as well as a decrease in re-experiencing symptoms of PTSD, but it apparently had no effect on the numbing or avoidance symptoms. This study met several gold standards. PTSD symptoms were the treatment targets and were adequately assessed. Concomitant symptoms such as depression and anxiety were also assessed by means of psychometrically sound self-report instruments. Patients were randomly assigned, and treatment was manualized. However, no blind evaluators were used and no adherence ratings were provided, while patients in the waiting-list condition continued to receive other treatments during the waiting time (Foa & Meadows, 1997).

Boudewyns and Hyer (1990) randomly assigned patients to either direct therapeutic exposure (DTE) or a control condition consisting of traditional individual counseling. The exposure treatment consisted of 10 to 12 sessions (50 minutes each) of either imaginal or in vivo exposure. Both groups also participated in the standard inpatient milieu program in the special PTSD unit at the hospital. This study met a number of gold standards, e.g., inclusion and exclusion criteria were clearly defined, as were the target symptoms, and diagnosis was determined by structured interviews conducted by trained evaluators. Manualized treatment was conducted by trained therapists. However, a second similar

study by Boudewyns et al. (1990) did not meet several of the gold standards. There was no indication of the treatment adherence ratings. Whereas the treatment condition was clearly defined, the control condition appears very flexible, and it is possible that some control patients informally received some components of DTE (Foa & Meadows, 1997). No blind assessments were conducted. Both studies found that DTE was more effective than the control condition on self-reported psychological functioning, although not on physiological responding. Foa and Meadows (1997), however, concluded that the efficacy of DTE for PTSD cannot be ascertained from these studies, as improvement was defined by change on general measures of psychological functioning, rather than on a specific measure of PTSD.

In a study of 45 female victims of sexual or nonsexual assault Foa et al. (1991) randomly allocated the participants to three treatment groups and a waiting-list control condition. The three treatment groups comprised of prolonged exposure (including imaginal and in vivo exposure), stress inoculation training, and supportive counseling. Supportive counseling comprised of sessions during which patients were encouraged to talk about the traumatic experience, about their reactions and emotions, and during which the therapists reassured them that their reactions were understandable, and that they were not 'going mad', nor was there any reason to feel guilty. Treatment targets were PTSD symptoms, and a diagnosis of PTSD was a prerequisite. Symptoms were evaluated before and after treatment, and at follow-up by means of psychometrically sound interviews and self-report questionnaires, with the interviews conducted by trained clinicians who were blind to treatment conditions. Treatment comprised of nine 90 minute sessions over a period of five weeks. To avoid therapist confound variables, the same therapists conducted all three groups under supervision. Immediately after treatment both the stress inoculation training and prolonged exposure groups showed improvement on all three PTSD symptom clusters, with the improvement of the stress inoculation training group higher than that of the prolonged exposure group. The stress inoculation training group as well as the control group showed improvement on the arousal symptoms but not on the avoidance or the re-experiencing symptoms. At follow-up evaluation after three and a half months, prolonged exposure was shown to be the most successful treatment procedure on all measures of psychopathology. This was a well controlled study as it met all the gold standard criteria, except for the absence of a required minimum severity threshold and interrater reliability assessments.

Richards et al. (1994) treated 14 patients with PTSD due to shipwrecks, criminal assault, sexual assault or motor vehicle accidents, in two groups (four sessions of imaginal exposure followed by four sessions of in vivo exposure, or vice versa). Treatment as well as measuring was standardised, and several process measures such as Subjective units of discomfort ratings during exposure were included. At post-treatment evaluation and at follow-up after one year none of the patients complied with PTSD criteria. Both types of exposure were effective in reducing PTSD symptoms. In vivo exposure appeared more effective for avoidance symptoms regardless of whether it was conducted before or after the imaginal exposures. In this study, treatment protocols were clearly defined, and the targets of PTSD symptoms were adequately measured, with all patients meeting PTSD criteria at pre-treatment. However, the study did not include a control group, no measures of treatment adherence were reported, and no blind evaluations were included with which to judge outcome.

In a study similar to the Foa et al. (1991) study, Foa et al. (1995) compared prolonged exposure, stress inoculation training, a combination of these treatments, and a waiting-list control condition. At post-treatment all the treatments showed significant improvement in PTSD and depressive symptoms, with no improvement for the controls. These effects were maintained at follow-up after six months. An assessment of patients who reached good end-state functioning (defined by criterion scores on PTSD symptoms, and measures of anxiety and depression) showed that 21% of patients receiving stress inoculation training, 46% of patients participating in prolonged exposure, and 32% of patients in the combined treatment achieved this goal at post-treatment. At follow-up after six months, 75%, 68% and 50% of patients in the prolonged exposure, stress inoculation training, and combined groups respectively lost the diagnosis of PTSD. All patients in the control condition retained a PTSD diagnosis. This was also a well controlled study, meeting most of the gold standards, except for the absence of a required minimum severity threshold and interrater reliability assessments.

Thompson et al. (1995) conducted a study consisting of eight weekly sessions of imaginal and in vivo exposure with 23 victims of different traumas, all of whom conformed to the DSM-III-R criteria for PTSD based on the Clinician Administered PTSD Scale, as well as a minimum threshold of symptom severity. Patients improved significantly on several measures at post-treatment, showing reductions of 42% on the Impact of Events Scale, 61% on the General Health Questionnaire, 38% on the Symptom Checklist-90, and 35% on the Clinician Administered PTSD Scale. The study did not include a control group and no blind evaluators were used.

Marks et al. (1998), in a controlled study, compared four conditions: exposure therapy, cognitive restructuring, exposure therapy combined with cognitive restructuring, and relaxation training. Participants were 87 men and women, who had PTSD following a range of traumatic stressors. Seventy-seven (88.5%) completed treatment and 52 (59.8%) completed the the 36-week follow-up. The results showed the cognitive restructuring, exposure therapy, as well as the combined treatment to be overall more effective than relaxation training, but there were no major differences in the results of the three treatments. Treatment gains were maintained at follow-up after six months. This study met most of the gold standards, except that no indications of treatment adherence were reported.

In a further controlled study by Foa et al. (1999) prolonged exposure, stress inoculation training, and a combination of prolonged exposure and stress inoculation training were investigated. Ninety-six female assault victims with chronic PTSD were randomly assigned to one of four conditions, namely prolonged exposure, stress inoculation training, a combination of prolonged exposure and stress inoculation training, or a waiting-list control condition. Treatment consisted of nine individual sessions, conducted in accordance to treatment manuals. Independent evaluations were conducted at pretreatment, posttreatment, and 3-, 6-, and 12-months follow-up. In this study all three active treatments reduced the severity of PTSD and depression compared to the waiting-list condition, but did not differ significantly from each other. These gains were maintained throughout the follow-up period. This study was also a well controlled study and met all the gold standard criteria with the exception of specification of measures of adherence.

In a study by Taylor et al. (2003) sixty participants were assigned to one of three treatment conditions, namely prolonged exposure, relaxation training, or eye movement desensitization and reprocessing (EMDR). Compared with EMDR and relaxation training, exposure therapy produced significantly larger reductions in avoidance and re-experiencing symptoms. Exposure therapy also proved to be superior in terms of the rate at which avoidance symptoms were reduced. This study had some methodological problems: no control condition was included, no blind evaluators were used and no indications of treatment adherence were reported.

Exposure as a component of a combined treatment program has been studied by Echeburua, de Corral, Sarasua, and Zubizzaretta (1996), Echeburua, de Corral, Zubizzaretta, and Sarasua (1997), Frueh, Turner, Beidel, Mirabella, and Jones (1996), and Hickling and Blanchard (1997). Echeburua et al.

(1997) conducted a study to test the comparative effectiveness of two therapeutic modalities in the treatment of chronic PTSD in victims of sexual aggression, namely self-exposure and cognitive restructuring, and progressive relaxation training. Their sample consisted of 20 victims of adult rape or adult victims of childhood sexual abuse. Participants were selected according to DSM-III-R criteria for PTSD. A multigroup experimental design with repeated measures (pre-treatment, post-treatment, and at one, three, six, and 12 months follow-up) was used. Most treated patients improved, but the recovery rate was higher on all measures in the exposure and cognitive restructuring group at posttreatment and at follow-up.

Exposure therapy has more empirical evidence for its efficacy than any other treatment developed for trauma-related symptoms (Rothbaum et al., 2000). Foa (2006) concluded that the results of studies such as those described previously "show that programs of cognitive-behavioural therapy can be effective in the management of patients with PTSD. Prolonged exposure therapy, stress inoculation training, and cognitive therapy can all provide benefits to PTSD patients. Therapy that includes both in vivo and imaginal exposure produces excellent outcomes. However, prolonged exposure therapy is not enhanced by the addition of stress inoculaton training or cognitive therapy. Prolonged exposure is a safe treatment that is accepted by patients, and its benefits remain apparent after programs of therapy have finished" (Foa, 2006. p 42.).

There is also a need for further research on the effect of exposure therapy on brain functioning in PTSD. Schwartz et al. (1996), focusing on the treatment of obsessive compulsive disorder (OCD), investigated the effect of cognitive–behaviour therapy on brain functioning as measured by positron emission tomography (PET). This method is analogous to the method used in single photon emission computed tomography (SPECT). SPECT is founded on the assumption that some chemical compounds distribute to the brain in a way that is reflective of brain physiology, and that these compounds can be bound to radioactive elements whose emissions can be externally detected. SPECT is a method to determine the three-dimensional distribution of a radiotracer ("radiopharmaceutical") within the human body. The radiopharmaceuticals used in SPECT emit a single gamma ray, which is detected and its point of origin determined solely on the basis of its trajectory. SPECT imaging of regional cerebral blood flow (rCBF) is performed with a compound that flows into the brain with the arterial blood and is transferred across the blood-brain barrier in a manner dependant on the concentration gradient. Areas

receiving high arterial blood perfusion would receive large quantities of this compound and vice versa. By means of the SPECT imaging the rCBF, indicating brain activity, can therefore be recorded.
In previous studies (Schwartz et al., 1996) it was found that OCD symptoms coincided with significant correlations of brain activity between the orbital gyri and the head of the caudate nucleus and the orbital gyri and the thalamus on the right. According to PET scans these correlations decreased significantly after effective behaviour modification therapy. The question may therefore be posed whether PTSD symptoms will also coincide with increased brain activity, as indicated by Kaplan and Sadock (1998), and whether effective exposure therapy will correlate with changed brain activity as reflected by SPECT scans.

4.4 CONCLUSION

The preceding review of outcome studies on exposure treatment for PTSD showed that since 1989 about 11 outcome studies were published. These studies investigated the effectiveness of exposure treatment for PTSD resulting from a broad spectrum of traumas, including sexual assault, criminal violence, combat, motor vehicle accidents, etc. The results of these studies showed that the effectiveness of exposure treatment does not depend on the type of trauma preceding PTSD.

Inspection of the available studies revealed large variations in design. Only a few studies compared exposure treatment to a control condition (either a waiting-list condition or a standard psychosocial treatment or counseling). Most studies compared prolonged exposure to other cognitive-behavioural procedures such as stress inoculation training, cognitive restructuring, relaxation training, or combinations of these treatments.

In general, these studies are methodologically sound and to a large extent met the accepted methodological criteria for outcome studies, the so-called gold standards, described by Foa and Meadows (1997). The most common exceptions were the absence of a control condition, blind evaluators to assess treatment outcome not being used, ratings of adherence to treatment not being specified, and symptom severity ratings not provided.

However, all outcome studies on the efficacy of exposure treatment for PTSD were based on American and British samples. No outcome studies were reported in which the effectiveness of exposure treatment for PTSD was investigated in cultural contexts different from the USA or UK. Similarly, no studies, which investigated the possible effects of prolonged exposure treatment on brain functioning in PTSD, are currently available.

5 METHOD

5.1 PROBLEM STATEMENT, OBJECTIVES AND HYPOTHESES

All available outcome studies supporting the effectiveness of prolonged exposure as psychological treatment for PTSD were based on American and British samples. No studies were identified in which the effectiveness of exposure treatment for PTSD was investigated outside the USA or UK, also not in South Africa with its culturally diverse and less affluent population. It is generally accepted that cultural variables may impact on psychological procedures, and that a procedure valid and proven in one culture, may not be effective and valid in a different cultural context (APA, 2000; Cantlie, 1994; Eagle, 2002; Gerrity & Scurfield, 1996; Kakar, 1985; Nordstrom, 1992; Roland, 1996; Rosenthal & Friedman, 1992; Stamm & Friedman, 2000). There is consequently a need to investigate the effectiveness of exposure treatment for PTSD in non-American and non-British samples.

Secondly, the majority of studies investigating the effectiveness of exposure treatment for PTSD did so by examining possible changes in PTSD symptoms at post-treatment and follow-up, mostly using the Clinician-Administered PTSD Scale administered by blind evaluators. Only a few studies (e.g., Cooper & Clum, 1989; Keane et al., 1989; Foa et al., 1995) also examined the effect of exposure treatment on associated symptoms such as depression and anxiety, associated with PTSD, and dysfunctional beliefs, thereby providing insight into the process of change.

Thirdly, none of the available outcome studies investigated the possible effect of exposure treatment on brain functioning, Kolb (1987) suggested that possible changes in brain functioning associated with PTSD could be expected in regions that have previously been implicated in the pathophysiology of PTSD. Nadal and Moscovitch (1998) showed that the superior frontal regions, medial-temporal area, cerebellum, neocortex and hipocampus are all implicated in implicit memory and the retrieval of specific episodes and their contextual framework. It can therefore be expected that initial changes in these areas as shown on the pre-treatment SPECT scans may be reversed on the post-treatment SPECT scans. Schwartz et al. (1996) showed that symptoms of obsessive-compulsive disorder coincided with significant correlations of brain activity between the orbital gyri and the head of the caudate nucleus and the orbital gyri and the thalamus on the right. By means of positron emission tomography (PET scans) they were able to demonstrate that these correlations decreased significantly after effective behaviour modification for obsessive-compulsive disordered patients. The question may therefore be posed as to whether effective exposure

treatment for PTSD will similarly be associated with changed brain activity in the brain regions previously implicated in PTSD, namely the superior frontal region, the medial-temporal area, the cerebellum, neocortex, and the hipocampus.

Based on these considerations the objectives of the present study were to investigate the effectiveness of prolonged exposure

- in significantly reducing PTSD symptoms in a South African sample of female rape survivors;
- in significantly reducing the co-morbid symptoms of depression, anxiety, and dysfunctional cognitions, associated with PTSD; and
- in altering changed brain functioning in specific brain areas implicated in PTSD such as the superior frontal region, medial-temporal area, cerebellum, neocortex, and hippocampus.

The study therefore examines the following hypotheses:

Hypothesis 1 Prolonged exposure treatment will result in a significant reduction of all three symptom clusters of PTSD at post-treatment and this improvement will be maintained at follow-up after three months.

Hypothesis 2: Prolonged exposure treatment will result in a significant reduction of the associated symptoms of depression, anxiety, and dysfunctional cognition.

Hypothesis 3: Prolonged exposure treatment will result in altering changed brain functioning in specific brain areas implicated in PTSD such as the superior frontal region, medial-temporal area, cerebellum, neocortex, and hipocampus.

5.2 RESEARCH DESIGN

The current study is a randomized controlled trial and is based on a test-retest-follow-up design. Participants, who met the selection criteria, were randomly assigned to either a treatment group, who received prolonged exposure therapy, or a wait-list control condition, in which patients stayed untreated for the duration of the treatment program, after which they received the same treatment regime. Assessments were done by a trained independent evaluator for all participants before treatment, immediately after treatment, and for the treatment group again at follow-up after three months.

The study was designed to adhere to the methodological criteria or gold standards for treatment outcome research, described by Foa and Meadows (1997).

- The target symptoms in the current study were defined as the diagnostic criteria for PTSD as set out in the Diagnostic and Statistical Manual for Mental Disorders: Fourth Edition (DSM-IV-TR, APA, 2000). In addition, the minimum diagnostic requirements of the Clinician Administered PTSD Scale (CAPS, Blake et al., 1995) namely one or more scores of ≥ 1 for frequency and ≥ 2 for intensity on the re-experiencing symptom cluster, three or more scores of ≥ 1 for frequency and ≥ 2 for intensity on the avoidance symptom cluster, and two or more scores of ≥ 1 for frequency and ≥ 2 for intensity on the hyper-arousal symptom cluster, were used as the minimum diagnostic requirements for inclusion in the study.

- Well-known measuring instruments commonly used in most of the exposure treatment outcome studies were incorporated in the present study, including the Clinician Administered PTSD Scale (to determine a PTSD diagnoses and the severity of the symptoms) (Blake et al., 1995), the Structured Clinical Interview for DSM-IV (to ascertain whether any co-morbid psychiatric diagnosis was present, which would then exclude such a person from the study) (First, Gibbons, Spitzer, & Williams, 1996), the Beck Depression Inventory (to measure the presence of depressive symptoms) (Beck et al., 1988), the Beck Anxiety Inventory (to determine the participants' experience of symptoms of anxiety) (Beck et al., 1988), and the Weekly Ideas Questionnaire (to measure dysfunctional cognitions associated with PTSD) (Foa, Clark, Ehlers, Orsillo, & Tolin, 1999).

- Only one evaluator was used. At the time of the first assessment, participants had not been allocated to any of the two groups and therefore the assessor could not ascertain in this way to which group each participant belonged. Participants were instructed not to reveal their treatment or waiting-list status to the evaluator at the time of the post-treatment evaluation. Unfortunately, as only one assessor was used, it was not possible to determine inter-rater reliability.

- The assessor was a senior clinical psychologist with 16 years experience in clinical diagnosis, psychotherapy and training / supervision of students. She was also trained beforehand in the administration and scoring of the CAPS and the SCID.

- Manualized, replicable, specific treatment programs: The Prolonged Exposure Treatment Manual, developed by Foa, was used in the present study, with written permission by Foa. This manual was used by Foa in her own outcome studies in the USA. The manual gives detailed guidelines for conducting the therapy sessions.

- Unbiased assignment to treatment: Participants were randomly assigned to either the treatment or the control group. Pieces of paper with either "Treatment Group" or "Control Group" written on it were put into a container. With each new participant, a secretary drew a piece of paper from the container, indicating the group to which the patient was allocated.

- Treatment adherence: In order to ensure treatment integrity, audio recordings of each session were made. A random selection of these recordings was made by a senior clinical psychologist, experienced in exposure treatment, and verified against the treatment manual. In all cases it was determined that treatment was delivered according to the manual guidelines.

5.3 PARTICIPANTS

5.3.1 Description of Participants

Participants were 29 females who met the diagnostic criteria for PTSD (DSM-IV-TR, APA, 2000) (mean age = 24.69 years; *SD* = 5.87). In all cases PTSD developed as a result of a single traumatic event, namely sexual assault. They were randomly assigned to a treatment group (mean age = 24.9 years; *SD* = 5.84) and a waiting list control group (mean age = 24.43 years; *SD* = 6.12) of 15 and 14 participants respectively. The treatment group consisted of 11 coloured and four white participants and the waiting list controls of 11 coloured and 3 white individuals. All participants were single, except for one individual in the treatment group, who was married. Six participants (40%) of the treatment group and nine (64.3%) from the waiting list control group were unemployed at the time of the study. Of the treatment group 10 (66.7%) did not complete high school, three (20%) obtained a grade 12 education, and two (13.4%) completed a post-matric qualification. Of the controls nine (64.3%) did not complete high school, while four (28.6%) obtained a grade 12 education and 1 (7.1%) completed a post-matric qualification. Nine participants (60%) from the treatment group and eight (57.1%) from the control group reported a monthly household income of less than R5000 (approximately 700 US dollars), while on average four to five people were

dependent on this income. These dependents included the participants' children, siblings, nieces, nephews, parents and grandparents. According to the All Media and Product Survey (AMPS) of 2005, 78% of South African Households have a household income off less than R5000. The sample in the present study thus seem representative of the general South African population in terms of household income.

The time between the traumatic event and inclusion in the study ranged between one and 26 months, with an average of 4.5 months for the treatment group and 4.6 months for the waiting list control group. Only two participants (13.3%) from the treatment group and two (14.29%) from the control group met the criteria for chronic PTSD, namely symptoms lasting longer than six months (APA, 2000).

The first inclusion criterion was a psychiatric diagnosis of PTSD as assessed by the independent evaluator by means of the Clinician Administered PTSD Scale (Blake et al., 1995). The minimum diagnostic requirements of the Clinician Administered PTSD Scale, namely one or more scores of $\geq$ 1 for frequency and $\geq$ 2 for intensity on the re-experiencing symptom cluster, three or more scores of $\geq$ 1 for frequency and $\geq$ 2 for intensity on the avoidance symptom cluster, and two or more scores of $\geq$ 1 for frequency and $\geq$ 2 for intensity on the hyper-arousal symptom cluster, were used as the minimum diagnostic requirements for inclusion in the study.

Apart from a diagnosis of PTSD following an incident of sexual assault, participants were included in the study only if they were female, at least 18 years of age and able to converse in either English or Afrikaans.

The exclusion criteria for this study were the following:

- A current or previous diagnosis of psychiatric disorder other than a diagnosis of PTSD (including organic mental disorder, schizophrenia or paranoid disorders, major depressive disorder severe enough to require immediate psychiatric treatment, bipolar mood disorder, major depressive disorder accompanied by delusions, hallucinations or bizarre behaviour, current alcohol or drug abuse), as evaluated by the independent assessor by means of the Structured Clinical Interview for DSM-IV (First, Spitzer, Gibbon, & Williams, 1998)
- Assault by spouse or other family member
- Current use of psycho-active medication

- Pregnancy
- Lactation
- Any serious medical condition requiring treatment

5.3.2 Selection of Participants

Participants were recruited from local health professionals (including medical practitioners, psychologists and social workers), psychiatric hospitals, general hospitals, community health clinics, the South African Police Service, victim assistance agencies, as well as by means of advertisements in the local media (both radio and newspapers).

The researcher visited each of the referral sources, explained the objectives of the study as well as the treatment offered, and supplied the referral sources with forms for informed, written consent to disclose contact details of possible participants to the researcher. The referral sources contacted the researcher with the contact details of potential participants. The researcher then contacted the potential participant and arranged for the first meeting to explain the study. The researcher also maintained weekly contact with the referral sources to remind them of the study and to enquire about any potential participants.

Sixty-eight potential participants were originally recruited, of whom 40 met the inclusion criteria. Of these 40 participants, 11 (27.5%) dropped out. Two participants (5%) dropped out at the beginning of the process due to their fear of the SPECT scans. A further four participants (10%) dropped out due to transport problems. They were not able to attend regular sessions, as they were dependent on public transport. Most of the time they did not have the finances to pay for transport and when they had the money, the public transport was unreliable or unsafe. Two of these participants dropped out before the pre-treatment SPECT scans and the other two after sessions four and five, after very irregular attendance up to that stage due to transport problems. Three participants (7.5%) dropped out due to work circumstances. They were the sole breadwinners and the only source of income in households with up to eight people depending on that income. Therefore they could not just leave their work and their employers were not accommodating in terms of regular leave for treatment. Two of these dropped out after session three and one participant stopped the therapy after session seven, when she was offered permanent employment. Two participants (5%) dropped out due to the intensity of their psychopathology, especially the intensity of the avoidance symptoms. These two were not able to tolerate the initial heightened

anxiety due to the prolonged imaginal exposure. They dropped out after sessions two and five respectively, in other words after the start of the prolonged imaginal exposure sessions.

5.4 MEASURING INSTRUMENTS

Assessment at pre-treatment, post-treatment and at follow-up included structured interviews, conducted by an independent assessor, who was blind to treatment condition, as well as self-report questionnaires.

5.4.1 Structured Interviews

5.4.1.1 Clinician-Administered PTSD Scale (CAPS) (Blake, 1994, Blake et al., 1995)

The CAPS was developed at the National Center for Posttraumatic Stress Disorder and was designed to overcome the limitations of other available PTSD interviews (Blake, Weathers, Nagy, Kaloupek, & Klauminjer, 1990). The CAPS is intended for clinicians and clinical researchers who have a working knowledge of PTSD, and consists of 30 items, which assess all 17 symptoms of PTSD as described in the DSM-IV (APA, 1994), as well as a range of associated, frequently observed features. Included in the CAPS are ratings for social and occupational functioning and an assessment of the validity of the patient's responses.

The CAPS is a structured diagnostic interview, which provides both dichotomous and continuous scores for individual symptoms and for the disorder, thus providing information about the presence or absence of PTSD as well as the overall severity of symptomatology. The CAPS is unique in that it contains separate ratings for the frequency and intensity of each symptom. Symptom frequency and intensity are rated on separate 5-point Likert scales. Standard prompt questions and suggested follow-up questions are provided regarding both the frequency and the severity of each individual symptom (Blake, 1994).

Another key feature of the CAPS is that it contains specific anchors and behavioural references on which to base all ratings. This feature provides an explicit guide for the interviewer and is intended to increase reliability. Furthermore, the CAPS can also be administered by paraprofessionals, provided they have experience in diagnostic interviewing, are familiar with PTSD and its associated symptoms, and undergo thorough training in the CAPS.

If administered in its entirety (i.e., asking all the questions regarding associated features, functional impairments, and response validity ratings), the CAPS takes approximately one hour to complete. Psychometric data on the performance of the CAPS demonstrate unusual strength in distinguishing clinical cases from non-clinical cases of PTSD. Weathers and his colleagues (1992, 2001) found test-retest reliability correlations between 0.90 and 0.98 across three clinicians for 60 separate male veteran subjects. The internal consistency proved to be equally impressive, with alpha at 0.94 across all three primary symptom clusters. Weathers et al. (1992a, 1992b) indicated the following psychometric properties: the convergent validity of the CAPS was evident from correlations with other established measures of PTSD. The correlation of the CAPS was 0.91 with the Mississippi Scale, 0.77 with the Keane PTSD Scale of the MMPI-2, and 0.89 with the SCID-PTSD symptom score (Keane, Caddell, & Taylor, 1988; Keane, Malloy, & Fairbank, 1984). The CAPS was found to have 84% sensitivity, 95% specificity, 89% efficiency, and a kappa of 0.78 against the SCID, when used as a continuous measure. Using the CAPS as a diagnostic measure, a kappa of 0.72 was found, as compared with the SCID diagnosis. It seems clear from this data that the CAPS is considered to be a sound measure of PTSD with excellent psychometric properties, whether used as a diagnostic or a continuous measure (Blake et al., 1995; Blanchard, Hickling, Taylor, Loos, & Forneris, 1995).

5.4.1.2 Structured Clinical Interview for DSM-IV (SCID) (First et al., 1996)

The SCID is considered to be the most widely used semi-structured interview to assess Axis I and Axis II psychiatric disorders. It consists of separate modules for the most common diagnostic categories and provides information across a broad range of clinical conditions. The structure of the SCID is a trichotomous decision of each criterion item for the diagnosis: 2 = present, 1 = subthreshold, and 0 = absent. The numeric ratings have no quantitative value, but are simply codes to indicate the categorical decision. The SCID also has a standard format of "skip outs". These are decision rules that allow the interviewer to end administration of any particular module as soon as it is clear that the diagnostic criteria will not be met.

Although the current version of the SCID represents more or less the state of the art for establishing psychiatric diagnoses in a research setting, there is an absence of reliability and validity data regarding its use (Werner, 2001). Only weighted kappa across all diagnoses is available: 0.60 for patient samples and 0.37 for non-patient samples. In defence of the SCID, however, in many

studies (Foa & Tolin, 2000; Weathers, Ruscio, & Keane, 1999) it is used as the "gold standard" against which other diagnostic interviews are measured.

The primary limitation of the SCID is that it permits only a trichotomous rating of a symptom (i.e. present, sub-threshold or absent), placing clinicians in a forced choice situation.

In the current study, the SCID was used to identify any co-morbid psychiatric diagnosis, which would then exclude the potential participant from the study.

5.4.2 Self-report measures

5.4.2.1 PTSD Symptom Scale (PSS-SR) (Foa, Riggs, Dancu, & Rothbaum, 1993)

The PSS-SR is a 17-item self-report questionnaire to determine the presence as well as the severity of PTSD symptoms as experienced by a patient. Each item of the questionnaire corresponds to one of the seventeen DSM-III-R (APA, 1987) diagnostic criteria for PTSD. The severity of each item over the past two weeks is rated by using a 4-point Likert scale: 0 = not at all, 1 = a little bit, 2 = somewhat, 3 = very much. The diagnosis of PTSD is confirmed when at least one re-experiencing, three avoidance, and two arousal symptoms were endorsed by individuals, who were traumatised at least one month prior to the assessment.

Cronbach's alpha for the total score of the PSS-SR was 0.91, while alpha coefficients for the subscales were 0.78, 0.80, and 0.82 for the re-experiencing, avoidance, and arousal scales respectively. The test-retest reliability of the overall severity score of the PSS-SR was 0.77. The re-experiencing subscale showed a test-retest reliability of 0.66 ($p<0.001$), while the reliability coefficient of the avoidance subscale was 0.56 ($p<0.005$), and the test-retest reliability of the arousal subscale was 0.71 ($p<0.005$). The PSS-SR total score significantly correlated to all other measures with which it was compared: Rape Aftermath Symptom Test (RAST) 0.81 ($p<0.001$), Impact of Event Scale intrusion (IES) 0.81 ($p<0.001$), Beck Depression Inventory (BDI) 0.80 ($p<0.001$), State Trait Anxiety Inventory (STAI) Trait Scale 0.56 ($p<0.001$), Impact of Event Scale avoidance 0.53 ($p<0.001$), and State Trait Anxiety Inventory (STAI) State Scale 0.52 ($p<0.001$). When correlated with the SCID, the specificity of the PSS-SR was 100%, the positive predictive power was 100%, and the negative predictive power was 82%. Overall the PSS-SR proved to identify the PTSD status of patients with 86% accuracy (Foa et al., 1993).

5.4.2.2 Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Beck, Steer, & Garbin, 1988)

The BDI is a 21-item self-report inventory measuring an individual's experience of depression. The BDI is considered to be one of the most popular instruments for measuring the degree of depression in psychiatric patients diagnosed with depression (Piotrowski, Sherry, & Keller, 1985), and to identify depression in the normal population (Steer, Beck, & Garrison, 1986). In addition to the obvious advantages regarding time and cost, one of the most important advantages of self-report measures of depression is the fact that it eliminates the clinician's prejudice and expectations (Lader, 1981).

Each of the 21 symptoms is rated on a scale from 0 to 3. Beck et al. (1988) described the following cut-off points: <10 = none or minimal depression; 10 to 18 = light to moderate depression; 19 to 29 = moderate to severe depression; and 30 to 63 = severe depression. According to Beck (1967) the average BDI scores are as follows: minimal depression 10.9 (*SD* = 8.1), light depression 18.7 (*SD* = 10.2), moderate depression 25.4 (*SD* = 9.6), and severe depression 30.0 (*SD* = 10.4).

In an overview of research on the psychometric properties of the BDI over the past 25 years, Beck et al. (1988) observed that in all the studies included in the overview no distinction was made between the original version (Beck at el., 1961) and the revised version (Beck et al., 1979) of the BDI. In a study with 204 underate students Lightfoot and Oliver (1985) found a Pearson correlation of 0,94 between the 1961 and 1979 versions of the BDI. This brought them to the conclusion that the difference between the two versions is not substantial.

In terms of internal consistency, a meta-analysis of 25 studies by Beck et al. (1988) showed an average alpha co-efficient of 0,86 for psychiatric patients, and of 0,81 for non-psychiatric populations. To examine the stability of the BDI, Beck et al. (1988) described the results of ten studies. No meta-analysis could however be carried out, as the test-retest intervals varied from hours to weeks. For psychiatric patients in the studies, the Pearson correlations varied from 0,48 to 0,86, and for non-psychiatric participants it varied between 0,60 and 0,83. The high correlations for the non-psychiatric groups are an indication that the BDI demonstrated sufficient stability over a period of a week.

Moran and Lambert (1983) compared the BDI's content with the DSM-III criteria (APA, 1980) for depression and came to the conclusion that the BDI adequately reflects six of the nine DSM-III

criteria. Beck et al. (1988) stated that the BDI purposefully excludes questions on increased sleep and appetite. They argued that, as increased sleep and appetite very often occur in the normal population, the inclusion of these two symptoms would lead to a large number of "false positive responses". The BDI does not include questions on agitation, because this is a clinically observable symptom, which would not be applicable for a self-report instrument.

Beck et al. (1988) quoted 35 studies in which correlations between BDI scores and a variety of other measures of depression were reported. They came to the conclusion that the concurrent validity of the BDI is high and reported indications that the BDI showed stronger correlations with clinical assessments of depression in psychiatric samples than in normal populations.

Beck et al. (1988) reported several studies in which the BDI succeeded in distinguishing between psychiatric and non-psychiatric populations, i.e. where significant differences were found between the BDI scores of depressed patients, non-depressed patients (e.g., alcoholics), and normal subjects. Regarding the differentiation between different types of depression, Beck et al. (1988) reported one study in which the BDI significantly distinguished between Major Depressive Disorder (higher BDI scores) and Distymic Disorder (lower BDI scores).

Beck et al. (1988) concluded that the BDI has strong construct validity. This conclusion is based on the fact that BDI scores in numerous studies showed significant correlations with specific physiological, behavioural, and attitudinal variables, which could be expected to accompany heightened depression levels. The BDI correlated significantly positive with REM sleep retardation (indicative of a sleep disorder), suicidal behaviour, alcoholism, maladjustment, a variety of medical symptoms (e.g., headache and digestive problems), loneliness, and stress. Further significant correlations were found between BDI scores and self-reported anxiety (Baker & Jessup, 1980; DeLeon, Skodol, & Rosenthal, 1978). Steer, Beck, Riskind and Brown (1986) found that patients with depressive disorders (Major Depressive Disorder and Distymic Disorder) had significantly higher BDI scores (average = 26.37; $SD = 6.94$) than patients with a Generalised Anxiety Disorder (average = 14.46; $SD = 6.10$) ($t = 9.02$, $p < 0.001$).

Factor analysis (Clark, Cavanaugh, & Gibbons, 1983) showed that BDI scores represent one underlying general syndrome of depression, with three highly inter-correlating factors, namely negative self-evaluation, impairment of work performance, and somatic disorder.

5.4.2.3 Beck Anxiety Inventory (BAI) (Beck, Epstein, Brown, & Steer, 1988)

The BAI is a 21-item self-report inventory for measuring the severity of anxiety in psychiatric populations. Each of the 21 items on the BAI describes a common symptom of anxiety. The respondent is asked to rate how much he or she has been bothered by each symptom over the past week on a 4-point Likert scale ranging from 0 (not at all) to 3 (severely – I could barely stand it). The items are summed to obtain a total score ranging from 0 to 63.

The BAI was developed to address the need for an instrument that would reliably discriminate anxiety from depression while displaying convergent validity. The initial item pool of 86 items was drawn from three pre-existing scales, namely the Anxiety Checklist, the Physician's Desk Reference Checklist, and the Situational Anxiety Checklist (Beck, Brown, et al., 1988).

The BAI was proved to have high internal consistency (α = 0.92) and item-total correlations ranging from 0.30 to 0.71 with a median of 0.60. The test-retest reliability coefficient was established at 0.75. Furthermore the BAI showed good concurrent and discriminant validity. It was shown to be able to discriminate homogeneous and heterogeneous anxious diagnostic groups from other psychiatric groups. Correlations with measures of related constructs were shown to be generally positive and high, and those with unrelated constructs were low (Beck et al., 1988).

5.4.2.4 Weekly Ideas Questionnaire (WIQ) (Foa, Clark, Ehlers, Orsillo, & Tolin, 1999)

The Weekly Ideas Questionnaire (WIQ) is a 25-item self-report inventory measuring the appraisals of trauma and its sequelae involved in the development and persistence of PTSD (Foa et al., 1999). The respondents are asked to rate each item on a 6-point Likert scale from 1 (disagree strongly) to 6 (agree strongly). Low scale scores indicate stronger endorsement of negative cognitions.

The WIQ was developed, based on the recognition by Foa and Riggs (1993) and Foa and Rothbaum (1998) that two basic dysfunctional cognitions mediate the development of PTSD, namely, the world is completely dangerous, and one's self is totally incompetent. Foa and Rothbaum (1998) argued that if PTSD is mediated by these cognitive distortions, then successful treatment would correct these cognitions. Indeed, after cognitive-behavioural therapy, patients with chronic PTSD reported more positive views about themselves and about the world than before treatment (Foa & Rothbaum, 1998).

The WIQ showed good psychometric properties. Cronbach's alpha pointed to high internal consistency (α = .97) for the total score. The test-retest reliability coefficient for a one week retest interval was established at 0.74 for the total score and 0.85 for a three week retest interval. The WIQ has good discriminative validity. Mann-Whitney *U* tests indicated that traumatized individuals with PTSD scored lower on the WIQ than traumatized individuals without PTSD or non-traumatized individuals.

5.4.3 Single Photon Emission Computed Tomography (SPECT)

SPECT is an imaging technique to determine the three-dimensional distribution of a radiotracer (radio-pharmaceutical) within the human body. SPECT imaging of regional cerebral blood flow (rCBF) is performed with a radiotracer that flows into the brain with the arterial blood and is transferred across the blood-brain barrier in a manner dependant on the concentration gradient. Areas receiving high arterial blood perfusion would receive large quantities of this compound and vice versa. The radiopharmaceutical is highly lipophilic, ensuring rapid crossing of the blood-brain barrier during first pass. Once inside the neurons, it is converted to a hydrophilic species, which, not being able to cross the blood-brain barrier, remains trapped in the neurons (in direct proportion to rCBF at the time of injection) for up to eight hours after injection. By means of SPECT imaging the rCBF, indicating brain metabolic activity, can therefore be recorded.

Resting single photon emission computed tomography was conducted before and within one week of completing the prolonged exposure treatment intervention. Participants laid supine in a quiet dimly lit room for 30 minutes prior to injection of the radiopharmaceutical. Apart from administration of the injection by a physician, they remained alone in the room during this period. Participants were asked to remain at rest for 10 minutes after the injection of the radio-pharmaceutical to allow uptake of the radio-pharmaceutical in the brain. This was done to achieve a baseline state of rCBF, minimising the effect of random external stimuli on rCBF.

An injection of 555 MBq (15 mCi) of technetium-99m hexamethylpropylene amine oxime (Tc-99m HMPAO) was given into an arm vein through a previously placed intravenous cannula. After the 10 minute rest period, SPECT imaging of the brain was performed, with the participant's head supported by a headrest, using a dual detector gamma camera equipped with fanbeam collimators.

Data were acquired in the step-and-shoot mode, using a 360 degree circular orbit, with the detectors of the gamma camera as close as possible to the patient's head. The radius of rotation was noted for each participant and the same measurements were used for the follow-up. Data were acquired using a 128 x 128 image matrix in 3-degree steps of 15 seconds per step. Data were reconstructed by filtered back projection, using a Metz filter (power=5, FWHM=14mm). The Chang method (μ = 0.11/cm) was used for attenuation correction (Chang, 1978). The final reconstructed voxel size was 1.7x1.7x3.9 mm^3. Image files were changed from interfile to analyse format, using conversion.

5.5 PROCEDURE

A total of 68 individuals were referred by the referral sources for participation in the study. Each potential participant gave the referral source informed, written consent to disclose her contact details to the researcher. The researcher then contacted each individual and scheduled individual meetings with each one.

During this first meeting, the study was explained to the individual in terms of the following information:

- The purpose of the study is to determine the effectiveness and efficacy of exposure therapy for PTSD, as well as to determine changes in brain activity due to this treatment.
- Participation in the study is voluntary and no person will be forced or coerced to participate.
- An independent evaluator will conduct the clinical evaluations in order to determine whether the potential participants meet the inclusion criteria of the study.
- In case it is found that a potential participant does not meet the criteria for inclusion in the study, she will be referred for appropriate treatment.
- If the potential participant meets the criteria for inclusion in the study, a SPECT scan will be arranged at the Department of Nuclear Medicine, Tygerberg Hospital. A SPECT scan entails the following:
    - The purpose of a SPECT scan is to determine change in brain metabolic activity by measuring the amount of blood flow to specific areas of the brain.
    - The participant lies on a bed and the doctor sets up an intravenous infusion of normal saline.

- After 30 minutes, when a resting baseline state has been obtained, the doctor injects the radioactive substance into the infusion. The amount of radiation the participant is exposed to is equivalent to the amount of radiation exposure associated with a chest X-ray series.
- Ten minutes later the intravenous infusion is removed.
- The gamma camera is then set up and the scanning proceeds, lasting approximately 30 minutes.
- The participant is never left alone, but is always accompanied by a member of the team at the Department of Nuclear Medicine.

- Participants are then randomly allocated to the treatment or the wait-list control group.
- If a participant is allocated to the treatment group, treatment will start the following week for nine consecutive weekly sessions of 90 minutes each.
- If a participant is allocated to the wait-list control group, she will receive no treatment for the following nine weeks. The participant is given the researcher's contact number in case of any emergency during this period.
- After completion of the treatment programme and the waiting period, a second evaluation is done by the independent evaluator.
- Immediately after this evaluation a second SPECT scan is arranged and performed, as described above.
- Participants in the wait-list control group will now receive the same treatment programme as the treated group.
- If a participant is allocated to the treatment group, a follow-up evaluation is done by the independent evaluator three months after the end of the treatment programme.
- No payment is given to participants and treatment is free of charge.
- All information gathered during the study is confidential. Data from the study may be published, but participants' identities will not be revealed.
- Participants will have access to the processed results after completion of the study.
- Participants may at any stage withdraw from the study and this will not negatively influence any treatment possibilities in future.

After this explanation, written, informed consent to participate in the study was obtained from each individual. At this stage nine of the 68 referred individuals (13.24%) decided not to participate in the study.

Individuals were then individually assessed by the independent evaluator in terms of the inclusion criteria using the CAPS (to determine the presence of a PTSD diagnosis), and the SCID (to exclude any co-morbid diagnosis). Of the remaining 59 potential participants, seven (11.86%) did not attend the evaluation appointments despite several reminders. Of the remaining 52 referrals, 40 individuals met the inclusion criteria of the study and were randomly assigned to the treatment and waiting-list control groups, with 20 participants allocated to each group.

A SPECT scan was then arranged for each participant. This was done in collaboration with the Department of Nuclear Medicine of the Health Sciences Faculty at Stellenbosch University. Of the 40 participants who met the inclusion criteria, only 21 received SPECT scans before the commencement of treatment or the waiting period. This was due to several reasons. Firstly, two participants were scared of the prospect of the scan and therefore dropped out even before the treatment process started. A further two participants did not attend the scan appointment due to transport difficulties, and dropped out of the project. A third reason for the lower numbers of pre-treatment scans was the fact that the radiopharmaceutical (HMPAO) was not available for a lengthy period of time due to withdrawal by the providers and therefore 15 patients could not receive the initial SPECT scans.

Immediately following the SPECT scan, treatment, or the waiting period for the control group, commenced for each individual patient. Treatment consisted of nine consecutive weekly sessions of 90 minutes each of individual exposure therapy, as described in 4.7. Treatment was conducted by the researcher according to Foa's Manual for Prolonged Exposure, as described in Foa et al. (1991). To ensure treatment integrity, sessions were recorded onto audiotape, with the informed, written consent of participants. These tapes were individually assessed by a senior clinical psychologist, experienced in cognitive-behaviour therapy and exposure treatment in particular. The assessor indicated that he was satisfied that treatment adhered to the treatment manual.

Of the 40 patients who met the inclusion criteria for the study, only 29 completed the study. Of these, 15 were part of the treatment group and 14 were in the wait-list control group. As indicated, two participants dropped out at the beginning of the process due to their fear of the SPECT scans. Four participants dropped out due to transport problems: two of these dropped out before the first SPECT scan and the remaining two dropped out after sessions four and five. These four participants showed irregular attendance up to that stage due to transport problems. A further three participants dropped out due to work circumstances. They were the main breadwinners and the

only source of income in their households with up to eight dependents. Of these three, two dropped out after session three and one dropped out after session seven, when she was offered permanent employment. Two participants dropped out after sessions three and five as a result of the severity of their psychopathology, as they could not tolerate the initial heightened anxiety due to the prolonged exposure.

One week after completion of treatment, participants in both the treatment and the control conditions were again assessed by the independent evaluator, using the CAPS to determine the participant's diagnostic status. Participants were requested not to reveal their treatment or control status to the evaluator to ensure blind assessments. Only eight of the 29 participants returned for the second SPECT scan. The main reason given for not attending was that the scan process made them anxious and they were now feeling well and did not want to feel anxious again. Of these eight participants, who attended the second scan appointment, two revealed at the appointment that they were pregnant and therefore they could not be subjected to the scans, while the scan data of two participants were lost during an upgrading of the computer system at the Department of Nuclear Medicine at Tygerberg Hospital. Therefore, at the end of the study, only four SPECT scans were available for analysis. All four of these participants were part of the treatment group.

After the post-treatment / post-waiting period assessments, participants in the waiting-list control group received the same manualised prolonged exposure therapy individually.

Follow-up assessments of participants in the treatment group were done three months after termination of treatment. These assessments were done by the same independent evaluator, using the CAPS, to determine participants' diagnostic status at this stage.

The procedure is graphically represented in Figure 1.

	Assessment 1	T1	T2	T3	T4	T5	T6	T7	T8	T9	Assessment 2	Assessment 3
Treatment Group	CAPS SCID SPECT		PSS-SR BDI-II BAI		PSS-SR BDI-II BAI		PSS-SR BDI-II BAI		PSS-SR BDI-II BAI		CAPS SPECT	CAPS
Control Group	CAPS SCID SPECT										CAPS SPECT	Treatment (T1 – T9)

Figure 1. Schematic Representation of Research Procedure.

5.6 TREATMENT

Treatment was provided by the researcher, a female clinical psychologist with training and experience in cognitive-behaviour therapy, and particularly in exposure treatment. Treatment was conducted according the Prolonged Exposure Treatment Manual for PTSD, developed by Foa et al. (1997). Permission to use this manual was granted by Professor Edna Foa. This manual was devised as a guide to therapists to implement a brief cognitive-behavioural treatment programme for PTSD, consisting of prolonged imaginal and in vivo exposure. Treatment included the following procedures: education about common reactions to trauma, breathing retraining, repeated prolonged imaginal exposure to the traumatic memories, and repeated in vivo exposure to situations the patient is avoiding because of trauma-related fear. This manualized treatment programme consists of nine consecutive weekly treatment sessions of 90 minutes each. The manual provides the option of adding three additional sessions for those patients who do not show at least 70% improvement in severity of PTSD symptoms by session eight.

Session one began by presenting the patient with an overview of the treatment programme and a general rationale for prolonged exposure. The next part of the session focused on the collection of information about the trauma as well as the patient's reactions to it. Collection of this information was done according to the Standardized Assault Interview, developed by Foa et al. (1997), and which is included in the manual. This was followed by the introduction of breathing retraining. The role that breathing play in how people feel as well as the importance of exhaling slowly was explained to the patient. Correct breathing was demonstrated to the patient, after which she was instructed to take a normal breath and to exhale slowly while saying the word "calm" or "relax". She is then requested to pause and slowly count to four before taking the next breath. The patient was asked to repeat the full sequence 10 to 15 times. An audio-recording was made of this breathing exercise. At this point the handout Breathing Retraining was given to the patient. For homework, she was asked to review the Rationale for Treatment handout given to her, and to practice breathing retraining on a daily basis for 10 minutes three times per day..

Session two started with a review of the homework from session one. The patient was then asked to talk in detail about the trauma and it's effect on her. Common reactions to sexual assault were discussed using the Common Reactions to Assault handout from the manual. This discussion was didactic and interactive. The following common reactions to assault were highlighted: fear and anxiety, corresponding experiences of changes in the bodily sensations, feelings, and thoughts, triggers or cues (e.g., time of day, places, smells, men approaching, etc.) activating the fears, re-

experiencing or reliving the trauma in the form of flashbacks, nightmares, and intrusive memories of the event, difficulty in concentrating, arousal, agitation, feeling jittery, feeling overly alert, being easily startled, and having trouble sleeping, feelings of irritability, the fight or flight reaction, the tendency to avoid people, places or things that remind of the event, emotional numbness; sadness and feeling depressed, loss of interest in pleasurable activities, feeling of loosing control, feelings of guilt and shame about actions or lack of actions, feelings of anger, negative self-image, disruption in relationships, loss of interest in sexual activities, and activating memories to past traumatic experiences.

At this point the Common Reactions to Assault handout was given to the patient and she was instructed to read it daily and to share it with significant others.

The treatment programme was then outlined and the general rationale for exposure treatment was presented, namely the importance of confronting avoided material in order to give the mind the opportunity to process the traumatic experience. The therapist and patient then jointly constructed a hierarchy of real-life situations that the patient was avoiding. The objective safety of each of thse situations was evaluated. The Subjective Units of Discomfort Scale (SUDS) was introduced and each of the situations was rated according to the SUD scale. The patient would begin by confronting the least threatening and anxiety provoking situation on the hierarchy by means of in vivo exposure after the session. The procedure of in vivo exposure was then explained to the patient, namely to enter the avoided situation and remain in that situation for 30 to 45 minutes or until her SUDS score decreased by at least 50%. This session was concluded by identifying in vivo assignments for homework. The patient was encouraged to continue with the breathing exercises throughout the day when she felt anxious, and to read the Common Reactions to Assault handout daily.

Session three started with a discussion of the homework from session two. The patient was then presented with the rationale for prolonged imaginal exposure. It was explained to her that the first reaction to trauma is to want to forget it or avoid being reminded of it. However, this avoidance does not make the memories disappear, but rather reinforces the intrusive re-experiences of the trauma. It was explained that the goal of this treatment is to help her process the memories related to the trauma by having her remember them for an extended period of time. Staying with these memories instead of running away from them will help decrease the anxiety and fear. She was told that the aim of the treatment was thus to have her control the memories, rather than the memories

controlling her. During imaginal exposure, the patient was instructed to relive the trauma in her imagination for approximately 60 minutes, while she told the therapist what she was reliving. She was instructed to imagine the event in her mind as vividly as possible, as if it was happening there and then, and to tell it to the therapist in the present tense, in order to help her re-experience the full intensity of the event. Throughout the imaginal exposure the patient was asked for ratings of her anxiety at the time on the SUDS scale. She was also asked to score the vividness of the image on a scale from 0 to 100. By using these scores, the patient's progress could be monitored in terms of reduction in anxiety following exposure. After 60 minutes the participant was asked to terminate the imaginal exposure by opening her eyes and taking several deep breaths. Assigned homework included practicing the breathing exercise, listening to the audiotape of the imaginal exposure daily, and continuing with the in vivo exposure.

Sessions four to nine (or 12) started with a discussion of the homework from the previous week. The prolonged imaginal exposure was then resumed for 45 minutes per session. The remaining time of each session was spent discussing the in vivo exposure and the homework assignments. As treatment progressed, the patient was encouraged to describe the assault in much more detail during the imaginal reliving. During session eight, the patient's progress in treatment was assessed by means of the SUDS ratings and the PSS-SR, BDI, and BAI scores in order to determine the reduction in severity of PTSD and related symptoms. Based on these measures, it was decided whether to continue with treatment to session 12, or whether to terminate after session nine. For all patients in this study, treatment was terminated after session nine.

During the final treatment session, the homework of the previous week was discussed. This was followed by 30 minutes of imaginal exposure and a discussion of the in vivo exposure. The progress made during treatment was then reviewed, followed by a discussion of follow-up assessments and treatment termination.

6. RESULTS

6.1 OBJECTIVES AND HYPOTHESES

The objectives of the present study were to investigate the effectiveness of prolonged exposure

- in significantly reducing PTSD symptoms in a South African sample of female rape survivors;
- in significantly reducing the associated symptoms of depression, anxiety, and dysfunctional cognitions, and
- in altering changed brain functioning in specific brain areas implicated in PTSD such as the superior frontal region, medial-temporal area, cerebellum, neocortex, and hipocampus.

The study examined the following hypotheses:

Hypothesis 1: Prolonged exposure treatment will result in a significant reduction of all three PTSD symptom clusters at post-treatment, and this improvement will be maintained at follow-up after three months.

Hypothesis 2: Prolonged exposure treatment will result in a significant reduction of the associated symptoms of depression, anxiety, and dysfunctional cognitions.

Hypothesis 3: Prolonged exposure treatment will result in altering changed brain functioning in specific brain areas implicated in PTSD such as the superior frontal region, medial-temporal area, cerebellum, neocortex, and hipocampus.

6.2 STATISTICAL ANALYSIS

An inspection of the data revealed that it did not meet the assumptions of normality. Data transformations such as log transformation, square root transformation, and reciprocal transformation did not have the necessary effects. Consequently, and also due to the small samples, nonparametric procedures (including the Mann-Whitney U Test, the Friedman Repeated Measures Test, and the Wilcoxon Test) were used to analyze the data.

The Mann-Whitney U Test for independent samples was used for comparisons of the treatment and control groups. Friedman's Repeated Measures Test was used to determine whether there were

significant differences overall between the consecutive measures of the treatment group. In case of significant differences, the Wilcoxon Test was used to determine between which consecutive measures significant differences exist. For the Wilcoxon Test, the Bonferroni correction was applied with regard to the α-level to control the family-wise error rate. A difference was only accepted as significant if its p-value is less than α (.05) divided by the number of comparisons. In the case of the CAPS scale there were two post-hoc comparisons. The reported α-level must therefore be compared to .025 in stead of the usual .05, and in the case of the measures of posttraumatic symptoms, depression, anxiety, and dysfunctional thoughts there were three post-hoc comparisons. Consequently, the reported α-level must therefore be compared to .017 (Field, 2005).

The data was analyzed by means of the Statistical Package for the Social Sciences (SPSS) (Nie, Hull, Jenkins, & Steinbrenner, 1975).

Statistical analyses of the SPECT scans were conducted on a voxel-by-voxel basis using Statistical Parametric Mapping (Friston, Frith, Liddle, & Frackowiak, 1991), and a MATLAB routine written to perform a statistical subtraction between two coregistered SPECT scans. The realign function was used to co-register baseline and post-treatment SPECT images, and generate a mean image for each participant. The realigned mean images were then normalised to the Montreal Neurological Institute (MNI) standard anatomical space with 4x4x4 mm^3 voxels, and to a value of 50 using proportional scaling. This was achieved using 12 affine transformations and 7x8x7 non-linear basis functions. This transform function was then applied to the separate realigned images for each subject. The normalised images were then smoothed using a 3D Gaussian kernel with a FWHM of 12mm. A subtraction analysis of the spatially normalised images was then performed for each individual. Using a z score threshold of 2 and a minimum spatial extent of 10 voxels, clusters of increased and decreased regional perfusion were generated for each participant, and superimposed onto a T1-weighted MRI study in MNI space using MRIcro. Statistical Parametric Mapping was used because of the small sample size, although it is becoming the preferred method of analysis even for larger sets of data.

6.3 COMPARISON OF TREATMENT AND CONTROL GROUPS ON SCORES ON THE CLINICIAN ADMINISTERED PTSD SCALE

The means and standard deviations for scores on the Clinician Administered PTSD Scale at pre-treatment, post-treatment and follow-up are shown in Table 1 for the treatment group and Table 2 for the control group.

Table 1

Means and Standard Deviations for Scores on the Clinician Administered PTSD Scale at Pre-treatment, Post-treatment, and Follow-up for the Treatment Group (n = 15)

	Pre-treatment		Post-treatment		Follow-up	
	M	*SD*	*M*	*SD*	*M*	*SD*
Total Severity Score	77.80	5.92	10.13	2.13	7.93	1.44
Re-experiencing Score	24.27	2.28	3.80	0.78	2.60	0.74
Avoidance Score	26.47	3.11	2.93	0.80	2.67	0.62
Arousal Score	27.07	2.63	3.40	1.30	2.67	0.82

Table 2

Means and Standard Deviations for Scores on the Clinician Administered PTSD Scale at Pre-treatment and Post-treatment for the Control Group (n = 14)

	Pre-treatment		Post-treatment	
	M	*SD*	*M*	*SD*
Total Severity Score	72.07	2.81	69.14	2.91
Re-experiencing Score	23.29	2.70	21.14	2.18
Avoidance Score	25.00	1.57	26.14	1.61
Arousal Score	23.79	1.81	21.86	1.66

The treatment and control groups were compared in terms of their scores on the Clinician Administered PTSD Scale at pre-treatment and post-treatment by means of the Mann-Whitney U test. These results are shown in Table 3.

Table 3

Comparison of Treatment (n = 15) and Control Groups (n = 14) on Scores on the Clinician Administered PTSD Scale at Pre-treatment and at Post-treatment

		Total	Re-experiencing	Avoidance	Arousal
Pre-treatment	Mann-Whitney				
	U	45.50	83.00	64.50	27.00
	z	2.61	0.97	1.79	3.43
	p	.009	.332	.074	.001
Post-treatment	Mann-Whitney				
	U	.00	.00	.00	.00
	z	4.60	4.63	4.63	4.66
	p	.000	.000	.000	.000

Table 3 shows significant differences between the treatment group and the control group on the total CAPS score and the CAPS score for the arousal symptom cluster at pre-treatment. From table 3 it is clear that there were also significant differences between the treatment group and the control group on all the post-treatment measures at post-treatment. On the total CAPS scores at post-treatment, the treatment group (M = 10.13; SD = 2.13) scored significantly lower than the control group (M = 69.14; SD = 2.91), with Mann-Whitney U = 0, z = 4.60, $p < .01$. The treatment group (M = 3.80; SD = .78) scored significantly lower than the controls (M = 21.14; SD = 2.18), with Mann-Whitney U = 0, z = 4.63, $p < .01$ on the CAPS scores for the re-experiencing PTSD symptom cluster at post-treatment.

When examining the CAPS scores for the avoidance PTSD symptom cluster at post-treatment, it is clear that the treatment group (M = 2.93; SD = .80) scored significantly lower than the control group (M = 26.14; SD = 1.61) (with Mann-Whitney U = 0, z = 4.63, p < .01). A significant difference is also evident when the CAPS scores for the hyperarousal PTSD symptom cluster at post-treatment are examined. The treatment group (M = 3.40; SD = 1.30) scored significantly lower than the control group (M = 21.86; SD = 1.66) (with Mann-Whitney U = 0, z = 4.66, p < .01).

Scores on the Clinician Administered PTSD Scale at the three stages of measurement were compared by means of Friedman's Repeated Measures Test for the treatment group. The results are shown in Tables 4 to 11.

Table 4

Comparison of the Total Severity Score on the Clinician Administered PTSD Scale at Three Stages of Measurement by means of the Friedman Test for the Treatment Group (n = 15)

Chi-Square	*df*	*p*
29.525	2	.000

Table 4 shows that there were significant overall differences between the total CAPS scores of the treatment group at pre-treatment (M = 77.80; SD = 5.92), post-treatment (M = 10.13; SD = 2.13), and at three month follow-up (M = 7.93; SD = 1.44).

In order to determine between which stages of measurement significant differences occurred, total severity scores were compared between stages of measurement by means of the Wilcoxon Test (Table 5).

Table 5

Comparison of Total Severity Scores on the Clinician Administered PTSD Scale at Different Stages of Measurement for the Treatment Group

	z	*p*
Pre - Post	3.41	.001
Post – Follow-up	3.30	.001

According to Table 5, using the Wilcoxon test (with a Bonferroni correction of $\alpha = .05$ to $\alpha = .025$ for significance to control for the family wise error rate), there was a significant decrease in the total CAPS score from pre-treatment to post-treatment, with $z = 3.413$; $p < .01$, as well as a significant decrease from post-treatment to three month follow-up, with $z = 3.330$; $p < .01$.

Table 6

Comparison of Clinician Administered PTSD Scale Scores for the Re-experiencing Symptom Cluster at Three Stages of Measurement by means of the Friedman Test for the Treatment Group ($n = 15$)

Chi-Square	*df*	*p*
29.103	2	.000

Tables 6 shows significant differences between the CAPS scores for the re-experiencing PTSD symptom cluster of the treatment group at pre-treatment ($M = 24.27$; $SD = 2.28$), post treatment ($M = 3.80$; $SD = .78$), and three month follow-up ($M = 2.60$; $SD = .74$).

Table 7

Comparison of Clinician Administered PTSD Scale Scores for the Re-experiencing Symptom Cluster at Different Stages of Measurement for the Treatment Group

	z	p
Pre - Post	3.42	.001
Post – Follow-up	3.29	.001

Table 7 shows a significant decrease in CAPS scores for the re-experiencing PTSD symptom cluster from pre-treatment to post-treatment, with $z = 3.417$; $p < .01$, as well as a significant decrease from post-treatment to three months follow-up, with $z = 3.286$; $p < .01$.

Table 8

Comparison of Clinician Administered PTSD Scale Scores for the Avoidance Symptom Cluster at Three Stages of Measurement by means of the Friedman Test for the Treatment Group ($n = 15$)

Chi-Square	*df*	*p*
27.098	2	.000

Table 8 shows significant differences between the CAPS scores for the avoidance PTSD symptom cluster of the treatment group at pre-treatment ($M = 26.47$; $SD = 3.11$), post-treatment ($M = 2.93$; $SD = .80$), and at three months follow-up ($M = 2.67$; $SD = .62$).

Table 9

Comparison of Clinician Administered PTSD Scale Scores for the Avoidance Symptom Cluster at Different Stages of Measurement for the Treatment Group

	z	*p*
Pre - Post	3.41	.001
Post – Follow-up	1.63	.102

According to Table 9 a significant decrease in the CAPS scores for the avoidance PTSD symptom cluster from pre-treatment to post-treatment, with $z = 3.413$; $p < .01$ is evident. There were, however, no further significant decreases in CAPS scores for the avoidance PTSD symptom cluster from post-treatment to three months follow-up ($z = 1.633$; $p = .102$). It is however clear that the improvement from pre-treatment to post-treatment was maintained through the three month follow-up period.

Table 10

Comparison of Clinician Administered PTSD Scale Scores for the Hyperarousal Symptom Cluster at Three Stages of Measurement by means of the Friedman Test for the Treatment Group ($n = 15$)

Chi-Square	*df*	*p*
27.887	2	.000

As shown by Table 10 there were significant differences between the CAPS scores for the hyperarousal PTSD symptom cluster of the treatment group at pre-treatment ($M = 27.07$; $SD = 2.63$), post-treatment ($M = 3.40$; $SD = 1.30$), and at three month follow-up ($M = 2.67$; $SD = .82$).

Table 11

Comparison of Clinician Administered PTSD Scale Scores for the Hyperarousal Symptom Cluster at Different Stages of Measurement for the Treatment Group

	z	*p*
Pre - Post	3.43	.001
Post – Follow-up	2.60	.009

Table 11 shows a significant decrease in the CAPS score for the hyperarousal PTSD symptom cluster from pre-treatment to post-treatment, with $z = 3.431$; $p < .01$, as well as a significant decrease from post-treatment to three months follow-up, with $z = 2.598$; $p < .01$.

6.4 COMPARISON OF POSTTRAUMATIC SYMPTOMS, DEPRESSION, ANXIETY AND DYSFUNCTIONAL COGNITIONS AT DIFFERENT TIMES OF MEASUREMENT DURING TREATMENT

In order to investigate the process of change, four variables, namely posttraumatic symptoms, depression, anxiety, and dysfunctional cognitions, were measured at sessions two, four, six, and eight by means of the PTSD Symptom Scale – Self-Report (PSS-SR), Beck Depression Inventory II (BDI-II), Beck Anxiety Inventory (BAI), and Weekly Ideas Questionaire (WI). Results of these analyses are reported in Tables 12 to 23.

6.4.1 Posttraumatic Stress Disorder Symptom Scale – Self-Report (PSS-SR)

Table 12

Means and Standard Deviations for Scores on the PTSD Symptom Scale – Self-report at Different Treatment Sessions

	M	*SD*
Session 2	32.00	4.42
Session 4	26.60	4.14
Session 6	16.40	1.77
Session 8	5.73	1.39

Table 13

Comparison of Scores on the PTSD Symptom Scale at four Treatment Sessions by means of the Friedman Test

Chi-Square	*df*	*p*
45.000	3	.000

Table 13 shows significant differences between the PSS-SR scores of the treatment group at session 2 (M = 32.00; SD = 4.42), session 4 (M = 26.60; SD = 4.14), session 6 (M = 16.40; SD = 1.77), and at session 8 (M = 5.73; SD = 1.39).

Table 14

Comparison of Scores on the PTSD Symptom Scale at Sessions 2, 4, 6, and 8 for the Treatment Group

	z	*p*
Sessions 2 – 4	3.42	.001
Sessions 4 – 6	3.42	.001
Sessions 6 - 8	3.43	.001

Table 14 indicates significant decreases in the PSS-SR scores from treatment session 2 to session 4 (with $z = 3.42$; $p < .01$), from treatment session 4 to session 6 (with $z = 3.42$; $p < .01$), and from session 6 to session 8 (with $z = 3.42$; $p < .01$).

6.4.2 Beck Depression Inventory-II (BDI-II)

The results of the analysis of the Beck Depression Inventory (BDI) Scores at four treatment sessions are reported in Tables 15 to 17.

Table 15

Means and Standard Deviations for Scores on the Beck Depression Inventory-II at Different Treatment Sessions

	M	*SD*
Session 2	18.53	2.30
Session 4	12.87	2.36
Session 6	7.60	2.20
Session 8	4.13	1.51

Table 16

Comparison of Scores on the Beck Depression Inventory-II at four Treatment Sessions by means of the Friedman Test

Chi-Square	*df*	*p*
44.720	3	.000

Table 16 shows significant differences in BDI scores between sessions 2 ($M = 18.53$; $SD = 2.30$), 4 ($M = 12.87$; SD 2.36), 6 ($M = 7.60$; $SD = 2.20$), and 8 ($M = 4.13$; $SD = 1.51$).

Table 17

Comparison of Scores on the Beck Depression Inventory-II at Sessions 2, 4, 6, and 8

	z	*p*
Sessions 2 – 4	3.43	.001
Sessions 4 – 6	3.43	.001
Sessions 6 - 8	3.31	.001

The post-hoc comparisons, as shown in Table 17, indicated significant decreases in BDI scores from sessions 2 to 4 (with $z = 3.43$; $p < .01$), from sessions 4 to 6 (with $z = 3.43$; $p < .01$), and from sessions 6 to 8 (with $z = 3.31$; $p < .01$).

6.4.3 Beck Anxiety Inventory (BAI)

The results of the analysis of the Beck Anxiety Inventory (BAI) at four treatment sessions are presented in Tables 18 to 20.

Table 18

Means and Standard Deviations for Scores on the Beck Anxiety Inventory at Different Treatment Sessions

	M	*SD*
Session 2	33.27	3.54
Session 4	24.53	1.77
Session 6	16.00	1.20
Session 8	4.73	0.96

Table 19

Comparison of Scores on the Beck Anxiety Inventory at four Treatment Sessions by means of the Friedman Test

Chi-Square	*df*	*p*
45.00	3	.000

Table 19 shows significant differences in BAI scores between sessions 2 ($M = 33.27$; $SD = 3.56$), 4 ($M = 24.53$; $SD = 1.77$), 6 ($M = 16.00$; SD = 1.20), and 8 ($M = 4.73$; $SD = .96$).

Table 20

Comparison of Scores on the Beck Anxiety Inventory at Sessions 2, 4, 6, and 8

	z	*p*
Sessions 2 – 4	3.42	.001
Sessions 4 – 6	3.43	.001
Sessions 6 - 8	3.45	.001

Table 20 shows significant decreases in BAI scores from sessions 2 to 4 (with $z = 3.42$; $p < .01$), from sessions 4 to 6 (with $z = 3.43$; $p < .01$), and from sessions 6 to 8 (with $z = 3.45$; $p < .01$).

6.4.4 Weekly Ideas Questionaire

The results of the analysis of scores on the Weekly Ideas Questionnaire at four treatment sessions are presented in Tables 21 to 23.

Table 21

Means and Standard Deviations for Scores on the Weekly Ideas Questionaire at Different Treatment Sessions

	M	*SD*
Session 2	81.47	5.88
Session 4	94.07	4.20
Session 6	103.53	4.90
Session 8	115.00	2.65

Table 22

Comparison of Scores on the Weekly Ideas Questionaire at four Treatment Sessions by means of the Friedman Test

Chi-Square	*df*	*p*
45.00	3	.000

Table 22 shows significant differences in scores on the Weekly Ideas Questionaire (WI) between sessions 2 (M = 81.47; SD = 5.88), 4 (M = 94.07; SD = 4.20), 6 (M = 103.53; SD = 4.90), and 8 (M = 115.00; SD = 2.65).

Table 23

Comparison of Scores on the Weekly Ideas Questionaire at Sessions 2, 4, 6, and 8

	z	p
Sessions 2 – 4	3.42	.001
Sessions 4 – 6	3.41	.001
Sessions 6 - 8	3.42	.001

The post-hoc comparisons, shown in Table 23, indicate significant increases in scores on the Weekly Ideas Questionnaire from sessions 2 to 4 (with z = 3.42; p < .01), from sessions 4 to 6 (with z = 3.41; p < .01), and from sessions 6 to 8 (with z = 3.42; p < .01).

6.5 ANALYSIS OF SPECT DATA

As indicated in section 5.5, SPECT data of only four participants in the treatment group were obtained at pre-treatment as well as at post-treatment. The results from individual case evaluations of these four sets of SPECT data are reported below.

Statistical analyses were conducted on a voxel-by-voxel basis using Statistical Parametric Mapping (SPM99) (Friston et al., 1991), and a MATLAB routine written to perform a statistical subtraction between two coregistered SPECT scans. The realign function was used to co-register baseline and post-treatment SPECT images, and generate a mean image for each patient. The realigned mean images were then normalised to the Montreal Neurological Institute (MNI) standard anatomical space with 4x4x4 mm^3 voxels, and to a value of 50 using proportional scaling. This was achieved using 12

affine transformations and 7x8x7 non-linear basis functions. This transform function was then applied to the separate realigned images for each patient. The normalised images were then smoothed using a 3D Gaussian kernel with a FWHM of 12mm. A subtraction analysis of the spatially normalised images was then performed for each individual. Using a *z* score threshold of 2 and a minimum spatial extent of 10 voxels, clusters of increased and decreased regional perfusion were generated for each patient, and superimposed onto a T1-weighted MRI study in MNI space using MRIcro.

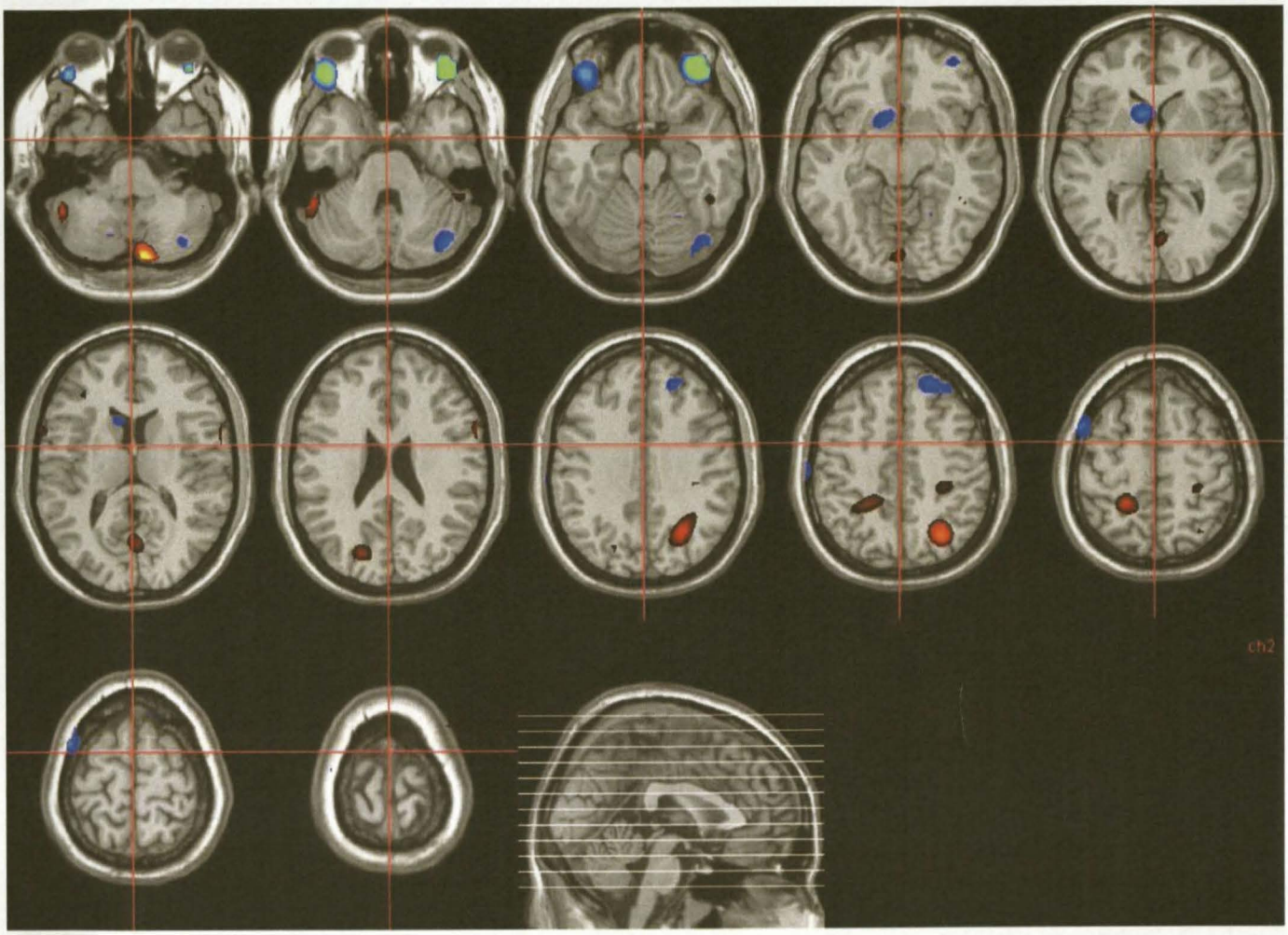

Figure 2. Selected Axial SPECT Slices co-registered to MRI showing Changes in rCBF in Patient 1, before and after Treatment.

Figure 2 shows increased perfusion in the parieto-occipital regions and the cerebellum, and decreased perfusion in the superior frontal cortex, cerebellum, and the caudate on the SPECT image of patient 1, after treatment. Areas of decreased perfusion is indicated by the colours green and blue on the image, while red represents increased perfusion.

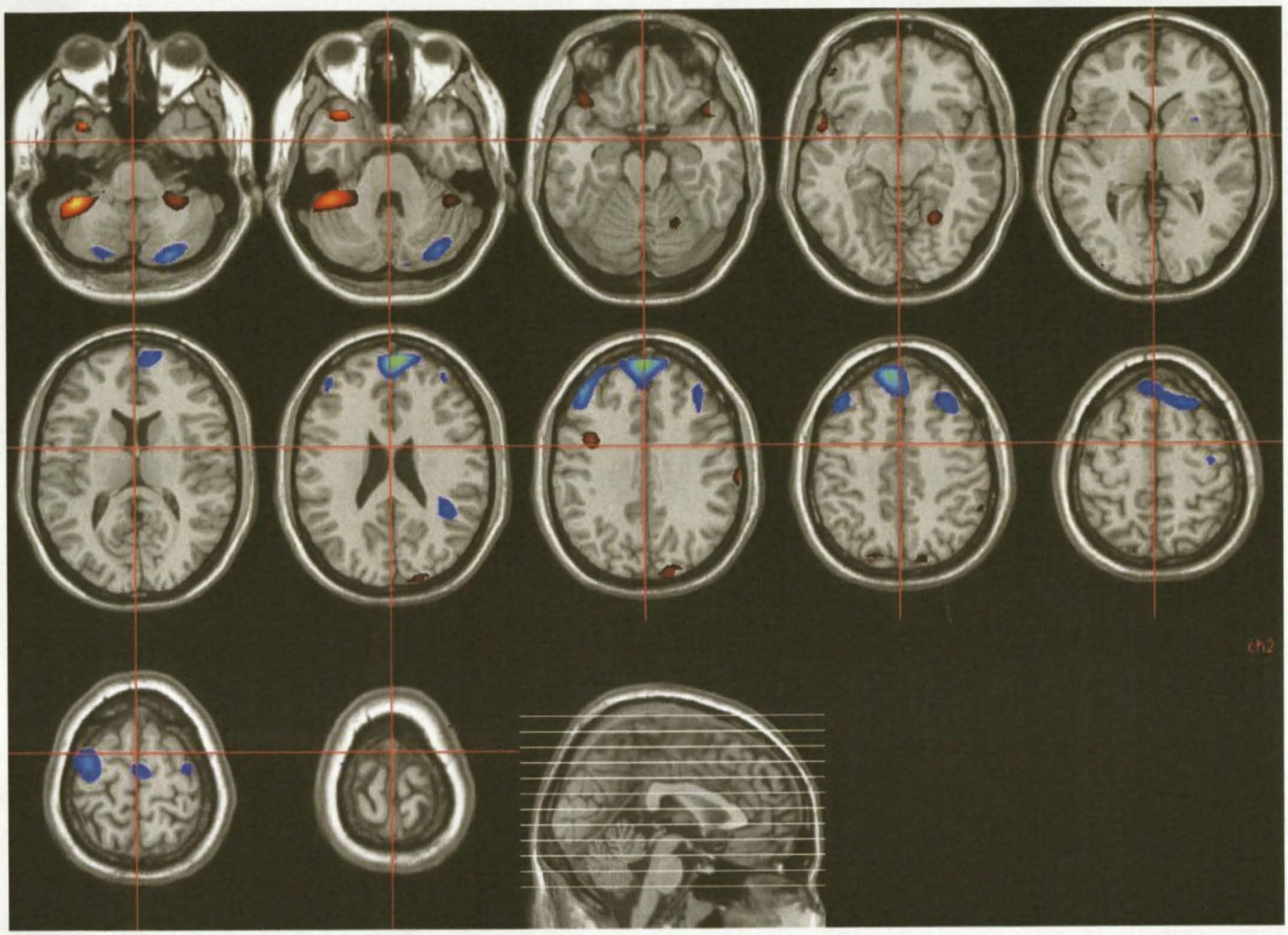

Figure 3. Selected Axial SPECT Slices co-registered to MRI showing Changes in rCBF in Patient 2, before and after Treatment.

From Figure 3 it is clear that patient 2's SPECT scan showed increased perfusion in the parieto-occipital regions, cerebellum, and the anterior temporal region. Decreased perfusion seems evident in the superior frontal cortex, mid frontal cortex, parieto-occipital region, and cerebellum.

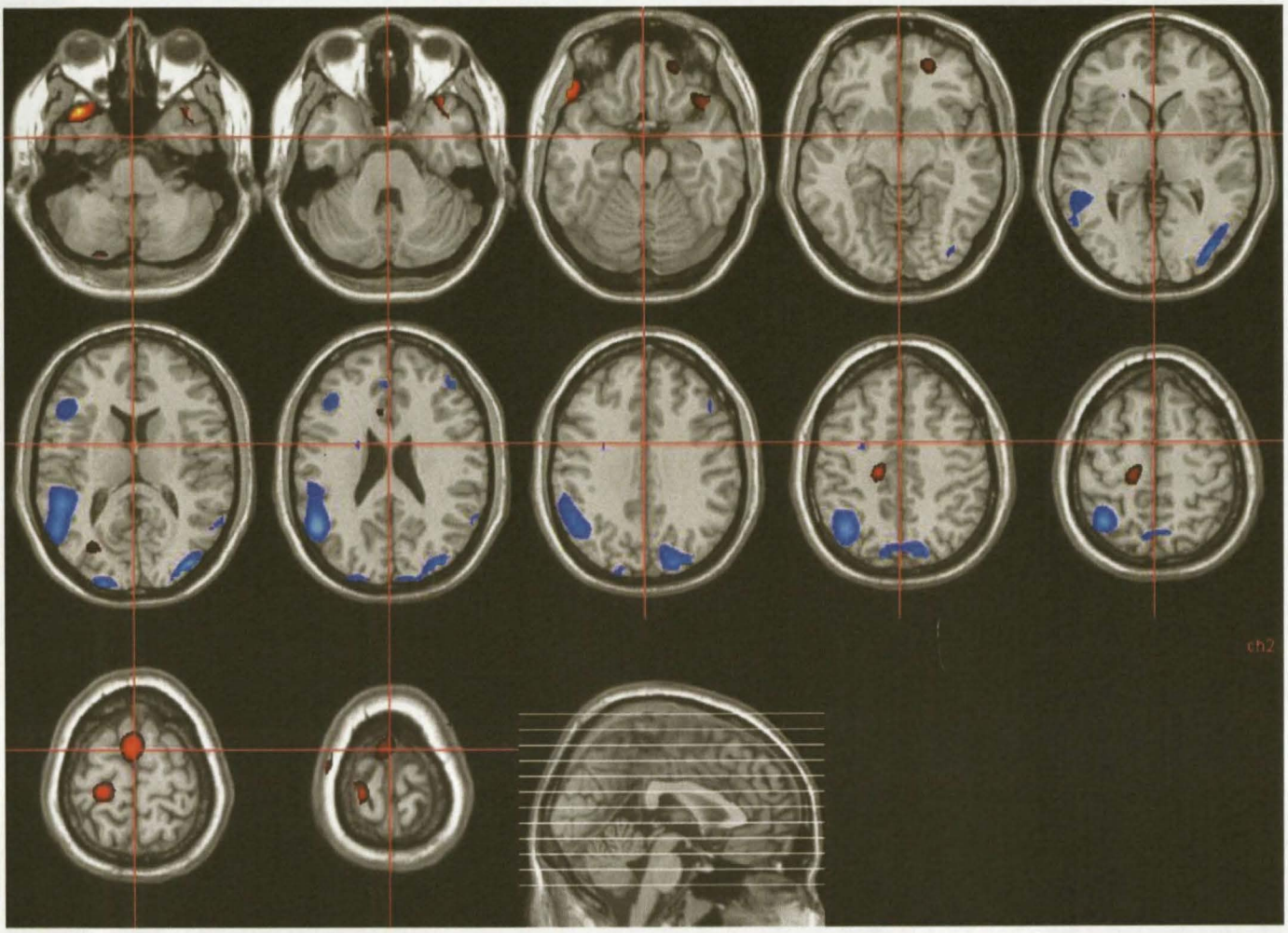

Figure 4. Selected Axial SPECT Slices co-registered to MRI showing Changes in rCBF in Patient 3, before and after Treatment.

Figure 4 indicates predominent decreased perfusion in the mid frontal cortex and the parieto-occipital regions on the SPECT scan of patient 3.

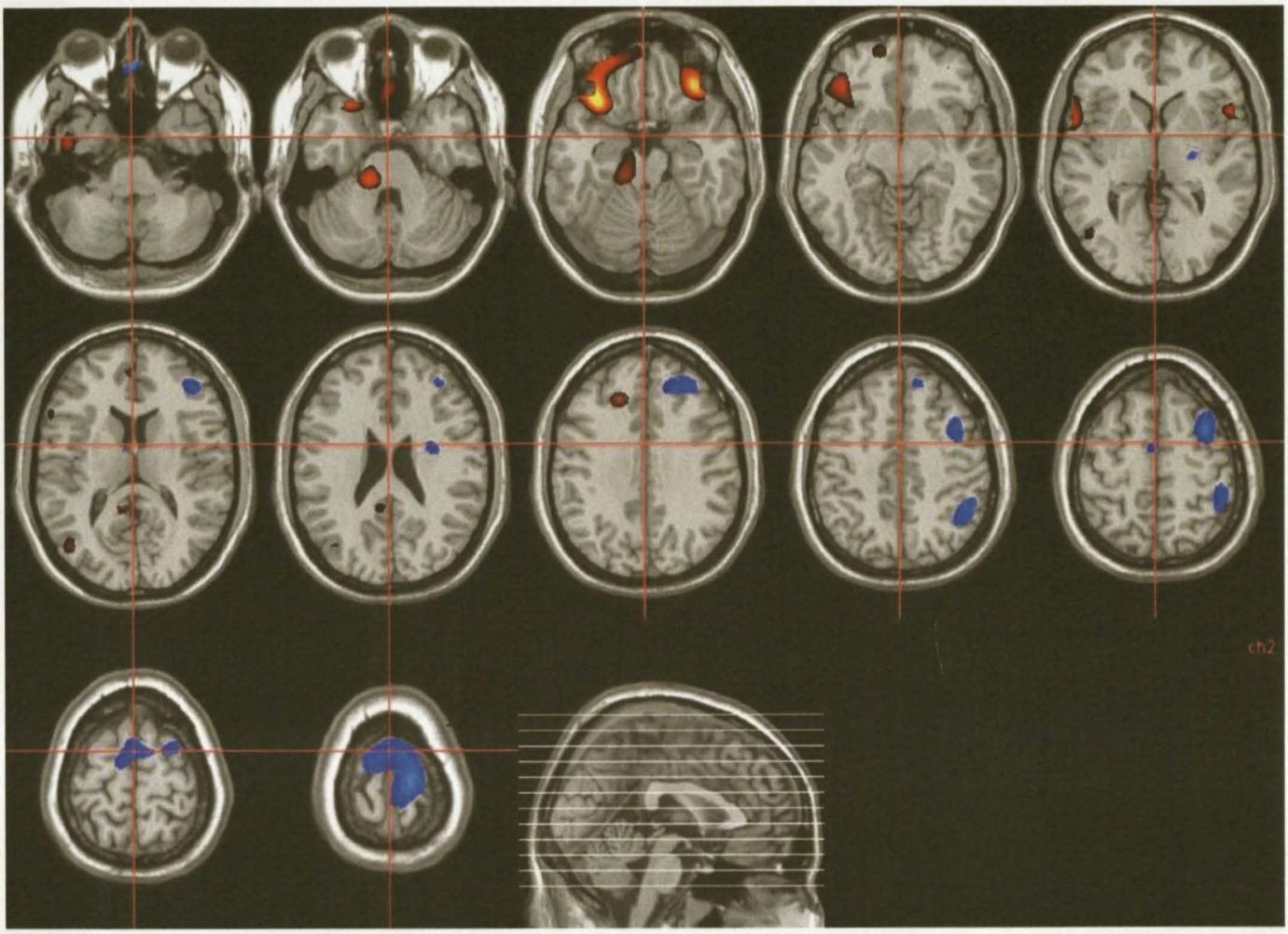

Figure 5. Selected Axial SPECT Slices co-registered to MRI showing Changes in rCBF in Patient 4, before and after Treatment.

From Figure 5 increased perfusion in the orbito-frontal region and decreased perfusion in the superior frontal cortex and the parieto-occipital area are evident on the SPECT scan of patient 4.

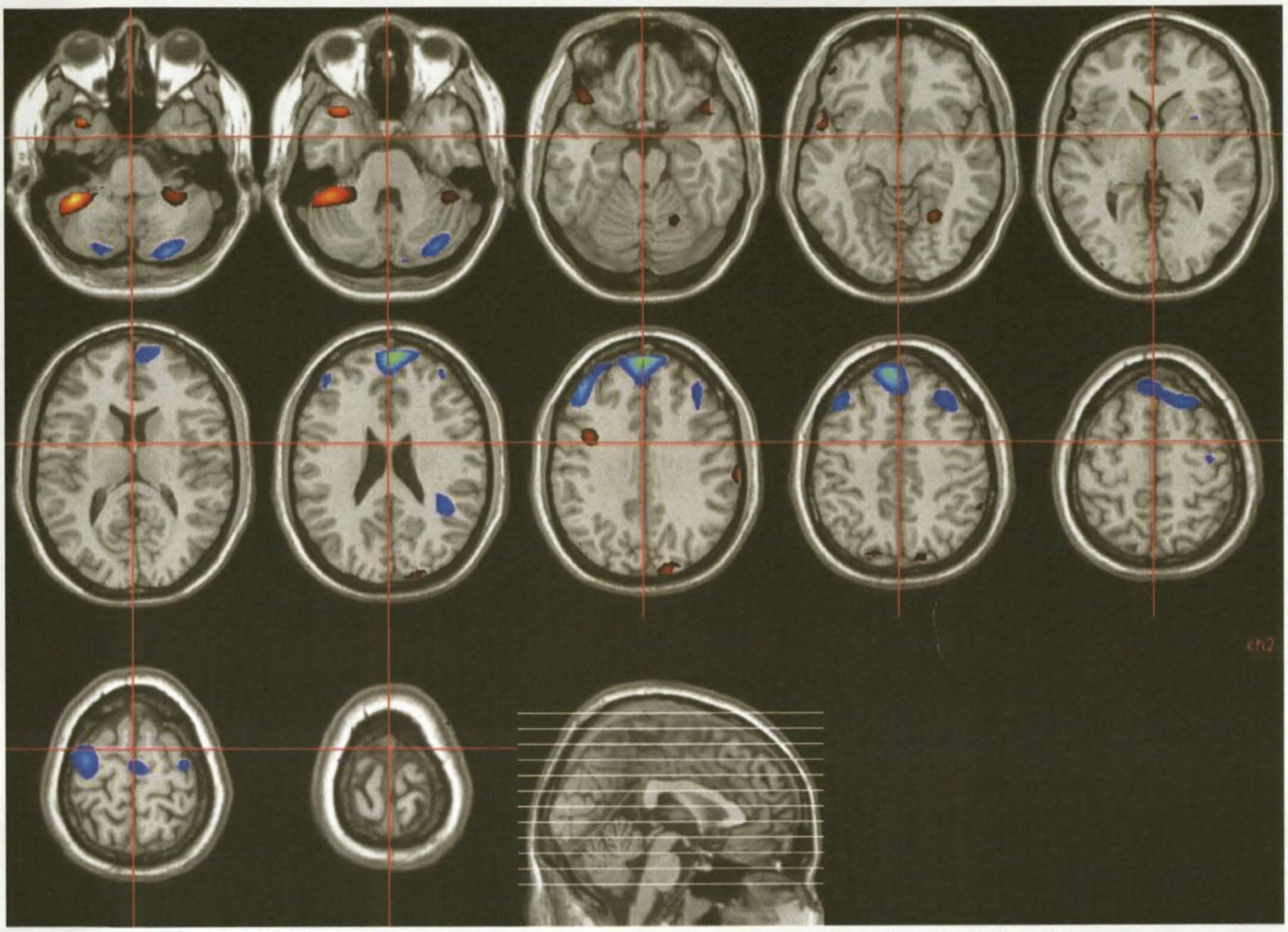

Figure 6. Selected Axial SPECT Slices co-registered to MRI showing Changes in rCBF in all 4 Patients.

From the subtraction SPECT images, it was found that changes in rCBF occurred in regions that have previously been implicated in the pathophysiology of PTSD, namely the superior frontal regions, the medial-temporal area, the cerebellum, neocortex, and the hipocampus. Figure 6 demonstrates reduced perfusion (blue-green) bilaterally in the superior and mid frontal regions. Mixed, bilateral changes in perfusion are also noticeable in the cerebellum.

Table 24

Summary of the Direction of Change in rCBF, following Prolonged Exposure Therapy

	Number of Patients out of 4	
Brain regions	Increased perfusion post-treatment	Decreased perfusion post-treatment
Superior frontal	0	3
Mid frontal	0	2
Orbito-frontal	1	0
Parieto-occipital	2	2
Cerebellum	2	2
Caudate	0	1
Anterior temporal	1	0

From Table 24 it is clear that the most consistent finding was that of reduced post-treatment perfusion in the superior frontal brain regions in three of the four patients. Mixed changes in both the parieto-occipital regions as well as the cerebellum were found, both within and between patients.

7 DISCUSSION

7.1 INTRODUCTION

The broad objective of the present study was to investigate the effectiveness of prolonged exposure treatment for PTSD in a South African sample of female survivors of sexual assault meeting the diagnostic criteria for PTSD. The effectiveness of exposure treatment was firstly examined in terms of its effect on the three symptom clusters of PTSD (re-experiencing, avoidance, and arousal) as assessed by an independent blind evaluator by means of the Clinician Administered PTSD Scale. Secondly, the effectiveness of exposure treatment was investigated in terms of possible changes in the associated symptoms of depression, anxiety, and dysfunctional cognitions. Finally, the effectiveness of exposure treatment was also investigated in terms of possible changes in brain metabolic activity, as reflected in altered regional cerebral blood flow (rCBF), measured by means of Single Photon Emission Computed Tomography.

These objectives were investigated in terms of the following hypotheses:

Hypothesis 1: Prolonged exposure treatment will result in a significant reduction of all three PTSD symptom clusters at post-treatment, and this improvement will be maintained at follow-up after three months.

Hypothesis 2: Prolonged exposure treatment will result in a significant reduction of the associated symptoms of depression, anxiety, and dysfunctional cognitions associated with PTSD.

Hypothesis 3: Prolonged exposure treatment will result in altering changed brain functioning in specific brain areas implicated in PTSD such as the superior frontal region, medial-temporal area, cerebellum, neocortex, and hipocampus.

The results of the study will now be discussed in terms of these hypotheses.

7.2 EFFECTS OF EXPOSURE TREATMENT ON PTSD SYMPTOMS

According to hypothesis one it is expected that prolonged exposure treatment will result in a significant reduction of all three PTSD symptom clusters at post-treatment and that this improvement will be maintained at follow-up after three months. This hypothesis was examined by means of the Clinician Administered PTSD Scale, administered by an independent blind evaluator at pre-treatment, post-treatment, and at follow-up.

Even though the treatment group scored significantly higher than the control group on total PTSD severity and on the arousal PTSD symptom cluster, the results show that the treatment group scored significantly lower than the untreated controls on total PTSD severity, as well as on all three PTSD symptom clusters (re-experiencing, avoidance, and arousal) at post-treatment. For the treatment group, there were significant decreases in PTSD severity for the re-experiencing and arousal clusters from pre-treatment to post-treatment, as well as further significant decreases from post-treatment to follow-up. For the avoidance symptom cluster a significant decrease was found from pre-treatment to post-treatment, but no further significant decrease from post-treatment to follow-up .

These results indicate that the treated group, in terms of an independent assessment by means of the Clinician Administered PTSD Scale, did not meet the diagnostic criteria for PTSD at post-treatment. These improvements were maintained at follow-up after three months, for total PTSD severity as well as for all three symptom clusters. As a matter of fact, treated individuals showed further improvement from post-treatment to follow-up on total PTSD severity as well as the re-experiencing and arousal symptom clusters, while not receiving active treatment anymore.

While none of the patients in the treatment group met the diagnostic criteria for PTSD at post-treatment and follow-up, all patients in the control condition maintained their PTSD diagnoses at the post-waiting period.

The results therefore offered support for hypothesis one that prolonged exposure treatment will result in a significant reduction of all three PTSD symptom clusters at post-treatment, and that this improvement will be maintained at follow-up after three months.

These results are in accordance with those of previous studies showing prolonged exposure to be an effective psychological treatment for PTSD (Boudewyns & Hyer, 1990; Boudewyns et al., 1990; Cooper & Clum, 1989; Foa et al., 1999; Foa et al., 1995; Foa et al., 1991; Keane et al., 1989; Marks et al., 1998; Richards et al., 1994; Taylor et al., 2003; Thompson et al., 1995). Of particilar importance are the studies by Foa et al. (1991) and Foa et al. (1995). Although these studies compared prolonged exposure, stress inoculation training, supportive counceling, and a waiting-list control condition (Foa et al.,1991) or prolonged exposure, stress inoculation training, a combination of these two treatments, and a waiting-list control condition (Foa et al., 1995), they incorporated the same manualized exposure treatment program, consisting of nine sessions of 90 minutes each, as in the present study. Participants were also female patients with PTSD following sexual violence, and assessments were similar to those of the present study. These two studies, similar in methodology to the present study, also showed prolonged exposure treatment to be effective in significantly reducing PTSD symptoms, and that the improvements following exposure treatment were maintained at follow-up.

7.3 EFFECTS OF EXPOSURE TREATMENT ON CO-MORBID SYMPTOMS ASSOCIATED WITH PTSD

According to hypothesis two, prolonged exposure treatment will result in a significant reduction of PTSD symptoms as well as the symptoms of dpression, anxiety, and dysfunctional cognitions associated with PTSD.

In order to investigate this hypothesis, participants in the treatment group completed the PTSD Symptom Scale (Foa et al., 1993), the Beck Depression Inventory II (Beck et al., 1988), the Beck Anxiety Inventory (Beck et al., 1988), and the Weekly Ideas Questionnaire (Foa et al., 1999) during sessions two, four, six, and eight. These symptoms were assessed at several sessions during treatment, not only to examine the effect of exposure treatment on these associated symptoms, but also to gain an impression of the process change during treatment.

Posttraumatic symptoms, measured by means of self-report PTSD Symptom Scale, decreased significantly from session two to session four, and from session four to session six, and again from session six to session eight. This implies that improvement in PTSD symptoms does not occur at a particular point during treatment, but that improvement represents a gradual process occuring over the full course of treatment.

Similar results were found for the symptoms of depression. The mean score of 18.53 on the Beck Depression Inventory at session two (which is just under the cut-off of 19, indicating moderate to severe depression) decreased significantly to 12.87 (light to moderate depression) at session four, and to 7.60 and 4.13 (none to minimum depression) at sessions six and eight respectively. Again, as was found for PTSD symptoms, this result indicates that improvement represents a gradual process occurring over the full course of treatment.

A similar gradual improvement was found for anxiety symptoms, measured by means of the Beck Anxiety Inventory. Anxiety symptoms decreased significantly from session two to session four, from session four to session six, and again from session six to session eight.

Scores on the Weekly Ideas Questionnaire showed a significant increase (indicating more functional thinking) from session two to session four, from session four to session six, and again from session six to session eight. This improvement in dysfunctional cognitions occurred despite the fact that dysfunctional cognitions were not directly targeted during the treatment program (e.g., by means of cognitive restructuring). It shows that exposure treatment indirectly affects dysfunctional PTSD cognitions.

The same pattern of improvement was thus manifested for self-reported PTSD symptoms, depression, anxiety, and dysfunctional thinking, confirming hypothesis two. Improvement did not occur at a specific point during treatment, but rather represents a gradual process spanning the course of treatment. This gradual improvement process was also demonstrated by Foa et al. (1991, 1997). In these studies the participants also showed significant gradual decreases in self-reported PTSD symptoms, depression, anxiety, and dysfunctional thinking. One possible explanation for this result may be found in Foa's Emotional Processing Theory of PTSD (Foa & Kozak, 1986). According to this theory, fear is represented in memory as a network of structures made up of associated stimulus,

response, and meaning elements, designed as a program to escape or avoid threatening danger (Rauch & Foa, 2006). This theory postulates that the fear structures of trauma survivors with PTSD include two basic dysfunctional sets of cognitions underlying the development and maintenance of PTSD, firstly, that the world is totally dangerous, and secondly, that the patient is totally helpless and incompetent. Successful reduction of PTSD can only be accomplished by integrating the information in the fear structures with the existing memory structures. Effective treatment therefore requires modification of the pathological elements of the fear structures, by engaging the patient with the daily activities, which he or she is continuously avoiding, as well as with the traumatic memories. This implies a systematic, repeated and gradual confrontation with the traumatic memories and with the avoided trauma-related situations. From this it can be deducted that improvement will be a gradual process, as demonstrated by the results in this section, rather than a "once-off" occurence at a particuar point during treatment.

7.4 BRAIN METABOLIC CHANGES AFTER EXPOSURE TREATMENT

According to hypothesis three, prolonged exposure treatment will result in altered brain functioning in specific brain areas implicated in PTSD such as the superior frontal region, medial-temporal area, cerebellum, neocortex, and hipocampus.

The main finding in this case series examining the effects of prolonged exposure on resting rCBF measured with SPECT, demonstrated a lowering of resting CBF in post-treatment scans in superior and mid frontal brain regions in three out of four patients. It is clearly demonstrated that measurable changes in resting brain perfusion following effective exposure treatment therapy for PTSD occurred, and that there is some overlap with the hypothesized effects of extinction within frontal brain regions.

The involvement of the prefrontal cortical regions is in line with mounting evidence of a role for this region in modulating excessive amygdala outflows reminiscent of an activated fear response characteristic of PTSD (Rauch, Shin, & Phelps, 2006). In a recent review by Roffman, Marci, Glick, Dougherty, and Rauch (2005) it was speculated that effective cognitive-behaviour therapy relies on an optimally functioning medial prefrontal cortex. Analogous animal and human studies suggest that the mPFC is crucial to retention and recall of acquired extinction (Lebron, Milad, & Quirk, 2004;

Gottfried & Dolan, 2004; Phelps, Delgado, Nearing, & Le Doux, 2004). In this model, however, it may be anticipated that mPFC function might need to be enhanced in order to regulate amygdala outflows.

The observed attenuation then of prefrontal rCBF post-PE in the current series may at first glance appear to be at odds with this hypothesis. Interestingly, however, the present finding is consistent with studies of cognitive-behavioural therapy (CBT) in depression (Goldapple et al., 2004) and cognitive-behavioural therapy in spider phobia (Paquette et al., 2003). In these studies, attenuation of frontal activation correlated with symptom improvements. It can be speculated that in the former, overall cognition in these regions may be improved and reflect as a normalizing of brain function and consequently a reduced demand on the prefrontal cortex. It may also be speculated that reduced symptoms translate to a lesser pre-occupation with anxiety-perpetuating ruminations and result in lower resting brain perfusion. The findings of the current study appear to be in line with these findings. Taken together, it is interesting to consider the overlap in effect of both cognitive-behavioural therapy and prolonged exposure in a range of disorders.

While the current series do not permit conclusions on the differential effect of psychotherapy and pharmacotherapy in PTSD, it does make for interesting comparisons in PTSD and other disorders. In PTSD, a single study examining rCBF in a SPECT study pre- and post-SSRI treatment Seedat et al. (2004) demonstrated an attenuation of medial temporal perfusion following treatment. Interestingly no effects on frontal brain regions, most notably the mPFC were seen. We may begin to speculate on the differential effect of pharmacotherapy and cognitive-behaviour therapy on rCBF in PTSD. This is supported by reports that treatment specific patterns of response exist in depression (Goldapple et al., 2004). Put differently, different treatments may target different components of the functionally connected circuit of brain regions. It has been proposed that antidepressant pharmacotherapy may target sub-cortical and limbic regions resulting in a "bottom-up" effect on frontal structures, while psychotherapy preferentially target frontal and prefrontal brain regions, exerting an enhanced (normalised) "top-down" control over sub-cortical and limbic regions (Milad, Rauch, Pitman, & Quirk, 2006; Roffman et al., 2005). While this theory is not without appeal, a study in social anxiety disorder does not seem to bear it out. Furmark et al. (2002) compared the effects of cognitive-behaviour therapy and citalopram on brain function in a positron emission tomography (PET) study and demonstrated that treatment response to citalopram and not cognitive-behaviour therapy correlated with reduced brain metabolic activity in the vPFC. Similarly, in a study of interpersonal therapy for depression, Brody et

al. (2001), in addition to attenuated prefrontal perfusion following interpersonal therapy, demonstrated higher perfusion in the same region in a comparative paroxetine group. From these mixed findings, it is impossible to draw firm general conclusions as to whether different disorders or different treatments contribute to the present mix of findings.

The current study does however prove the hypothesis that significant improvement in PTSD symptoms, brought about by prolonged exposure treatment, will be accompanied by significant change in cerebral blood flow in certain areas within the brain, indicating improved brain functioning. It is suggested that further research on this aspect is indicated.

One additional methodological consideration when comparing studies in this area, is the variable use of activation and rest to examine brain function. It may be that resting perfusion pattern differs considerably from emotionally activated states and that at rest rCBF is significantly different from healthy controls (Bonne et al., 2003; Sachinvala, Kling, Suffin, Lake, & Cohen, 2000). A number of treatment studies have employed resting scans before and after treatment, and have findings broadly in line with patho-physiology models, suggesting that resting rCBF measurements are not without merit and continue to deserve attention. Findings from a recent study in PTSD include higher limbic perfusion, lower frontal perfusion, and interestingly, higher cerebellar perfusion compared to healthy controls (Bonne et al., 2003). The findings in that study in the cerebellum are interesting in the light of findings in this region in the current study. Together these studies highlight the putative role played by the cerebellum in regulating aspects of the autonomic, motor and emotional behaviour in the phenomenology of PTSD. The bi-directional nature of the findings in the current study may be difficult to interpret in the light of the size of the present series, but deserves future attention.

The current series should serve to encourage the continued exploration of the link between prolonged exposure and extinction. Animal models of extinction have been extremely useful in elucidating the functional anatomy of the fear/stress response, and will continue to influence the exploration of the psychobiology of PTSD. Recent suggestions that possible deficits in extinction of fear conditioned responses, mediated through amygdala and medial prefrontal cortex (Quirk, Garcia, & Gonzalez-Lima, 2006), may constitute a risk factor for the development of PTSD (Charney, 2004; Milad et al., 2006)

have lent further support to this line of investigation. In so doing, the existing neuro-anatomical models of PTSD have also been strengthened (Pitman, Shin, & Rauch, 2001; Shin, Rauch, & Pitman, 2006). The process of extinction leads to an attenuation of fear responses through repeated exposure to a conditioned stimulus (memory of the trauma) which, in the absence of the unconditioned stimulus, eventually results in the "uncoupling" of the conditioned stimulus – unconditioned stimulus association (Hermans, Craske, Mineka, & Lovibond, 2006). This suggests that extinction is not so much an unlearning of a conditional response, but the relearning of a new response to the conditioned stimulus (Hermans et al., 2006). In fact extinction does not eliminate the association between the unconditioned stimulus and conditioned stimulus as there is now considerable evidence that the extinct conditioned response may in fact be recovered, and that this recovery is largely context dependent (Bouton, 2004; Vansteenwegen et al., 2006). This has implications for the value of a variety of psychotherapy techniques in that patients establish safe and trusted mechanisms through which they are able to confront rising fear in the face of environmental triggers. This may account in part for the long-term efficacy of therapy techniques and is in line with the maintained treatment response observed at three months follow-up in the current study.

The similarities with the processes involved in prolonged exposure are clear. As alluded to above, a number of lines of evidence now point to the central role played by the ventro-medial prefrontal cortex (vmPFC) in regulating exaggerated fear responses, mediated by limbic structures (Milad et al., 2006; Quirk et al., 2006). The need for a functioning mPFC in cognitive-behaviour therapy (Roffman et al., 2005) and the same regions' involvement in PTSD, would seem to lend further support to already mounting evidence for the notion of a failure of "top-down" fear attenuation and habituation mechanisms within the mPFC over the amygdala (Bremner, 2003; Shin et al., 2004). In the current study the observed attenuation of frontal perfusion seems likely to reflect a normalizing of brain function with enhanced prefrontal efficiency in regulating sub-cortical fear-driven outflows.

In conclusion, the effectiveness of prolonged exposure treatment in the current SPECT series of four case studies as well as in the complete study, is in line with available literature on cognitive-behaviour therapy for PTSD. The apparent overlap in the mechanisms of symptom reduction using prolonged exposure and the concept of fear extinction derived from animal models provides further interesting insights into the mechanisms through which prolonged exposure works. The findings of the current

study also add to questions on the differential mechanisms that may be involved in psychotherapy and pharmacotherapy in effecting a treatment response in PTSD.

Effective prolonged exposure therapy for PTSD is associated with attenuation of frontal rCBF measured with resting SPECT before and after treatment. These changes are in line with findings using a variety of psychotherapy techniques in a variety of mood and anxiety disorders. Further exploration of the overlap between PE and extinction, may in future provide us with clearer ideas on the mechanisms of response to treatment in PTSD, and will help focus pursuit of future treatment options.

7.5 GENERAL CONCLUSION

The results of the present study showed prolonged exposure as effective in significantly reducing all three symptom clusters of PTSD to the point where all treated patients no longer met the diagnostic criteria for PTSD. These improvements at the end of treatment were maintained for at least three months after treatment. Prolonged exposure treatment was also effective in significantly reducing the depression, anxiety, and dysfunctional thinking associated with PTSD. In addition, based on four case studies, there are indications that prolonged exposure treatment also facilitates a reduction in perfusion bilaterally in the superior and mid frontal regions, as well as mixed, bilateral changes in perfusion in the cerebellum and parieto-occipital regions.

Thus, positive results were obtained for all the dimensions of improvement (PTSD symptomatology, associated symptoms, and brain metabolic functioning) included in this study. As previous studies demonstrated the effectiveness of exposure treatment for PTSD with either American or British samples, this was the first study to investigate the effectiveness of exposure treatment in a South African sample of female survivors of sexual violence. This sample consisted of females of primarily a lower socio-economic status. Sixty percent of women in the treatment group received a household income of less than R5000 (approximately 700 US dollars) per month, while four to six people (children, siblings, nieces, parents, grandparents) were dependent on this income. Six of the 15 individuals in the treatment group were unemployed at the time of the study, while 10 (66.7%) did not complete high school. The results of the present study therefore showed the effectiveness of exposure

treatment for PTSD patients from a completely different cultural context, and offers support for the use of exposure as treatment for PTSD outside the USA and the UK.

7.6 LIMITATIONS OF THE PRESENT STUDY

One of the main strengths of the present study is that it largely adhered to the methodological criteria or so-called gold standards for therapy outcome studies (Foa & Meadows, 1997): target symptoms were clearly defined and symptom severity was specified, reliable and valid measures were used, an independent blind evaluator was included and properly trained, manualized treatment was provided in order to standardize treatment, patients were randomly assigned to the treatment and control conditions, treatment was provided by the same clinical psychologist, and adherence to the treatment manual was extensively monitored. The main methodological shortcoming was that the study used only one instead of two blind evaluators.

Another limitation of the study was the small sample size, 15 and 14 patients in the treatment and control groups respectively, while SPECT studies for only four patients were available at the end of treatment. This, to a large extent, reflects on the problems encountered in doing psychotherapy outcome research in a developing country, with low socio-economic status patients not familiar with psychotherapeutic treatment. Problems regarding referral emerged from the beginning of the study. In order to obtain potential participants who had not yet been treated for PTSD in the past, several referral sources were visited. Hospitals, including community medical care centres (day hospitals), were visited in an area with a radius of approximately fifty kilometers. When an appointment could be arranged with the responsible staff member, the aim of the research was explained. Due to shortages of staff, just arranging an appointment with the person concerned, often proved quite difficult. In general, the staff at these institutions received the researcher cordially, and were quite willing to assist in referring patients. However, the crucial shortages of staff proved a stumbling block. As the study was not directly connected with the urgent day-to-day workload of the hospital, the referral of patients to the study was often not seen as a priority. However, the bulk of referrals did eventually come from this source. Staff shortages, combined with a high crime rate, was the cause of a similar situation at the police stations contacted with a view to possible referrals. The police officers were cordial and quite enthusiastic to refer possible subjects, but probably due to the stress on short-staffed stations, and in spite of the high incidence of rape cases, no referrals were received from this potential source.

Advertisements in the daily press did not produce any reaction, nor did several interviews on local radio stations. Private practitioners as well as the combined Tygerberg/Stikland psychiatric hospital was eventually responsible for a large percentage of the referrals.

In addition, as the majority of patients came from lower-income areas, financial factors played an important role in attendance of treatment and drop-out. It often happened that patients did not turn up for scheduled treatment sessions without notifying the therapist in advance of any problems. Upon enquiry, the reason for not attending treatment was given as a shortage of bus fare. In some cases, when patients were given the money for traveling to the next session, it was used to buy food instead and the appointment was not kept. Eventually a number of patients had to be fetched for each session and then returned home again, at times over a distance of more than forty kilometers. Apart from the financial implications, this proved to be a time-consuming exercise. Other variables also impacted on adherence to treatment, for example, one of the patients in the current study, who lived in a low-income community approximately thirty kilometers from the therapist's rooms, was very co-operative and willing to participate in the study, and the initial, pre-treatment SPECT scan was done with no attendant problems or fears. Due to financial problems she was fetched for the first few therapy sessions and taken home again afterwards. Then the fact emerged that she was the sole provider for a household consisting of her mother, herself and several children. Up to that stage she had succeeded in juggling her casual job at a nearby grocery store in such a way that her therapy sessions could be fitted in. She was obviously responding well to the therapy. Her employer, however, was in no way sympathetic or interested in making concessions to help her. Consequently she was faced with his ultimatum to be available at all times, in case one of the regular employees did not turn up, or else he would get another casual employee to replace her. Being the sole provider for the extended family, she had no choice but to terminate her therapy sessions.

In contrast to the above, a lack of commitment by some patients was a definite problem. In a number of cases patients 'disappeared' before their treatment had been completed. As soon as they started 'feeling better' and the PTSD symptoms were not as intrusive any more, these patients stopped attending treatment. Tracking these patients often proved quite difficult as some of them did not really have a definite fixed address, but were living with relatives, and when the PTSD symptoms became less severe, they had moved to other relatives, some even to a different country, e.g. Namibia.

Fear of the unknown was a problem with several patients, particularly because they were not familiar with psychotherapeutic treatment. This was also particularly applicable to the prospect of the SPECT scans. In spite of detailed explanations and repeated reassurances about the procedure, the fear remained with some of the prospective participants, even after they had agreed to the procedure. This resulted in a few patients not turning up for the scheduled pre-treatment SPECT scans and not being prepared to continue with the treatment, even after being given the option of treatment only, without the scan component. Two actually had the pre-treatment scans done without objections, but were not prepared to have the post-treatment scans done.

These problems regarding the recruitment of patients and keeping them in treatment, highlight the difficulties of psychotherapy outcome research in a developing country with low socio-economic status patients not familiar with the requirements of psychotherapeutic treatment. Exposure treatment is normally associated with a high drop-out rate, simply because patients are required to confront their fears and avoidance behaviours. An interesting observation is that this was not the case in the present study. Of the 40 patients who met the inclusion criteria for the study, 11 dropped out (section 5.3.2). However, only two of these 11 patients dropped out due to the intensity of the exposure treatment. The rest (nine patients) dropped out due to transport problems (four patients), work commitments (three patients), or fear of the SPECT scans (two patients).

Two further problems also emerged regarding the SPECT scans. At a late stage in the study, the HMPAO radiopharmaceutical which was used in the SPECT procedure up till that point, was taken off the market. Initially it was not clear whether it was a permanent or a temporary arrangement. It did, however, mean that no scientifically comparable post-treatment SPECT scans could be performed for those patients who were still in the process of treatment. This was because scans performed using another agent would, from a scientific viewpoint, make the comparison between pre- and post-treatment scans difficult and potentially unreliable.

A second problem which hampered the SPECT scans evolved due to the necessary upgrading which had to be made to the electronic system of the Department of Nuclear Medicine at Tygerberg Hospital. During this upgrading, some of the data of this study had to be saved on optical discs. When the saved data, which included some of the pre-treatment SPECT scans of the current study, had to be recovered, one of the optical discs proved to be damaged and the data could not be retrieved. As the relevant pre-

treatment scans could not be re-done, they were lost and therefore the number of SPECT scans available for interpretation were further diminished.

7.7 RECOMMENDATIONS FOR FUTURE RESEARCH

Based on these considerations and the results of the present study, the following recommendations for future research are made:

7.7.1 Due to the difficulties in recruitment and in keeping patients in treatment, the number of participants was small. It is suggested that the study be expanded using a larger number of participants. Funds should be made available to reimburse participants for traveling expenses, and employers should be encouraged, with the consent of the patients, to allow patients to attend treatment.

7.7.2 A major problem for victims of trauma and belonging to low socio-economic status communities in South Africa is that of retraumatization. It is not uncommon for the trauma victim to stay in an abusive relationship for financial reasons, or to witness traumatization of a family member or friend. The present study did not monitor retraumatization in the study population. It is suggested that in a future study the possibility of retraumatization is monitored so as to determine its effect on the process of treatment outcome.

7.7.3 The current study excluded a large segment of the South African population due to the inability of the researcher to speak a traditional African language. There is a great need for research on the effective treatment of PTSD in non-Western communities, and it is suggested that further studies on the effectiveness of exposure treatment for PTSD include non-Western samples.

7.7.4 The SPECT-imaging in the present study was done in a resting state. Due to this, the investigation of the effect of the PTSD on rCBF is limited to when the participant is calm. As PTSD has a fluctuating coarse and exposure therapy focuses on the decrease of anxiety symptoms on imagining the trauma, a better understanding of the effect of treatment on brain metabolism would be gained by performing SPECT on imaginative activation of the trauma material.

7.7.5 The availability of exposure treatment for PTSD sufferers in South African communities seems to be a problem. Research is needed on the possibility and effect of training community clinic counselors in exposure therapy for PTSD, and on the supervision of such counselors, in order to bring the treatment closer to the people who need it.

References

Afana, A.H., Dalgard, O.S., Bjertness, E., Grunfeld, B., & Hauff, E. (2002). The prevalence and associated socio-demographic variables of posttraumatic stress disorder among patients attending primary care health care centres in the Gaza Strip. *Journal of Refugee Studies, 15*, 283 – 295.

Ahern, J., Galea, S., Resnick, H., Kilpatrick, D., Bucuvalas, M., Gold, J., et al. (2002). Television images and psychological symptoms after the September 11 terrorist attacks. *Psychiatry, 65,* 289 – 300.

American Psychiatric Association. (1980). *Diagnostic and statistical manual for mental disorders.* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual for mental disorders.* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual for mental disorders.* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual for mental disorders*. (4th ed. TR.). Washington, DC: Author.

Amick-McMullan, A., Kilpatrick, D., & Resnick, H. (1991). Homicide as a risk factor for PTSD among surviving family members. *Behaviour Modification, 15,* 545 – 559.

Amir, N., Stafford, J., Freshman, M.S., & Foa, E.B. (1998). Relationship between trauma narratives and trauma pathology. *Journal of Traumatic Stress, 11*, 385 – 392.

Andrews, G. (1991). The evaluation of psychotherapy. *Current Opinion Psychiatry, 4,* 379 – 383.

Arendt, M., & Elklit, A. (2001). Effectiveness of psychological debriefing. *Acta Psychiatrica Scandinavica, 104,* 423 – 437.

Argenti-Pillen, A. (2000). The discourse on trauma in non-western cultural contexts: Contributions of an ethnographic method. In A.Y. Shalev, R. Yehuda, & A.C. McFarlane, (Eds.), *International handbook of human response to trauma,* (pp.87 – 102). New York: Kluwer Academic/ Plenum Publishers.

Armstrong, K., O'Callahan, W., & Marmar, C.R. (1991). Debriefing Red Cross disaster personnel: The multiple stressor debriefing model. *Journal of Traumatic Stress, 4,* 581 – 593.

Arnow, B.A., Hart, S., Scott, C., Dea, R., Oçonnell, L., & Taylor, C.B. (1999). Childhood sexual abuse, psychological distress, and medical use among women. *Psychosomatic Medicine, 61,* 762 – 770.

Baddeley, A. (1997). *Human memory. Theory and practice. (Revised edition).* Hove, UK: Psychology Press.

Baddeley, A., Bueno, O., & Cahill L., (2000). The brain decade in debate, 1: Neurobiology of learning and memory. *Brazilian Journal of Medical and Biological Research, 33,* 993 – 1002.

Baker, C.K., Norris, F.H., Diaz, D.M.V., Perilla, J.L., Murphy, A.D., & Hill, E.G. (2005). Violence and PTSD in Mexico: Gender and regional differences. *Social Psychiatry and Psychiatrical Epidemiology, 40,* 519 – 528.

Baker, L.L., & Jessup, B.A. (1980). The psychophysiology of affective verbal and visual information processing in depression. *Cognitive Therapy and Research, 4,* 135 – 148.

Bale, L., Picetti, R., Contarino, A., Koob, G.F., Vale, W.W., & Lee, K-F. (2002). Mice deficient for both corticotropin-releasing factor receptor 1 (CRFR1) and CRFR2 have an impaired stress response and display sexually dichotomous anxiety-like behavior. *Journal of Neuroscience, 22,* 193 – 199.

Bart, P. (1975). *Unalienating abortion, demystifying depression, and restoring rape victims*. Paper presented at the 12th Annual Convention of the American Psychiatric Association, Anaheim, CA.

Basoglu, M., & Mineka, S. (1992). The role of uncontrollable and unpredictable stress in post-traumatic stress responses in torture survivors. In M. Basoglu (Ed.), *Torture and its consequences* (pp. 182 – 225). Cambridge: Cambridge University Press.

Beck, A.T. (1967). *Depression: Causes and treatment*. Philadelphia: University of Pennsylvania Press.

Beck, A.T., Emery, G., & Greenberg, R.L. (1985). *Anxiety disorders and phobias: A cognitive perspective.* New York: Basic Books.

Beck, A.T., Epstein, N., Brown, G., & Steer, R.A. (1988). An inventory for measuring clinical anxiety: psychometric properties. *Journal of Consulting and Clinical Psychology, 56,* 893 – 897.

Beck, A.T., Rush, A.J., Shaw, B.F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

Beck, A.T., Steer, R.A., & Garbin, M.G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8,* 77 – 100.

Beck, A.T., Ward, C.H., Mendelsohn, M., Mock, J., & Erbauch, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561 – 571.

Bennett, P., Owen, R.L., Koutsakis, S., & Bisson, J. (2002). Personality, social context and cognitive predictors of post-traumatic stress disorder in myocardial infarction patients. *Psychology and Health, 17*, 489 – 500.

Berger, D., Saito, S., Ono, Y., & Tezuka, I. (1994). Dissociation and child abuse histories in an eating disorder cohort in Japan. *Acta Psychiatrica Scandinavica, 90,* 274 – 280.

Berton, M.W., & Stabb, D. S. (1996). Exposure to violence and post-traumatic stress disorder in urban adolescents. *Adolescence, 31,* 490 – 499.

Bing, O., Moller, C., Engel., J.A., Soderpalm, B., & Heilig, M. (1993). Anxiolytic-like action of centrally administered galanin. *Neuroscience Letters, 164,* 17 – 20.

Bisson, J., Jenkins, P., Alexander, J., & Bannister, C. (1997). A randomized controlled trial of psychological debriefing for victims of acute burn trauma. *British Journal of Psychiatry, 171,* 78 – 81.

Bisson, J.I., McFarlane, A.C., & Rose, S. (2000). Psychological debriefing. In E.B. Foa, T.M. Keane, & M.J. Friedman (Eds.), *Effective treatments for PTSD* (pp.39 – 59). New York: Guilford Press.

Blake, D.D. (1994). Rationale and development of the clinician-administered PTSD scales. *PTSD Research Quarterly, 5*, 1 – 8.

Blake, D.D., Weathers, F.W., Nagy, L.M., Kaloupek, D.G., Gusman, F.D., Charney, D.S., et al. (1995). The development of a Clinician-administered PTSD Scale. *Journal of Traumatic Stress, 8,* 75 – 90.

Blake, D.D., Weathers, F.W., Nagy, L.M., Kaloupek, D.G., & Klauminzer, G. (1990). A clinician rating scale for assessing current and life-time PTSD: The CAPS-1. *Behavioral Therapy, 18*, 187 – 188.

Blanchard, E.B., & Hickling, E.J. (1997). *After the crash: Assessment and treatment of motor vehicle accident survivors.* Washington, DC: American Psychological Association.

Blanchard, E.B., & Hickling, E.J. (2004). *After the crash.* Washington, DC: American Psychological Association.

Blanchard, E.B., Hickling, E.J., Forneris, C.A., Taylor, A.E., Buckley, T.C., Loos, W.R. et al. (1997). Prediction of remission of acute posttraumatic stress disorder in motor vehicle accident victims. *Journal of Traumatic Stress, 10,* 215 – 234.

Blank, A., (1993). The longitudinal course of posttraumatic disorder. In J. Davidson & E.B.Foa (Eds.), *Post Traumatic Stress Disorder: DSM–IV and Beyond* (pp 3 – 22). Washington, DC: American Psychiatric Press.

Boehnlein, J.K. (1987). Clinical relevance of grief and mourning among Cambodian refugees. *Social Science and Medicine, 25,* 765 – 772.

Bonne, O., Gilboa, A., Louzoun, Y., Brandez, D., Yona, I., Lester, H., et al. (2003). Resting regional cerebral perfusion in recent posttraumatic stress disorder. *Biological Psychiatry, 54,* 1077 – 1086.

Bordow, S., & Porritt, D. (1979). An experimental evaluation of crisis intervention. *Social Science and Medicine, 13a,* 251 – 256.

Boscarino, J.A., Galea, S., Ahern, J., Resnick, H., & Vlahov, D. (2002). Utilization of mental health services following the September 11th terrorist attacks in Manhattan, New York City. *International Journal of Emergency Mental Health, 4,* 143 – 155.

Boscarino, J.A., Galea, S., Ahern, J., Resnick, H., & Vlahov, D. (2003). Psychiatric medicine use among Manhattan residents following the World Trade Center disaster. *Journal of Traumatic Stress, 16,* 301 – 306.

Boudewyns, P.A., & Hyer, L. (1990). Physiological response to combat memories and preliminary treatment outcome in Vietnam veterans: PTSD patients treated with direct therapeutic exposure. *Behavior Therapy, 21,* 63 – 87.

Boudewyns, P.A., & Hyer, L.A. (1996). Eye movement desensitization and reprocessing (EMDR) as treatment for posttraumatic stress disorder (PTSD). *Clinical Psychology and Psychotherapy, 3,* 185 – 195.

Boudewyns, P.A., Hyer, L., Woods, M.G., Harrison, W.R., & McCranie, E. (1990). PTSD among Vietnam veterans: An early look at treatment outcome using direct therapeutic exposure. *Journal of Therapeutic Stress, 3,* 359 – 368.

Boudewyns, P.A., Stwertka, S., Hyer, L., Albrecht, W., & Sperr, E. (1993). Eye movement desensitization for PTSD of combat: A treatment outcome pilot study. *Behavior Therapy, 16,* 29 – 33.

Bouton, M.E. (2004). Context and behavioral processes in extinction. *Learning and Memory, 11,* 485 – 494.

Bouwer, C., & Stein, D.J. (1998). Survivors of torture presenting at an anxiety disorders clinic: Symptomatology and pharmacotherapy. *Journal of Nervous and Mental Disease, 186,* 316 – 318.

Bower, G.B. (1981). Mood and memory. *American Psychologist, 36*, 129 – 148.

Bracken, P.J., Giller, J.E., & Summerfield, D. (1995). Psychological responses to war and atrocity: The limitations of current concepts. *Social Science and Medicine, 40,* 1073 – 1082.

Brady, K.T., Killeen, T.K., & Brewerton, T. (2000). Comorbidity of psychiatric disorders and posttraumatic stress disorder. *Journal of Clinical Psychiatry, 61*, 22 – 32.

Brady, K.T., Killeen, T., Saladin, M.E., Dansky, B., & Becker, S. (1994). Comorbid substance abuse and posttraumatic stress disorder. *American Journal of Addictions, 3,* 160 – 164.

Bramson, I., Dirkzwager, A.J.E., & Van Der Ploeg, H.M. (2000). Predeployment personality traits and exposure to trauma as predictors of posttraumatic stress symptoms: A prospective study of former peacekeepers. *American Journal of Psychiatry, 157,* 1115 – 1119.

Bremner, J.D. (1999). Traumatic memories lost and found: Can lost memories of abuse be found in the brain? In L. Williams & V. Banyard (Eds.), *Trauma and memory* (pp. 217 – 227). Thousand Oaks, CA: Sage.

Bremner, J.D. (2003). Functional neuroanatomical correlates of traumatic stress revisited 7 years later, this time with data. *Psychopharmacology Bulletin, 37,* 6 – 25.

Bremner, J.D., Narayan, M., Staib, L.H., Southwick, S.M., McGlashan, T., & Charney, D.S. (1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. *American Journal of Psychiatry, 156,* 1787 – 1795.

Bremner, J.D., Randall, P., Scott, T.M., Bronen, R.A., Seibyl, J.P., Southwick, S.M., et al., (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry, 152,* 973 – 981.

Bremner, J.D., Randall, P., Vermetten, E., Staib, E., Bronen, R.A., Mazure, C., et al., (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse: A preliminary report. *Biological Psychiatry, 41,* 23 – 32.

Bremner, J., Scott, T.M., Delaney, R.C., Southwick, S.M., Mason, J.W., Johnson D.R., et al., (1993). Deficits in short-term memory in posttraumatic stress disorder. *American Journal of Psychiatry, 150,* 1015 – 1019.

Bremner, J.D., Southwick, S.M., Brett, E., Fontana, A., Rosenheck, R., & Charney, D.S. (1992). Dissociation and posttraumatic stress disorder in Vietnam combat veterans. *American Journal of Psychiatry, 149,* 328 – 332.

Bremner, J.D., Southwick, S.M., & Charney, D.S. (1999). The neurobiology of posttraumatic stress disorder: An integration of animal and human research. In P.A. Saigh & J.D. Bremner (Eds.), *Posttraumatic stress disorder: A comprehensive text.* Boston, MA: Allyn & Bacon.

Breslau, N. (2001a). The epidemiology of posttraumatic stress disorder: What is the extent of the problem? *Journal of Clinical Psychiatry, 62,* 16 – 22.

Breslau, N. (2001b). Outcomes of posttraumatic stress disorder. *Journal of Clinical Psychiatry, 62,* 55 – 59.

Breslau, N., Chilcoat, H.D., Kessler, R.C., & Davis, G.C. (1999). Previous exposure to trauma and PTSD effects of subsequent trauma: Results from the Detroit Area Survey of Trauma. *American Journal of Psychiatry, 156,* 902 – 907.

Breslau, N., Chilcoat, H.D., Kessler, R.C., Peterson, E.L., & Lucia, V.C. (1999). Vulnerability to assault violence: Further specification of the sex difference in post-traumatic stress disorder. *Psychological Medicine, 29,* 813 – 821.

Breslau, N., & Davis, G.C., (1992). Posttraumatic stress disorder in an urban population of young adults: Risk factors for chronicity. *American Journal of Psychiatry, 149,* 671 – 675.

Breslau, N., Davis, G.C., Andreski, P., & Peterson, E. (1991). Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Archives of General Psychiatry, 48,* 216 – 222.

Breslau, N., Davis, G.C., & Schultz, L.R., (2003). Posttraumatic stress disorder and the incidence of nicotine, alcohol, and other drug disorders in persons who have experienced trauma. *Archives of General Psychiatry, 60,* 289 – 294.

Breslau, N., Kessler, R.C., Chilcoat, H.D., Schultz, L.R., Davis, G.C., & Andreski, P. (1998). Trauma and posttraumatic stress disorder in the community: The 1996 Detroit Area Survey of Trauma. *American Journal of Psychiatry, 55,* 626 – 632.

Breslau, N., Lucia, V.C., & Davis, G.C. (2004). Partial PTSD versus full PTSD: An empirical examination of associated impairment. *Psychological Medicine, 34,* 1205 – 1214.

Brewin, C.R. (1989). Cognitive change processes in psychotherapy. *Psychological Review, 96,* 379 – 394.

Brewin, C.R., Andrews, B., & Valentine, J.D., (2000). Meta-analysis of risk factors for post traumatic stress disorder in trauma-exposed adults. *Journal of Consulting Clinical Psychology 68,* 748 – 766.

Brewin, C.R., Dalgleish, T., & Joseph, S. (1996). A dual representation theory of posttraumatic stress disorder. *Psychological Review, 103,* 670 – 686.

Brewin, C.R., & Holmes, E.A. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review, 23,* 339 – 376.

Brody, A.L., Saxena, S., Stoessel, P., Gillies, L.A., Fairbanks, L.A., Alborzian, S., et al. (2001). Regional brain metabolic changes in patients with major depression treated with either paroxetine or interpersonal therapy: Preliminary findings. *Archives of General Psychiatry, 58,* 631 – 640.

Brom, D., Kleber, R.J., & Defares, P.B. (1989). Brief psychotherapy for posttraumatic stress disorders. *Journal of Consulting Clinical Psychology, 57,* 607 – 612.

Brown, T.A., & Barlow, D.H. (1992). Comorbidity among anxiety disorders: Implications for treatment and DSM-IV. *Journal of Consulting and Clinical Psychology, 60,* 835 – 844.

Brown, P.J., Rucupero, P.R., & Stout, R. (1995). PTSD substance abuse comorbidity and treatment utilization. *Addictive Behaviors, 20,* 251 – 254.

Bryant, R.A., & Harvey, A.G. (2000). *Acute stress disorder: A handbook of theory, assessment, and treatment.* Washington, DC: American Psychological Association.

Bulhan, H.A. (1985). *Franz Fanon and the psychology of oppression.* New York: Plenum Press.

Bunn, T., & Clarke, A. (1979). Crisis intervention: An experimental study of the effects of a brief period of counselling on the anxiety of relatives of seriously injured or ill hospital patients. *British Journal of Medical Psychology, 52,* 191 – 195.

Burgess, A.W., & Holmstrom, L.L. (1974a). The rape trauma syndrome. *American Journal of Psychology, 131,* 981 – 986.

Burgess, A.W., & Holmstrom, L.L. (1974b). *Rape: Victims of crisis.* Bowie, MD: R.J.Brady.

Burns, D. (1980). *Feeling good: The new mood therapy.* New York: William Morrow.

Byrne, C.A., & Riggs, D.S. (1996). The cycle of trauma: Relationship aggression in male Vietnam veterans with symptoms of posttraumatic stress disorder. *Violence and Victims, 20,* 549 – 559.

Cahill, L. (1997). The neurobiology of emotionally influenced memory. Implications for understanding traumatic memory. In R. Yehuda & A.C. McFarlane (Eds.), *Psychobiology of posttraumatic stress disorder.* New York: New York Academy of Sciences.

Cahill, L., & McGaugh, J.L. (1995). A novel demonstration of enhanced memory associated with emotional arousal. *Consciousness and Cognition: An Internationa Journal, 4,* 410 – 421.

Cantlie, A. (1994). Psychoanalysis and anthropology: Applied or misapplied? *The Psycoanalysis Newsletter, 14,* 2 – 7.

Carey, P.D., Stein, D.J., Zungu-Dirwayi, N., & Seedat, S. (2003). Trauma and posttraumatic stress disorder in an urban Xhosa primary care population: Prevalence, comorbidity, and service use patterns. *Journal of Nervous & Mental Disease, 191,* 230 – 236.

Carlson, J.G., Chemtob, C.M., Rusnak, K., Hedlund, N.L., & Muraoka, M.Y. (1998). Eye movement desensitization and reprocessing (EMDR) treatment for combat-related post-traumatic stress disorder. *Journal of Traumatic Stress, 11,* 3 – 24.

Carlson, J.G., & Rosser-Hogan, R. (1991). Trauma experiences, post-traumatic stress, dissociation, and depression in Cambodian refugees. *American Journal of Psychiatry, 148,* 1548 – 1551.

Carlson, J.G., & Rosser-Hogan, R. (1993). Mental health status of Cambodian refugees ten years after leaving their homes. *American Journal of Orthopsychiatry, 63,* 223 – 231.

Caspi, A., Sugden, K., Moffitt, T.E., Taylor, A., Craig, I.W., Harrington, H., et al., (2003). Influence of life stress on depression: Moderation by a polymorhism in the 5-HTT gene. *Science, 301,* 386 – 389.

Chang, L.T. (1978). A method for attenuation correction in radionuclide computed tomography. *IEEE Trans Nuclear Science, 25,* 638 – 643.

Charney, D.S. (2004). Psychobiological mechanisms of resilience and vulnerability: Implications for successful adaptation to extreme stress. *American Journal of Psychiatry, 161,* 195 – 216.

Charney, D.S., Deutch, A.Y., Krystal, J.H., Southwick, S.M., & Davis, M.. (1993). Psychobiological mechanisms of posttraumatic stress disorder. *Archives of General Psychiatry, 50,* 294– 305.

Chemtob, C.M. (1996). Posttraumatic stress disorder, trauma, and culture. In F.L. Mak and C.C. Nadelson (Eds.), *International Review of Psychiatry. 2,* 257 – 292. Washington DC: American Psychiatric Press.

Christianson, S.A. (1992). Emotional stress and eye-witness memory: A critical review. *Psychological Bulletin, 112,* 284 – 309.

Clark, D.C., Cavanaugh, S.V., & Gibbons, R.D. (1983). The core symptoms of depression in medical and psychiatric patients. *Journal of Nervous and Mental Diseases, 171,* 705 – 713.

Clark, L.A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology, 103,* 103 – 116.

Coffey, S.F., Gudmundsdottir, B., Beck, G., Palyo, S.A., & Miller, L. (2006). Screening for PTSD in motor vehicle accident survivors using the PSS-SR and IES. *Journal of Traumatic Stress, 19,* 119 – 128.

Comijs, H.C., Penninx, B.W.J.H., Knipscheer, K.P.M., & Van Tilburg, W. (1999). Psychological distress in victims of elder mistreatment: The effects of social support and coping. *Journal of Gerontology: Psychological Sciences, 54B,* 240 – 245.

Conlon, L., Fahy, T.J., & Conroy, R. (1999). PTSD in ambulant RTA victims: A randomized controlled trial of debriefing. *Journal of Psychosomatic Research, 46,* 37 – 44.

Conway, M.A., & Pleydell-Pearce, C.W. (2000). The construction of autobiographical memories in the self-memory system. *Psychological Review, 27,* 261 – 286.

Cook, J.M. (2001). Posttraumatic stress disorder in older adults. *PTSD Research Quarterly, 12,* 1 – 7.

Cook, J.M., Riggs, D.S., Thompson, R., Coyne, J.C., & Sheikh, J.I. (2004). Posttraumatic stress disorder and current relationship functioning among World War II ex-prisoners of war. *Journal of Family Psychology, 18,* 36 – 45.

Cooper, N.A., & Clum, G.A. (1989). Imaginal flooding as a supplementary treatment for PTSD in combat veterans: A controlled study. *Behavior Therapy, 3,* 381 – 391.

Costa e Silva, J.A. (1998). The public health impact of anxiety disorders: A WHO perspective. *Acta Psychiatrica Scandinavia, 98,* 2 – 5.

Cox, B.J., MacPherson, P.S.R., Enns, M.W., & McWilliams, L.A. (2004). Neuroticism and self-critisism associated with posttraumatic stress disorder in a nationally representative sample. *Behaviour Research and Therapy, 42,* 105 – 114.

Cryer, L., & Beutler, L. (1980). Group therapy: An alternative treatment approach for rape victims. *Journal of Sex and Marital Therapy, 6,* 40 – 46.

Davidson, J.R.T., Hughes, D., Blazer, D.G., & George, L.K. (1991). Posttraumatic stress disorder in the community: An epidemiological study. *Psychological Medicine, 21,* 713 – 721.

Davidson, J.R.T., Rampes, H., Eisen, M., Fisher, P., Smith, R.D., & Malik, M.L. (1998). Psychiatric disorders in primary care patients receiving complementary medical treatments. *Comprehensive Psychiatry, 39,* 16 – 20.

Davidson, J., Swartz, M., Storck, M., Krishnan, R.R., & Hammett, E. (1985). A diagnostic and family study of posttraumatic stress disorder. *American Journal of Psychiatry, 142,* 90 – 93.

Davis, L. (1990). *The courage to heal workbook: For women and men survivors of child sexual abuse.* New York: Harper & Row.

Dawes, A. (1990). The effects of political violence on children: A consideration of South African and related studies. *International Journal of Psychology, 25,* 13 – 31.

De Bellis, M.D. (2001). Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy. *Development and Psychopathology, 13,* 539 – 564.

Deering, C.G., Glover, S.G., Ready, D., Eddleman, H.C., & Alarcon, R.D. (1996). Unique patterns of comorbidity in posttraumatic stress disorder from different sources of trauma. *Comprehensive Psychiatry, 37,* 336 – 346.

De Jong, J.T.V.M., Komproe, I.H., Van Ommeren, M., El Masri, M., Mesfin, A., Noureddine, K.,et al. (2001). Lifetime events and posttraumatic stress disorder in 4 postcolonial settings. *Journal of the American Medical Association, 286,* 555 – 562.

Della Femina, D., Yeager, C.A., & Lewis, D.O. (1990). Child abuse: Adolescent records vs. adult recall. *Child Abuse and Neglect, 14,* 227 – 231.

DeLeon, G., Skodol, A., & Rosenthal, M.S. (1978). Phoenix House: Changes in psychopathological signs of resident drug addicts. *Archives of General Psychiatry, 28,* 131 – 135.

Delisi, L.E., Maurizio, A., Yost, M., Papparozzi, C.F., Fulchino, C., Katz, C.L., et al. (2003). A survey of New Yorkers after the September 11, 2001, terrorist attacks. *American Journal of Psychiatry, 160,* 780 – 783.

DePrince, A.P., Chu, A., & Visvanathan, P. (2006). Dissociation and posttraumatic stress disorder (PTSD). *PTSD Research Quarterly, 17,* 1 – 7.

Derogatis, L.R. (1983). *SCL-90: Administration, scoring and procedures manual for the revised version.* Baltimore: Clinical Psychometry Research.

Devilly, G.J., Spence, S.H., & Rapee, R.M. (1998). Statistical and reliable change with eye movement desensitization and reprocessing: Treating trauma within a veteran population. *Behavior Therapy, 29,* 435 – 455.

Dienstbier, R.A. (1989). Arousal and physiological toughness: Implications for mental and physical health. *Psychological Review, 96,* 84 – 100.

Dodge, C.S., Hope, D.A., Heimberg, R.G., & Becker, R.E. (1988). Evaluation of the Social Interaction Self-Statement Test with a social phobia population. *Cognitive Therapy and Research, 12,* 211 – 222.

Draguns, J.G. (1994). Pathological and clinical aspects. In L. Adler & P.G. Uew. (Eds.), *Cross-cultural topics in psychology.* Pp. 165 – 177. Westport, CT: Praeger/Greenwood.

Du Plessis, E.D. (1998). *The assumptive world theory and the cognitive processing of trauma.* Unpublished master's thesis, University of Stellenbosch, Stellenbosch, South Africa.

Dunmore, E., Clark, D.M., & Ehlers, A. (2001). A prospective examination of the role of cognitive factors in persistent posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy, 39,* 1063 – 1084.

Dyregrov, A. (1989). Caring for helpers in disaster situations: Psychological debriefing. *Disaster Management, 2,* 25 – 30.

Eagle, G. (2002). The political conundrums of post-traumatic stress disorder. In D. Hooks & G. Eagle (Eds.), *Psychopathology and social prejudice* (pp. 75 – 91). Cape Town: University of Cape Town Press.

Echeburua, E., de Corral, P., Sarasua, B., & Zubizarreta, I. (1996). Treatment of acute posttraumatic stress disorder in rape victims: An experimental study. *Journal of Anxiety Disorders, 10,* 185 – 199.

Echeburua, E., de Corral, P., Zubizarreta, I., & Sarasua, B. (1997). Psychological treatment of chronic posttraumatic stress disorder in victims of sexual aggression. *Behavior Modification, 21,* 433 – 456.

Ehlers, A., & Clark, D.M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38,* 319 – 345.

Ehlers, A., Hackmann, A., & Michael, T. (2004). Intrusive re-experiencing in posttraumatic stress disorder: Phenomenology, theory and therapy. *Memory, 12,* 403 – 415.

Ehlers, A., Hackmann, A., Steil, R., Clohessy, S., Wenninger, K., & Winter, H. (2002). The nature of intrusive memories after trauma: The warning signal hypothesis. *Behaviour Research and Therapy, 40,* 1021 – 1028.

Ehlers, A., Mayou, R.A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of Abnormal Psychology, 107,* 508 – 519.

Ehlers, A., & Steil, R. (1995). Maintenance of intrusive memories in posttraumatic stress disorder: A cognitive approach. *Behavioural and Cognitive Psychotherapy, 23,* 217 – 249.

Eisenbruch, M. (1991). From posttraumatic stress disorder to cultural bereavement: Diagnosis of Southeast Asian refugees. *Social Science and Medicine, 33,* 673 – 680.

Ellis, A. (1962). *Reason and emotion in psychotherapy.* New York: Lyle Stuart.

Emsley, R., & Coetzer, P. (1996). Disability claims on psychiatric grounds. *South African Medical Journal, 86,* 646.

Emsley, R.A., Seedat, S., & Stein, D.J. (2003). Posttraumatic stress disorder and occupational disability in South African Security Force members. *Journal of Nervous and Mental Disease, 191,* 237 – 241.

Engdahl, B.E., Harkness, A.R., Eberly, R.E., Page, W.F., & Bielinski, J. (1993). Structural models of captivity trauma, resilience, and trauma response among former prisoners of war 20 to 40 years after release. *Social Psychiatry and Psychiatric Epidemiology, 28,* 109 – 115.

Evans, H.I. (1978). Psychotherapy for the rape victim: Some treatment models. *Hospital and Community Psychiatry, 29,* 309 – 312.

Fairbank, J.A., & Keane, T.M. (1982). Flooding for combat-related stress disorders: Assessment of anxiety reduction across traumatic memories. *Behavior Therapy, 13,* 499 – 510.

Feeny, N.C., Zoellner, L.A., Fitzgibbons, L.A., & Foa, E.B. (2000). Exploring the roles of emotional numbing, depression, and dissociation in PTSD. *Journal of Traumatic Stress, 13,* 489 – 498.

Field, A. (2005). *Discovering statistics using SPSS (2nd ed.).* London: Sage Publications Ltd.

First, M.B., Spitzer, R.L., Gibbon, M., & Williams, J.B.W. (1998). *Structured Clinical Interview for DSM-IV Axis I Disorders – Clinician Edition.* Biometrics Research Department, New York State Research Institute.

Foa, E.B., (1995). *Failure of emotional processing: Post trauma psychopathology. Paper p*resented at the World Congress of Behavioural and Cognitive Therapies; Copenhagen, Denmark.

Foa, E.B. (2006). Psychosocial therapy for posttraumatic stress disorder. *Journal of Clinical Psychiatry, 67,* 40 – 45.

Foa, E.B., & Cahill, S.P. (2001). Emotional processing in psychological therapies. In N.J. Smelser & P.B. Bates (Eds.), *International encyclopedia of the social and behavioral science* (pp 12363 – 12369). New York: Elsevier.

Foa, E.B., Clark, D.M., Ehlers, A., Orsillo, S.M., & Tolin, D.F. (1999). The posttraumatic cognitions inventory (PTCI): development and validation. *Psychological Assessment, 11,* 303 – 314.

Foa, E.B., Dancu, C.V., Hembree, E.A., Jaycox, L.H., Meadows, E.A., & Street, G.P. (1999). A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. *Journal of Consulting and Clinical Psychology, 67,* 194 – 200.

Foa, E.B., Hearst-Ikeda, D.E., Dancu, C.V., Hembree, E.A., & Jaycox, L.H. (1997). *Prolonged exposure manual:* Allegheny University of the Health Sciences (East Falls Campus): Eastern Pennsylvania Psychiatric Institute.

Foa, E.B., Hearst-Ikeda, D.E., & Perry, K.J. (1995). Evaluation of a brief cognitive-behavioral program for the prevention of chronic PTSD in recent assault victims. *Journal of Consulting Clinical Psychology, 63,* 948 – 955.

Foa, E.B., Hembree, E.A., Cahill, S.P., Rauch, S.A., Riggs, D.S., Feeny, N.C., et al. (2005). Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: Outcome at academic and community clinics. *Journal of Consulting and Clinical Psychology, 73,* 953 – 964.

Foa, E.B., Keane,T.M., & Friedman, M.J. (2000). *Effective treatments for PTSD.* New York: Guilford Press.

Foa, E.B., & Kozak, M.J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99,* 20 – 35.

Foa, E.B., & Meadows, E.A. (1997). Psychosocial treatments for post-traumatic stress disorder: A critical review. *Annual Review of Psychology, 48,* 449 – 480.

Foa, E.B., Molnar, C., & Cashman, L. (1995). Change in rape narratives during exposure therapy for posttraumatic stress disorder. *Journal of Traumatic Stress, 8,* 675 – 690.

Foa, E.B., & Rauch, S.A.M. (2004). Cognitive changes during prolonged exposure versus prolonged exposure plus cognitive restructuring in female assault survivors with posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 72,* 879 – 884.

Foa, E.B., & Riggs, D.S. (1993). Posttraumatic stress disorder in rape victims. In J. Oldham, M.B. Riba, & A. Tasman (Eds.), *American psychiatric press review of psychiatry.* (pp. 273 – 303). Washington, DC: American Psychiatric Press.

Foa, E.B., Riggs, D.S., Dancu, C.V., & Rothbaum, B.O. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress, 6,* 459 – 473.

Foa, E.B., Riggs, D.S., & Gershuny, B.S. (1995). Arousal, numbing and intrusion: Symptom structure of post traumatic stress disorder following assault. *American Journal of Psychiatry, 152,* 116 – 120.

Foa, E.B., & Rothbaum, B.O. (1998). *Treating the trauma of rape. Cognitive behavior therapy for PTSD.* New York: Guilford Press.

Foa, E.B., Rothbaum, B.O., & Furr, J.M. (2002). Augmenting exposure therapy with other CBT procedures. *Psychiatric Annals, 33,* 47 – 53.

Foa, E.B., Rothbaum, B.O., & Kozak, M.J. (1989). Behavioral treatments of anxiety and depression. In P. Kendall & D. Watson (Eds.), *Anxiety and depression: Distinctive and overlapping features.* (pp. 413 – 454) New York: Academic Books.

Foa, E.B., Rothbaum, B.O., Riggs, D.S., & Murdock, T. (1991). Treatment of post-traumatic stress disorder in rape victims: A comparison between cognitive-behavioral procedures and counseling. *Journal of Consulting and Clinical Psychology, 59,* 715 – 723.

Foa, E.B., Stein, D.J., & McFarlane, A.C. (2006). Symptomatology and psychopathology of mental health problems after disaster. *Journal of Clinical Psychiatry, 67, 15 – 25.*

Foa, E.B., Steketee, G., & Rothbaum, B.O., (1989). Behavioral/cognitive conceptualization of post-traumatic stress disorder. *Behavior Therapy, 20,* 155 – 176.

Foa, E.B., & Tolin, D.F. (2000). Comparison of the PTSD scale – interview version and the clinician-administered PTSD Scale. *Journal of Traumatic Stress, 13,* 181 – 191.

Foa, E.B., Zinbarg, R., & Rothbaum, B.O. (1992). Uncontrollability and unpredictability in posttraumatic stress disorder: An animal model. *Psychological Bulletin, 112,* 218 – 238.

Forbes, D., Creamer, M., & Rycroft, P. (1994). Eye movement desensitization and reprocessing in posttraumatic stress disorder: A pilot study using assessment measures. *Journal of Behavior Therapy and Experimental Psychiatry, 25,* 113 – 120.

Foster, D., Davis, D., & Sandler, D. (1987). *Detention and torture in South Africa: Psychological, legal and historical studies.* New York: St Martin's Press.

Fox, S.S., & Scherl, D.J. (1972). Crisis intervention with victims of rape. *Social Work, 17,* 37 – 42.

Foy, D.W., Glynn, S.M., Schnurr, P.P., Jankowski, M.K., Wattenberg, M.S., Weiss, D.S., et al. (2000). Group therapy. In E.B. Foa, T.M.Keane & M.J. Friedman (Eds.), *Effective treatments for PTSD. (*pp 336 – 338). New York: Guilford Press.

Foy, D.W., Sipprelle, R.C., Reuger, D.B., & Carroll, E.M. (1984). Etiology of posttraumatic stress disorder in Vietnam veterans: Analysis of premilitary, military, and combat exposure influences. *Journal of Consulting and Clinical Psychology, 52,* 79 – 87.

Frank, E., Anderson, B., Stewart, B.D., Dancu, C., Hughes, C., & West, D. (1988). Efficacy of cognitive behavior therapy and systematic desensitization in the treatment of rape trauma. *Behavior Therapy, 19,* 403 – 420.

Freeman, T.W., Cardwell, D., Karson, C.N., & Komoroski, R.A. (1998). In vivo proton magnetic resonance spectroscopy of the medial temporal lobes of subjects with combat-related posttraumatic stress disorder. *Magnetic Resonance in Medicine, 40,* 66 – 71.

Friedman, M.J. (2002). Future pharmacotherapy for posttraumatic stress disorder: Prevention and treatment. *Psychiatric Clinics of North America, 25,* 427 – 441.

Friedman, M.J., Ashcraft, M.L., Beals, J.L., Keane, T.M., Manson, S.M., & Marsella, A.J. (1997). *Matsunaga Vietnam Veterans Project.* White River Junction: VA National Center for PTSD.

Friedman, M.J., Davidson, J.R.T., Mellman, T.A., & Southwick, S.M. (2000). Pharmacotherapy. In E.B. Foa, T.M. Keane & M.J. Friedman (Eds.), *Effective treatments for PTSD. (*pp.84 – 105). New York: Guilford Press

Friedman, M.J., & Marsella, A.J. (1996). Posttraumatic stress disorder: An overview of the concept. In A.J. Marsella, M.J. Friedman, E.T. Gerrity, & R. Scurfield (Eds.), *Ethnocultural aspects of post-traumatic stress disorder: Issues, research, and clinical applications. (*pp. 11 – 32). Washington, DC: American Psychological Association.

Friedman, M.J., & Rosenheck, R.A. (1996). PTSD as a persistent mental illness. In S.M. Soreff (Ed.), *Handbook for the treatment of the seriously mentally ill* (pp. 369 – 389). Seattle: Hogrefe & Huber.

Friedman, M.J., & Schnurr, P.P. (1995). The relationship between trauma, post-traumatic stress disorder and physical health. In M.J. Friedman, D.S. Charney, & A.Y. Deutch (Eds.), *Neurobiological and clinical consequences of stress: From normal adaptation to post-traumatic stress disorder.* (pp. 507 – 524). Philadelphia: Lippencott-Raven.

Friston, K.J., Frith, C.D., Liddle, P.F., & Frackowiak, R.S. (1991). Comparing functional (PET) images: The assessment of significant change. *Journal of Cerebral Blood Flow Metabolism, 11,* 690 – 699.

Frueh, B.C., Turner, S.M., Beidel, D.C., Mirabella, R.F., & Jones, W.J. (1996). Trauma management therapy: A preliminary evaluation of a multicomponent behavioral treatment for chronic combat-related PTSD. *Behavior Research and Therapy, 34,* 533 – 543.

Furmark, T., Tillfors, M., Marteinsdottir, I., Fischer, H., Pissiota, A., Langstrom, B., et al. (2002). Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. *Archives of General Psychiatry, 59,* 425 – 433.

Galea, S., Ahern, J., Resnick, H., Kilpatrick, D., Bucuvalas, M., Gold, J., et al. (2002). Psychological sequelae of the September 11 terrorist attacks in New York City. *New England Journal of Medicine, 346,* 982 – 987.

Galea, S., Vlahov, D., Resnick, H., Ahern, J., Susser, E., Gold, J., et al. (2003). Trends of probable post-traumatic stress disorder in New York City after the September 11 terrorist attacks. *American Journal of Epidemiology, 158,* 514 – 524.

Gelinas, D.J. (1983). The persisting negative effects of incest. *Psychiatry, 46,* 312 – 332.

Gerschuny, B.S., Cloitre, M., & Otto, M.W. (2003). Peri-traumatic dissociation and PTSD severity: Do event-related fears about death and control mediate their relation? *Behaviour Research and Therapy, 41,* 157 – 166.

Gerschuny, B.S., Najvits, L.M., Wood, P.K., & Hepner, M. (2004). Relation between trauma and psychopathology: Mediating roles of dissociation and fears about death and control. *Journal of Trauma and Dissociation, 5,* 101 – 117.

Gilbertson, M.W., Shenton, M.E., Ciszewski, A., Kasai, K., Lasko, N.B., Orr, S.P., et al. (2002) Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Natural Neuroscience, 5,* 1242 – 1247.

Giller, J.E., Bracken, P.J., & Kabaganda, S. (1991). Uganda: War, women, and rape. *The Lancet, 337,* 604.

Glenn, D.M., Beckham, J.C., Feldman, M.E., Kirby, A.C., Hertzberg, M.A., & Moore, S.D. (2002). Violence and hostility among families of Vietnam veterans with combat-related posttraumatic stress disorder. *Violence and Victims, 17,* 473 – 489.

Goenjian, A.K., Steinberg, A.M., & Najarian, L.M. (2000). Prospective study of posttraumatic stress, anxiety, and depressive reactions after earthquake and political violence. *American Journal of Psychiatry, 157,* 911 – 916.

Goldapple, K., Segal, Z., Garson, C., Lau, M., Bieling, P., Kennedy, B.L., et al. (2004). Modulation of cortical-limbic pathways in major depression. *Archives of General Psychiatry, 61,* 34 – 41.

Golier, J., & Yehuda, R. (2002). Neuropsychological processes in posttraumatic stress disorder. *Psychiatric Clinics of North America, 25,* 295 – 315.

Golier, J.A., Yehuda, R., Lupien, S.J., Harvey, P.D., Grossman, R., & Elkin, A. (2002). Memory performance in Holocaust survivors with posttraumatic stress disorder. *American Journal of Psychiatry, 159,* 1682 – 1688.

Goodman, L.A., Corcoran, C., Turner, K., Yuan, N., & Green, B.L. (1998). Assessing traumatic event exposure: General issues and preliminary findings for the Stressful Life Events Screening Questionnaire. *Journal of Traumatic Stress, 11,* 521 – 542.

Gosselin, P., & Matthews, W.J. (1995). Eye movement desensitization and reprocessing in the treatment of test anxiety: A study of the effects of expectancy and eye movement. *Journal of Behavior Therapy and Experimental Psychiatry, 26,* 331 – 337.

Gottfried, J.A., & Dolan, R.J. (2004). Human orbitofrontal cortex mediates extinction learning while accessing conditioned representations of value. *Natural Neuroscence, 7,* 1144 – 1152

Grace, M.C., Green, B.L., & Lindy, J.D. (1993). The Buffalo Creek disaster: A 14-year follow-up. In J.P. Wilson, & B. Raphael, (Eds.), *International handbook of traumatic stress syndromes.* (pp 441 – 449) New York: Plenum Press.

Green, B.L., Grace, M.C., Lindy, J.D., & Gleser, G.C. (1990). War stressors and symptom persistence in posttraumatic stress disorder. *Journal of Anxiety Disorders, 4,* 31 – 39.

Greenwald, R. (1996). The information gap in the EMDR controversy. *Professional Psychology: Research and Practice, 27,* 67 – 72.

Grigsby, J.P. (1987). The use of imagery in the treatment of posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 175,* 55 – 59.

Gurvits, T.G., Gilberston, M.W., Lasko, N.B., Tarhan, A.S., Simeon, D., Macklin, M.L., et al. (2000). Neurologic soft signs in chronic posttraumatic stress disorder. *Archives of General Psychiatry, 57,* 181 – 186.

Gurvits, T.V., Shenton, M.E., Hokama, H., Ohta, H., Orr, S.P., Lasko, N.B., et al. (1996). Magnetic resonance imaging study of hippocampal volume in chronic, combat-related posttraumatic stress disorder. *Biological Psychiatry, 40,* 1091 – 1099.

Gutierrez, V.M., Zaidi, L.Y., & Lanktree, C.B. (1990). *Group treatment with sexually abused latency-aged girls.* Paper presented at the Seventh Biennial Meeting of the National Symposium on Child Victimization, Atlanta.

Hackman, A., Ehlers, A., Speckens, A., & Clark, D.M. (2004). Characteristics and content of intrusive memories in PTSD and their changes with treatment. *Journal of Traumatic Stress, 17,* 231 - 240

Halligan, S.L., & Yehuda, R. (2000). Risk factors for PTSD. *PTSD Research Quarterly, 11,* 1 – 3.

Hariri, A.R., Mattay, V.S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., et al. (2002) Serotonin transporter genetic variation and the response of the human amygdala. *Science, 297,* 400 – 403.

Heilemann, M.V., Kury, F.S., & Lee, K.A. (2005). Trauma and posttraumatic stress disorder symptoms among low income women of Mexican descent in the United States. *Journal of Nervous and Mental Disorders, 193,* 665 – 672.

Heilig, M., Koob, G.F., Ekman, R., & Britton, K.T. (1994). Corticotropin-releasing factor and neuropeptide Y: Role in emotional integration. *Trends in Neuroscience, 17,* 80 – 85.

Helzer, J.E., Robins, L.N., & McEvoy, L. (1987). Post-traumatic stress disorder in the general population: Findings of the epidemiologic catchment area survey. *New England Journal of Medicine, 317,* 1630 – 1634.

Heptinstall, E. (1996). *Healing the hidden hurt: The emotional effects of children's accidents.* London: Child Accident Prevention Trust.

Herman, J.L. (1992). Complex PTSD: A syndrome in survivors of prolonged and repeated trauma. *Journal of Traumatic Stress, 5,* 377 – 391.

Hermans, D., Craske, M.G., Mineka, S., & Lovibond, P.F., (2006). Extinction in human fear conditioning. *Biological Psychiatry, 60,* 361 – 368.

Hickling, E.J., & Blanchard, E.B. (1997). The private practice psychologist and manual-based treatments: Post-traumatic stress disorder secondary to motor vehicle accidents. *Behavior Research and Therapy, 35,* 191 – 203.

Hinton, W.L., Chen, Y.J., Du, N., Tran, C.G., Lu, F.G., Miranda, J., et al. (1993). DSM-III-R disorders in Vietnamese refugees. Prevalence and correlates. *Journal of Nervous and Mental Disease,181,* 113 – 122.

Hobbs, M., & Adshead, G. (1996). Preventive psychological intervention for road crash survivors. In M. Mitchell (ed.), *The aftermath of road accidents: Psychological, social and legal perspectives.* (pp. 159 – 171). London, UK: Routledge.

Hobbs, M., Mayou, R., Harrison, B., & Warlock, P. (1996). A randomized trial of psychological debriefing for victims of road traffic accidents. *British Medical Journal, 313,* 1438 – 1439.

Hogben, G.L., & Cornfield, R.B. (1981). Treatment of traumatic war neurosis with phenelzine. *Archives of General Psychiatry, 38,* 440 – 445.

Holen, A. (1991). A longitudinal study of the occurrence and persistence of posttraumatic health problems in disaster survivors. *Stress Medicine, 7,* 11 – 17.

Holman, E.A., Silver, R.C., & Waitzkin, H. (2000). Traumatic life events in primary care patients: A study in an ethnically diverse sample. *Archives of Family Medicine, 9,* 802 – 810.

Holmes, E.A., Brown, R.J., Mansell, W., Fearon, R.P., Hunter, E.C.M., Frasquilho, F., et al. (2005). Are there two qualitatively distinct forms of dissociation? A review and some clinical implications. *Clinical Psychology Review, 25,* 1 – 23.

Hope, D.A., Rapee, R.M., Heimberg, R.G., & Dombeck, M.J. (1990). Representations of the self in social phobia: Vulnerability to social threat. *Cognitive Therapy and Research, 14,* 177 – 189.

Horowitz, M.J. (1976). *Stress response syndromes.* Northvale, NJ: Jason Aronson.

Horowitz, M.J. (1986). *Stress response syndromes (2nd ed.).* New York: Jason Aronson.

Hough, R.L., Canino, G.J., Abueg, F.R., & Gusman, F.D. (1996). PTSD and related stress disorders among Hispanics. In A.J. Marsella, M.J. Friedman, E.T. Gerrity, & R.M. Scurfield (Eds.), *Ethnocultural aspects of post-traumatic stress disorder: Issues, research, and clinical applications. (*pp. 301 – 338). Washington, DC: American Psychological Assiciation.

Hubbard, J., Realmuto, G.M., Northwood, A.K., & Masten, I.S. (1995). Comorbidity of psychiatric diagnoses with posttraumatic stress disorder in survivors of childhood trauma. *Journal of the American Academy of Child and Adolescent Psychiatry, 34,* 1167 – 1173.

Hudson, J.I., & Pope, H.G. (1990). Affective spectrum disorder: Does antidepressant response identify a family of disorders with a common pathophysiology? *American Journal of Psychiatry, 5,* 552 – 564.

Husum, H., & Mathe, A.A. (2002). Early life stress changes concentrations of neuropeptide Y and corticotropin-releasing hormone in adult rat brain. Lithium treatment modifies these changes. *Neuropsychopharmacology, 27,* 756 – 764.

Irving, P., & Long, A. (2001). Critical incident stress debriefing following traumatic life experiences. *Journal of Psychiatric and Mental Health Nursing, 8,* 307 – 314.

Janoff-Bulman, R. (1985). The aftermath of victimization: Rebuilding shattered assumptions. In C.R. Figley (Ed.), *Trauma and its wake: The study and treatment of post-traumatic stress* disorder. (pp. 15 – 35). New York: Brunner/Mazel.

Janoff-Bulman, R. (1992). *Shattered assumptions: Towards a new psychology of trauma.* New York: The Free Press.

Janoff-Bulman, R., & Frieze, J.H. (1983). A theoretical perspective for understanding reactions to victimization. *Journal of Social Issues, 39,* 1 – 17.

Jenkins, J.H. (1991). The state construction of affect: Political ethos and mental health among Salvadoran refugees. *Culture, Medicine and Psychiatry, 15,* 139 – 165.

Jenkins, M.A., Langlais, P.J., Delis, D., & Cohen, R. (1998). Learning and memory in rape victims with posttraumatic stress disorder. *American Journal of Psychiatry, 155,* 278 – 279.

Jensen, J.A. (1994). An investigation of Eye Movement Desensitization and Reprocessing (EMD/R) as a treatment for posttraumatic stress disorder (PTSD) symptoms of Vietnam combat veterans. *Behavior Therapy, 25,* 311 – 325.

Jiranek, D. (1993). Use of hypnosis in pain management and posttraumatic stress disorder. *Australian Journal of Clinical and Experimental Hypnosis, 21,* 75 – 84.

Joe, J. (1994). Traditional Indian health practices and cultural views. In D. Champagne (Ed.), *Native America portrait of the peoples.* (pp. 525 – 547). Detroit: Visible Ink Press.

Johnson, C.H., Gilmore, J.D., & Shenoy, R.Z. (1982). Use of a feeding procedure in the treatment of a stress-related anxiety disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 13,* 235 – 237.

Joseph, S., Williams, R., & Yule, W. (1997). *Understanding post-traumatic stress. A psychosocial perspective on PTSD and treatment.* Chichester, UK: Wiley.

Kagee, A. (2004). Do South African former detainees experience posttraumatic stress? Circumventing the demand characteristics of psychological assessment. *Transcultural Psychiatry, 41,* 323 – 336.

Kagee, A. (2005). Symptoms of distress and posttraumatic stress among South African former political detainees. *Ethnicity and Health, 10,* 169 – 179.

Kagee, A., & Naidoo, A.V. (2004). Reconceptualizing the sequelae of political torture: Limitations of a psychiatric paradigm. *Transcultural Psychiatry, 41,* 46 – 61.

Kakar, S. (1985). Psychoanalysis and non-Western cultures. *International Review of Psychoanalysis, 12,* 441 447.

Kaminer, D., Stein, J., Mbanga, I., & Zungu-Dirwayi, N. (2001). The truth and reconciliation commission in South Africa: Relation to psychiatric status and forgiveness among survivors of human rights abuses. *British Journal of Psychiatry, 178,* 373 – 377.

Kaplan, H.I., & Sadock, B.J. (1998). *Synopsis of psychiatry - Behavioural sciences/ clinical psychiatry (8th ed.).* Baltimore, MD: Williams & Wilkins.

Karamustafalioglu, O.K., Zohar, J., Guveli, M., Gal, G., Bakim, B., Fostick, L., et al. (2006). Natural course of posttraumatic stress disorder: A 20-month prospective study of Turkish earthquake survivors. *Journal of Clinical Psychiatry, 67,* 882 – 889.

Keane, T.M., Caddell, J.M., & Taylor, K.L. (1988). Mississippi scale for combat-related post-traumatic stress disorder: three studies in reliability and validity. *Journal of Consulting and Clinical Psychology, 56,* 85 – 90.

Keane, T.M., Fairbank, J.A., Caddell, J.M., & Zimering, R.T. (1989). Implosive (flooding) therapy reduces symptoms of PTSD in Vietnam combat veterans. *Behavior Therapy, 20,* 245 – 260.

Keane, T.M., & Kaloupek, D.G. (1982). Imaginal flooding in the treatment of post-traumatic stress disorder. *Journal of Consulting and Clinical Psychology, 50,* 138 – 140.

Keane, T.M., Malloy, P.F., & Fairbank, J.A. (1984). Empirical development of an MMPI subscale for the assessment of combat-related post-traumatic stress disorder. *Journal of Consulting and Clinical Psychology, 52,* 888 – 891.

Keane, T.M., & Wolfe, J.A. (1990). Comorbidity in posttraumatic stress disorder: An analysis of community and clinical studies. *Journal of Applied Social Psychology, 20,* 1776 – 1788.

Keane, T.M., Zimmerling, R.T., & Caddell, J.M. (1985). A behavioral formulation of post-traumatic stress disorder in Vietnam veterans. *The Behavior Therapist, 8,* 9 – 12.

Keats, D.M., Munro, D., & Mann, L. (1989). Heterogeneity in cross-cultural psychology. *Selected papers from the 9th International Conference of the International Association for Cross-cultural Psychology.* Newcastle, Australia: Author.

Kessler, R.C., Borges, G., & Walters, E.E. (1999). Prevalence of and risk factors for lifetime suicide attempts in the National Comorbidity Survey. *Archives of General Psychiatry, 56,* 617 – 626.

Kessler, R.C., Chiu, W.T., Demler, O., Merikangas, K.R., & Walters, E.E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 6,* 617 – 627.

Kessler, R.C., McGonagle, K.A., Zhao, S., Nelson, C., Hughes, M., Eshelman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. *Archives of General Psychiatry, 51,* 8 – 19.

Kessler, R.C., & McLeod, J.E. (1984). Sex differences in vulnerability to undesirable life events. *American Social Review, 49,* 620 – 631.

Kessler, R.C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry, 52,* 1048 – 1060.

Kilpatrick, D.G. (1983). Rape victims: Detection, assessment, and treatment. *The Clinical Psychologist, 36,* 92 – 95.

Kilpatrick, D.G., Veronen, L.J., & Resick, P.A. (1982). Psychological sequelae to rape: Assessment and treatment strategies. In D.M. Dolays & R.L. Meredith (Eds.), *Behavioral medicine: Assessment and treatment strategies* (pp. 473 – 497). New York: Plenum Press.

Kimonides, V.G., Khatibi, N.H., Svendsen, C.N., Sofroniew, M.V., & Herbert, J. (1998). Dehydroepiandrosterone (DHEA) and DHEA-sulphate (DHEAS) protect hippocampal neurons against excitatory amino acid-induced neurotoxicity. *Proceedings of the National Academy of Sciences of the United States of America, 95,* 1852 – 1857.

King, D.W., King, L.A., Foy, D.W., & Gudanowski, D.M. (1996). Prewar factors in combat-related posttraumatic stress disorder: Structural equational modeling with a national sample of female and male Vietnam veterans. *Journal of Consulting and Clinical Psychology, 64,* 520 – 531.

King, L.A., King, D.W., Fairbank, J.A., Keane, T.M., & Adams, G.A. (1998). Resilience-recovery factors in posttraumatic stress disorder among female and male Vietnam veterans: Hardiness, postwar social support, and additional stressful life events. *Journal of Personality and Social Psychology, 74,* 420 – 434.

Kingsbury, S.J. (1988). Hypnosis in the treatment of posttraumatic stress disorder: An isomorphic intervention. *American Journal of Clinical Hypnosis, 31,* 81 – 90.

Kirmayer, L.J. (1996). Confusion of the senses: Implications of ethnocultural variations in somatoform and dissociative disorders for PTSD. In A.J. Marsella, M.J. Friedman, E.T. Gerrity, & R.M. Scurfield (Eds.), *Ethnocultural aspects of posttraumatic stress disorder: Issues, research, and clinical applications* (pp. 131 – 163). Washington, DC: American Psychological Association.

Kitayama, N., Vaccarino, V., & Kuther, M., (2005). Magnetic resonance imaging (MRI) measurement of hippocampal volume in posttraumatic stress disorder: A meta-analysis. *Journal of Affective Disorders, 88,* 79 – 86.

Koenen, K.C., (2003). A brief introduction to genetics research in PTSD. *PTSD Research Quarterly, 14,* 1 – 2.

Koenen, K.C., Driver, K.L., Oscar-Berman, M., Wolfe, J., Folsom, S., Huang, M.T., et al. (2001). Measures of prefrontal system dysfunction in posttraumatic stress disorder. *Brain and Cognition*, 45, 64 – 78.

Koenen, K.C., Harley, R., Lyons, M.J., Wolfe, J., Simpson, J.C., Goldberg, J., et al. (2002). A twin registry study of familial and individual risk factors for trauma exposure and posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 190,* 209 – 218.

Koenen, K.C., Lyons, M.J., Goldberg, J., Simpson, J., Williams, W.M., Toomey, R., et al. (2003). A high risk twin study of combat-related PTSD comorbidity. *Twin Research, 6,* 218 – 226.

Kolb, L. (1987). A neuropsychological hypothesis explaining posttraumatic stress disorder. *American Journal of Psychiatry, 144,* 989 – 995.

Komesaroff, P.A., Esler, M.D., & Sudhir, K. (1999). Estrogen supplementation attenuates glucocorticoid and catecholamine responses to mental stress in perimenopausal women. *Journal of Clinical Endocrinology and Metabolism, 84,* 606 – 610.

Koopman, C., Classen, C., & Spiegel, D. (1994). Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, California firestorm. *American Journal of Psychiatry, 151,* 888 – 894.

Kopel, H., & Friedman, M. (1997). Posttraumatic symptoms in South African police exposed to violence. *Journal of Traumatic Stress, 10,* 307 – 317.

Kosslyn, S.M., Alpert, N.M., & Thompson, W.L. (1993). Visual imagery activates topographically organized visual cortex: PET investigations. *Journal of Cognitive Neuroscience, 5,* 263 – 287.

Krinsley, K.E., Gallagher, J.G., Weathers, F.W., Kutter, C.J., & Kaloupek, D.G. (2003). Consistency of retrospective reporting about exposure to traumatic events. *Journal of Traumatic Stress, 16,* 399 – 409.

Kroll, J., Habenicht, M., MacKenzie, T., Yang, M., Chan, S., Vang, T., et al. (1989). Depression and posttraumatic stress disorder in Southeast Asian refugees. *American Journal of Psychiatry,146,* 1592 – 1597.

Krueger, R.F., McGrue, M., & Iacono, W.G. (2001). The higher-order structure of common DSM mental disorders: Internalization, externalization, and their connections to personality. *Personality and Individual differences, 30,* 1245 – 1259.

Krystal, J.H., Southwick, S.M., & Charney, D.S. (1995). Posttraumatic stress disorder: Psychobiological mechanisms of traumatic remembrance. In D.L. Schacter (Ed.), *Memory distortions: How minds, brains, and societies reconstruct the past.* Cambridge, MA: Harvard University Press.

Kulka, R.A., Schlenger, W.E., Fairbank, J.A., Hough, R.L., Jordan, B.K., Marmar, C.R., et al. (1990). *Trauma and the Vietnam War generation: Report of findings from the National Vietnam Veterans Readjustment Study.* New York: Brunner/Mazel.

Lader, M. (1981). Assessment of depression. *British Journal of Clinical Pharmacology, 1,* 5 – 14.

Laffaye, C., Kennedy, C., & Stein, M.B. (2003). Posttraumatic stress disorder and health-related quality of life in female victims of intimate partner violence. *Violence and Victims, 18,* 227 – 238.

Lang, P.J. (1977). Imagery in therapy: An information processing analysis of fear. *Behavior Therapy, 8,* 862 – 886.

Lang, P.J. (1979). Bio-informational theory of emotional imagery. *Psychophysiology, 16,* 495 – 512.

Last, M. (1993). Non-Western concepts of disease. In W.F. Bynum & R. Poster (Eds.), *Companion Encyclopedia of the History of Medicine* (pp. 634 – 659). New York: Routledge

Lavender, T., & Walkinshaw, S.A. (1998). Can midwives reduce postpartum morbidity? A randomized trial. *Birth, 25,* 215 – 219.

Lebron, K., Milad, M.R., & Quirk, G.J. (2004). Delayed recall of fear extinction in rats with lesions of ventral medial prefrontal cortex. *Learning and Memory, 11,* 544 – 548.

Lecrubier, Y. (2004). Posttraumatic stress disorder in primary care: A hidden diagnosis. *Journal of Clinical Psychiatry, 65,* 49 – 54.

Lee, C., Slade, P., & Lygo, V. (1996). The influence of psychological debriefing on emotional adaption in women following early miscarriage: A preliminary study. *British Journal of Medical Psychology, 69,* 47 – 58.

Lee, K.A., Vaillant, G.E., Torrey, W.C., & Elder, G.H. (1995). A 50-year prospective study of the psychological sequelae of World War II combat. *American Journal of Psychiatry, 152,* 516 – 522.

Lenox, R.H., Gould, T.D., & Manji, H.K. (2002). Endophenotypes in bipolar disorder. *American Journal of Medical Genetics, 114,* 391 – 406.

Leonard, R., & Alison, L. (1999). Critical incident stress debriefing and its effects on coping strategies and anger in a sample of Australian police officers involved in shooting incidents. *Work and Stress, 13,* 144 – 161.

Leserman, J., Drossman, D.A., & Li, Z. (1995). The reliability and validity of a sexual and physical abuse history questionnaire in female patients with gastrointestinal disorders. *Behavioral Medicine, 21,* 141 – 150.

Leskin, G.A., Ruzek, J.I., Friedman, M.J., & Gusman, F.D. (1999). Effective clinical management of PTSD in primary care settings: Screening and treatment options. *Primary Care Psychiatry, 5,* 3 – 12.

Leung, J. (1994). Treatment of post-traumatic stress disorder with hypnosis. *Australian Journal of Clinical and Experimental Hypnosis, 22,* 87 – 96.

Lewin, T.J., Carr, V.J., & Webster, R.A. (1998). Recovery from post-earthquake psychological morbidity: Who suffers and who recovers? *Australian and New Zeeland Journal of Psychiatry, 32,* 15 – 20.

Lightfoot, S.L., & Oliver, J.M. (1985) The beck inventory: Psychometric properties in university students. *Journal of Personality Assessment, 49,* 434 – 436.

Lindy, J.D., Green, B.L., Grace, M., & Titchener, J. (1983). Psychotherapy with survivors of the Beverly Hills Supper Club fire. *American Journal of Psychotherapy, 4,* 593 – 610.

Lira, L., & Koss, M. (1999). Mexican American women's definitions of rape and sexual abuse. *Hispanic Journal of Behavioural Sciences, 21,* 236 – 254.

Litz, B.T., (1992). Emotional numbing in combat-related posttraumatic stress disorder: A critical review and reformulation. *Clinical Psychology Review, 12,* 417 – 432.

Litz, B.T., Weathers, F.W., Monaco, V., Herman, D.S., Wulfsohn, M., Marx, B., et al. (1996). Attention, arousal, and memory in posttraumatic stress disorder. *Journal of Traumatic Stress, 9,* 497 – 519.

Lohr, J.M., Tolin, D.F., & Lilienfeld, S.O. (1998). Efficacy of eye movement desensitization and reprocessing: Implications for behavior therapy. *Behavior Therapy, 29,* 123 – 156.

Lutz, C.A. (1988). *Unnatural emotions: Everyday sentiments on a Micronesian atoll and their challenge to Western theory.* Chicago: University of Chicago Press.

MacHovec, F.J. (1983). Treatment variables and the use of hypnosis in the brief therapy of post-traumatic stress disorders. *International Journal of Clinical and Experimental Hypnosis, 1,* 6 – 14.

Macklin, M.L., Metzger, L.J., Litz, B.T., McNally, R.J., Lasko, N.B., Orr, S.P., et al. (1998) Lower pre-combat intelligence is a risk factor for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 66,* 323 – 326.

Malvone, E.M., Bruch, M.A., & Heimberg, R.G. (1993). Focus of attention and social anxiety: The role of negative self-thoughts and perceived positive attributes of the other. *Cognitive Therapy and Research, 17,* 207 – 224.

March, J.S. (1993). What constitutes a stressor? The "A" criterion issue. In R.T. Davidson & E.B. Foa. *Posttraumatic Stress Disorder and beyond* (pp. 37 – 56). Washington, DC: American Psychiatric Press.

Marcus, S.V., Marquis, P., & Sakai, C. (1997). Controlled study of treatment of PTSD using EMDR in an HMO setting. *Psychotherapy, 34,* 307 – 315.

Markowitsch, H.J. (1995). Which brain regions are critically involved in the retrieval of old episodic memory? *Brain Research Reviews, 21,* 117 – 127.

Marks, I.M. (1987). *Fears, phobias and rituals.* New York: Oxford University Press

Marks, I., Lovell, K., Noshirvani, H., Livanou, M., & Thrasher, S. (1998). Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: A controlled study. *Archives of General Psychiatry, 55,* 317 – 325.

Marmar, C.R., Weiss, D.S., Metzler, T.J., Delucchi, K.L., Best, S.R., & Wentworth, K.A. (1999). Longitudinal course and predictors of continuing stress following critical incident exposure in emergency services personnel. *Journal of Nervous and Mental Disease, 187,* 15 – 22.

Marsella, A.J., Friedman, M.J., Gerrity, E.T., & Scurfield, R.M. (Eds.), (1996). *Ethnocultural aspects of posttraumatic stress disorder: Issues, research, and clinical applications.* (pp. 105 – 129). Washington, DC: American Psychological Association.

Marshall, R.D., & Rothbaum, B. (2004). Posttraumatic stress disorder and acute stress disorder. In D.J. Stein (Ed.), *Clinical manual of anxiety disorders.* (pp. 119 – 146). Washington, DC: American Psychiatric Publishing.

Martin, J., Anderson, J., Romans, S., Mullen, P., & O'Shea, M. (1993). Asking about child sexual abuse: Methodological implications of a two stage survey. *Child Abuse and Neglect, 17,* 383 – 392.

Masten, A.S., & Coatsworth, J.D. (1998). The development of competence in favorable and unfavorable environments: Lessons from research on successful children. *American Psychologist, 53,* 205 – 220.

Matsumoto, D. (1989). Cultural influences on the perception of emotion. *Journal of Cross-Cultural Psychology, 20,* 92 – 105.

Mattia, J.L., Heimberg, R.G., & Hope, D.A. (1993). The revised Stroop Color-naming Task in social phobias. *Behaviour Research and Therapy, 31,* 305 – 313.

Mayou, R., Bryant, B., & Duthie, R. (1993). Psychiatric consequences of road traffic accidents. *British Medical Journal, 307,* 647 – 651.

McCabe, K.A., & Gregory, S.S. (1998). Elderly victimization: An examination beyond the FBI's index crimes. *Research on aging, 20,* 363 – 372.

McCann, I.L., & Pearlman, L.A. (1990). *Psychological trauma and the adult survivor: Theory, therapy, and transformation.* New York: Brunner Mazel.

McCann, I.L., Sakheim, D.K., & Abrahamson, D.J. (1988). Trauma and victimization: A model of psychological adaptation. *The Counseling Psychologist, 16,* 531 – 594.

McEwen, B.S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine, 153,* 2093 – 2101.

McFarlane, A.C. (1988). The longitudinal course of posttraumatic morbidity: The range of outcomes and their predictors. *Journal of Nervous and Mental Disease, 176,* 30 – 39.

McFarlane, A.C. (1989). The aetiology of post-traumatic morbidity: Predisposing, precipitating and perpetuating factors. *British Journal of Psychiatry, 154,* 221 – 228.

McFarlane, A.C. (1992a). Avoidance and intrusion in posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 180,* 439 – 445.

McFarlane, A.C. (1992b). The phenomenology of posttraumatic stress disorder following a natural disaster. *Journal of Nervous and Mental Disease, 180,* 22 – 29.

McFarlane, A.C. (2000). Posttraumatic stress disorder: A model of the longitudinal course and the role of risk factors. *Journal of Clinical Psychiatry, 61,* 15 – 20.

McFarlane, A.C. (2002). Phenomenology of posttraumatic stress disorder. In D.J. Stein and E. Hollander (Eds). *Textbook of anxiety disorders. (*pp. 359 – 371). Washington DC: American Psychiatric Publishing.

McFarlane, A.C., & Papay P. (1992). Multiple diagnoses in posttraumatic stress disorder in the victims of a natural disaster. *Journal of Nervous and Mental Disorders, 180,* 498 – 504.

McGaugh J.L. (2004). The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annual Revue of Neuroscience, 27,* 1 – 28.

McNally, R.J. (1999). Research on eye movement desensitization and reprocessing (EMDR) as a treatment for PTSD. *PTSD Research Quarterly, 10,* 1 – 8.

McNally, R.J., Kaspi, S.P., Riemann, B.C., & Zeittin, S.B. (1990). Selective processing of threat cues in post-traumatic stress disorder. *Journal of Abnormal Psychology, 99,* 398 – 402.

Mechanic, M.B., Resick, P.A., & Griffin, M.G. (1998). A comparison of normal forgetting, psychopathology, and information-processing models of reported amnesia for recent sexual trauma. *Journal of Consulting and Clinical Psychology, 66,* 948 – 957.

Meichenbaum, D. (1975). Self-instructional methods. In F.H. Kanfer & A.P. Goldstein (Eds.), *Helping people change* (pp. 357 – 391). New York: Pergamon.

Mellman, T.A., & Davis, G.C. (1985). Combat-related flashbacks in posttraumatic stress disorder: Phenomenology and similarity to panic attacks. *Journal of Clinical Psychiatry, 46*, 379 – 382.

Michael, T. (2000). *The nature of trauma memory and intrusive cognitions in posttraumatic stress disorder.* Unpublished doctoral dissertation, University of Oxford, United Kingdom.

Milad, M.R., Rauch, S.L., Pitman, R.K., & Quirk, G.J. (2006). Fear extinction in rats: Implications for human brain imaging and anxiety disorders. *Biological Psychology, 73,* 61 – 71.

Miller, M.W. (2004). Personality and the development and expression of PTSD. *PTSD Research Quarterly, 15,* 1 – 4.

Miller, B., Downs, W., & Testa, M. (1993). Interrelationships between victimization experiences and women's alcohol use. *Journal of Studies on Alcohol, 11,* 109 – 117.

Miller-Burke, J., Attridge, M., & Fass, P.M. (1999). Impact of traumatic events and organizational response – study of bank robberies. *Journal of Occupational and Environmental Medicine, 42,* 73 – 83.

Mitchell, J.T. (1983). When disaster strikes. The critical incident stress debriefing process. *Journal of Emergency Medical Services, 8,* 36 – 39.

Moller, C., Sommer, W., Thorsell, A., & Heilig, M. (1999). Anxiogenic-like action of galanin after intra-amygdala administration in the rat. *Neuropsychofarmachology, 21,* 507 – 512.

Mollica, R.F., & Caspi-Yavin, Y. (1991). Measuring torture and torture-related symptoms. *Psychological Assessment, 3,* 581 – 587.

Mollica, R.F., Wyshak, G., & Lavelle, J. (1987). The psychosocial impact of war trauma and torture on Southeast Asian refugees. *American Journal of Psychiatry, 144,* 1567 – 1572.

Moran, P.W., & Lambert, M.J. (1983). A review of current assessment tools for monitoring changes in depression. In M.S. Lambert, E.R. Christensen, & S.S. DeJulio (Eds.), *The assessment of psychotherapy outcome.* New York: Wiley.

Morgan, C.A., Wang, S., Mason, J., Southwick, S.M., Fox, P., Hazlett, G., et al. (2000). Hormone profiles in humans experiencing military survival training. *Biological Psychiatry, 47,* 891 – 901.

Morgan, C.A., Wang, S., Southwick, S.M., Rasmusson, A., Hazlett, G., Hauger, R.L., et al. (2000). Plasma neuropeptide-Y concentrations in humans exposed to military survival training. *Biological Psychiatry, 47,* 902 – 909.

Murphy, S.A., Braun, T., Tillery, L., Cain, K.C., Johnson, L.C., & Beaton, R.D. (1999). PTSD among bereaved parents following the violent deaths of their 12 to 28 year-old children: A longitudinal prospective analysis. *Journal of Traumatic Stress, 12,* 273 – 291.

Murray, J., Ehlers, A., & Mayou, R.A. (2002). Dissociation and posttraumatic stress disorder: Two prospective studies of road traffic accident victims. *British Journal of Psychiatry, 180,* 363 – 368.

Nadel, L., & Moscovitch, M., (1998). Hippocampal contributions to cortical plasticity. *Neuropharmacology, 37,* 431 – 439.

Naidoo, A.V. (1996). Challenging the hegemony of Eurocentric psychology. *Journal of Community and Health Sciences, 2,* 9 – 16.

Najavits, L.M., & Garber, J. (1989). *A cognitive-behavioural group therapy curriculum for inpatient depressed adolescents and adults.* Unpublished manuscript, Vanderbilt University.

Najavits, L.M., Weiss, R.D., & Liese, B.S. (1996). Group cognitive-behavioural therapy for women with PTSD and Substance Use Disorder. *Journal of Substance Abuse Treatment, 13,* 13 – 22.

Nemeroff, C.B., Bremner, J.D., Foa, E.B., Mayberg, H.S., North, C.S., & Stein, M.B. (2006). Posttraumatic stress disorder: A state-of-the-science review. *Journal of Psychiatric Research, 40,* 1 – 21.

Neylan, T.C., Lenoci, M., Rothlind, J., Metzler, T.J., Schuff, N., Du, A., et al. (2004). Attention, learning, and memory in posttraumatic stress disorder. *Journal of Traumatic Stress, 17,* 41 – 46.

Nie, N.H., Hull, C.H., Jenkins, J.G., & Steinbrenner, K. (1975). Statistical package for the social sciences. In T.B. Pretorius. *Inferential statistics: Hypothesis testing and decision-making* (pp. 20 – 29). Cape Town: Percept Publishers.

Nixon, R.V., Nishith, P., & Resick P.A. (2004). The accumulative effect of trauma exposure on short-term and delayed verbal memory in a treatment-seeking sample of female rape victims. *Journal of Traumatic Stress, 17,* 31 – 35.

Nordstrom, C. (1992). The backyard front. In C. Nordstrom & J. Martin (Eds.), *The paths to domination, resistance, and terror* (pp. 260 – 274). Berkeley: University of California Press.

Norris, F.H. (1992). Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. *Journal of Consulting and Clinical Psychology, 60,* 409 – 418.

Norris, F.H., & Alegria, M. (2005). Mental health care for ethnic minority individuals and communities in the aftermath of disaster and mass violence. *CNS Spectrums, 10,* 123 – 131.

Norris, F.H., Friedman, M.J., & Watson, P.J. (2002). 60,000 disaster victims speak: An empirical review of the empirical literature, 1981 – 2001. *Psychiatry, 65,* 207 – 239.

Norris, F.H., Murphy, A.D., Baker, C.K., & Perilla, J.L. (2003). Severity, timing, and duration of reactions to trauma in the population: An example from Mexico. *Biological Psychiatry, 53,* 769 – 778.

Norris, F.H., Perilla, J.L., Ibanez, G., & Murphy, A. (2001). Sex differences in symptoms of posttraumatic stress: Does culture play a role? *Journal of Traumatic Stress, 14,* 7 – 28.

Norris, F., Perilla, J., & Murphy, A. (2001). Postdisaster stress in the United States and Mexico: A cross-cultural test of the multicriterion conceptual model of posttraumatic stress disorder. *Journal of Abnormal Psychology, 110,* 553 – 563.

Norris, F., Weisshaar, D., Conrad, L., Diaz, E., Murphy, A., & Ibanez, G. (2001). A qualitative analysis of posttraumatic stress disorder among Mexican victims of disaster. *Journal of Traumatic Stress, 14,* 741 – 756.

North, C.S., Nixon, S.J., Shariat, S., Mallonee, S., McMillen, J.C., Spitznagel, E.L., et al. (1999). Psychiatric disorders among survivors of the Oklahoma City bombing. *Journal of the American Medical Association, 282,* 755 – 762.

North, C.S., Smith, E.M., & Spitznagel, E.L. (1997). One-year follow-up of survivors of a mass shooting. *American Journal of Psychiatry, 154,* 1696 – 1702.

Nutt, D.J., & Malizia, A.L. (2001). New insights into the role of the GABA-benzodiazepine receptor in psychiatric disorder. *British Journal of Psychiatry, 179,* 390 – 396.

O'Toole, B.I., Marshall, R.P., Schureck, R.J., & Dobson, M. (1998). Risk factors for posttraumatic stress disorder in Australian Vietnam veterans. *Australian and New Zealand Journal of Psychiatry, 32,* 21 – 31.

Ozer, E.J., Best, S.R., Lipsey, T.L., & Weiss, D.S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin, 129,* 52 – 73.

Paker, M., Paker, O., & Yuksel, S. (1992). Psychosocial effects of torture: An empirical study of tortured and non-tortured political prisoners. In M. Basoglu (Ed.), *Torture and its consequences* (p. 72). Cambridge: Cambridge University Press.

Palinkas, L.A. (1995). Health under stress: Asian and Central American refugees and those left behind. *Social and Science Medicine, 40,* 1591 – 1596.

Paquette, V., Levesque, J., Mensour, B., Leroux, J.M., Beaudoin, G., Bourgouin, P., et al. (2003). "Change the mind and you change the brain": Effects of cognitive-behavioral therapy on the neural correlates of spider phobia. *Neuroimage, 18,* 401 – 409.

Peebles, M.J. (1989). Through a glass darkly: The psychoanalytic use of hypnosis with post-traumatic stress disorder. *International Journal of Clinical and Experimental Hypnosis, 37,* 192 – 206.

Perkonigg, A., Kessler, R.C., & Storz, S. (2000). Traumatic events and posttraumatic stress disorder in the community: Prevalence, risk factors and comorbidity. *Acta Psychiatrica Scandinavia, 101,* 46 – 59.

Phelps, E.A., Delgado, M.R., Nearing, K.I., & LeDoux, J.E. (2004). Extinction learning in humans: Role of the amygdala and vmPFC. *Neuron, 43,* 897 – 905.

Pillemer, K., & Finkelhor, D. (1988). The prevalence of elder abuse: A random sample survey. *The Gerontologist, 28,* 51 – 57.

Piotrowski, C., Sherry, D., & Keller, J.W. (1985). Psychodiagnostic test usage: A survey of the Society for Personality Assessment. *Journal of Personality Assessment, 49,* 115 – 119.

Pillay, B.J. (2000). Providing mental health services to survivors: A KwaZulu Natal perspective. *Ethnicity and Health, 5*, 269 – 272.

Pitman, R.K. (1989). Post-traumatic stress disorder, hormones, and memory. *Biological Psychiatry, 26,* 221 – 223.

Pitman, R.K., Altman, B., Greenwald, E., Longpre, R.E., Macklin, M., Poire, R.E., et al. (1991). Psychiatric complications during flooding therapy for posttraumatic stress disorder. *Journal of Clinical Psychiatry, 52,* 17 – 20.

Pitman, R.K., Orr, S.P., Altman, B., Longpre, R.E., Poire, R.E., & Macklin, M.L. (1996). Emotional processing during eye-movement desensitization and reprocessing (EMDR) therapy of Vietnam veterans with chronic post-traumatic stress disorder. *Comprehensive Psychiatry, 37,* 419 – 429.

Pitman, R.K., Shin, L.M., & Rauch, S.L. (2001). Investigating the pathogenesis of posttraumatic stress disorder with neuroimaging. *Journal of Clinical Psychiatry, 62,* 47 – 54.

Punamaki, R.L. & Suleiman, R. (1990). Predictors and effectiveness of coping with political violence among Palestinian children. *British Journal of Social Psychology, 29,* 67 – 77.

Pynoos, R.S., Frederick, C., & Nader, K. (1987). Life threat and posttraumatic stress in school-age children. *Archives of General Psychiatry, 44,* 1057 – 1063.

Quirk, G.J., Garcia, R., & Gonzalez-Lima, F. (2006). Prefrontal mechanisms in extinction of conditioned fear. *Biological Psychiatry, 60,* 337 – 343.

Rachman, S. (1980). Emotional processing. *Behaviour Research and Therapy, 18,* 51 – 60.

Rachman, S. (2001). Emotional processing, with special reference to post-traumatic stress disorder. *International Review of Psychiatry, 13,* 164 – 171.

Radant, A., Tsuang, D., Peskind, E.R., McFall, M., & Raskind, W. (2001). Biological markers and diagnostic accuracy in the genetics of posttraumatic stress disorder. *Psychiatry Research, 102,* 203 – 215.

Raphael, B. (1986). *When disaster strikes:A handbook for caring professions.* London: Hutchinson.

Rasmussen, A.M., & Charney, D.S. (1997). Animal models of relevance to PTSD. *Annals of the New York Academy of Sciences, 821,* 332 – 351.

Rasmussen, A. M., Hanger, R.L., Morgan, C.A., Bremner, J.D., Charney, D.S., & Southwick, S.M. (2000). Low baseline and yohimbine-stimulated plasma neuropeptide Y(NPY) levels in combat related PTSD. *Biological Psychiatry, 47,* 526 – 539.

Rauch, S., & Foa, E. (2006). Emotional Processing Theory (E.P.T.) and exposure therapy for PTSD. *Journal of Contemporary Psychotherapy, 36,* 61 – 65.

Rauch, S.L., Shin, L.M., & Phelps, E.A. (2006). Neurocircuitry models of posttraumatic stress disorder and extinction: Human neuroimaging research – past, present and future. *Biological Psychiatry, 60,* 376 – 382.

Rauch, S.L., Shin, L.M., & Whalen, P.J. (1998). Neuroimaging and the neuro-anatomy of posttraumatic stress disorder. *CNS Spectrum, 7,* 31 – 41.

Rauch, S.L., van der Kolk, B.A., & Fisler, R.E. (1996). A sympton provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. *Archives of General Psychiatry, 53,* 380 – 387.

Renfrey, G., & Spates, C.R. (1994). Eye movement desensitization: A partial dismantling study. *Journal of Behavior Therapy and Experimental Psychiatry, 25,* 231 – 239.

Reppesgaard, H.O. (1997). Studies on psychosocial problems among displaced people in Sri Lanka. *European Journal of Psychiatry, 11,* 223 – 234.

Resick, P.A., Jordan, C.G., Girelli, S.A., Hutter, C.K., & Marhoefer-Dvorak, S. (1988). A comparative victim study of behavioral group therapy for sexual assault victims. *Behavioral Therapy, 19,* 385 – 401.

Resick, P.A., Nishith, P., & Astin, M. (1998). *A controlled trial comparing cognitive processing therapy and prolonged exposure: Preliminary findings.* Paper presented at the Lake George Research Conference on posttraumatic stress disorder, Lake George, New York.

Resick, P.A., & Schnicke, M.K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting Clinical Psychology, 60,* 748 – 756.

Resnick, H., Galea, S., Kilpatrick, D., & Vlahov, D. (2004). Research on trauma and PTSD in the aftermath of 9/11. *PTSD Research Quarterly, 15,* 1 – 7.

Resnick, H.S., Kilpatrick, D.G., Dansky, B.S., Saunders, B.E., & Best, C.L. (1993). Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *Journal of Consulting and Clinical Psychology, 61,* 984 – 991.

Resnick, H.S., Yehuda, R., Pitman, R.K., & Foy, D.W. (1995). Effect of previous trauma on acute plasma cortisol level following rape. *American Journal of Psychiatry, 152,* 1675 – 1677.

Reynolds, M., & Brewin, C.R. (1998). Intrusive cognitions, coping strategies and emotional responses in depression, posttraumatic stress disorder and a non-clinical population. *Behavior Research and Therapy, 36,* 135 – 147.

Reynolds, M., & Brewin, C.R. (1999). Intrusive memories in depression and posttraumatic stress disorder. *Behaviour Research and Therapy, 37,* 201 – 215.

Richards, D.A., Lovell, K., & Marks, I.M. (1994). Posttraumatic stress disorder: Evaluation of a behavioral treatment program. *Journal of Traumatic Stress, 7,* 669 – 680.

Rieckert, J., & Möller, A.T. (2000). Rational-emotive behaviour therapy in the treatment of adult victims of childhood sexual abuse. *Journal of Rational-Emotive and Cognitive-Behaviour Therapy, 18*, 87 – 101.

Riggs, D.S., Byrne, C.A., Weathers, F.W., & Litz, B.T. (1998). The quality of the intimate relationships of male Vietnam veterans: Problems associated with posttraumatic stress disorder. *Journal of Traumatic Stress, 11,* 87 – 101.

Riggs, D.S., Rothbaum, B.O., & Foa, E.B. (1995). A prospective examination of symptoms of posttraumatic stress disorder in victims of non-sexual assault. *Journal of Interpersonal Violence, 10,* 201 – 214.

Robin, R.W., Chester, B., & Goldman, D. (1996). Cumulative trauma and PTSD in American-Indian communities. In A.J. Marsella, M.J. Friedman, E.T. Gerrity, & R.M. Scurfield (Eds.), *Ethnocultural aspects of post-traumatic stress disorder: Issues, research, and clinical applications.* (pp. 239 – 252). Washington, DC: American Psychological Association.

Robinson, H.M., Sigman, M.R., & Wilson, J.P. (1997). Duty-related stressors and PTSD symptoms in suburban police officers. *Psychological Report, 81,* 835 – 845.

Roemer, L., Litz, B.T., Orsillo, S.M., Ehlich, P.J., & Friedman, M.J. (1998). Increases in retrospective accounts of war-zone exposure over time: The role of PTSD symptom severity. *Journal of Traumatic Stress, 11,* 597 – 605.

Roffman, J.L., Marci, C.D., Glick, D.M., Dougherty, D.D., & Rauch, S.L. (2005). Neuroimaging and the functional neuroanatomy of psychotherapy. *Psychological Medicine, 35,* 1385 – 1398.

Roland, A. (1996). How universal is the psychoanalytic self? In R.M.P. Foster, M. Moskowitz, & R.J. Javier (Eds.), *Reaching across boundaries and class.* (pp. 71 – 90). Lanham, MD: Jason Aronson.

Rose, S., & Bisson, J. (1998). Brief early psychological interventions following trauma: A systematic review of the literature. *Journal of Traumatic Stress, 11,* 697 – 710.

Rose, S., Bisson, J., & Wessely, S. (2003). A systematic review of single-session psychological interventions ('Debriefing') following trauma. *Psychotherapy and Psychosomatics, 72,* 176 – 184.

Rose, S., Brewin, C.R., Andrews, B., & Kirk, M. (1999) A randomized controlled trial of individual psychological debriefing for victims of violent crime. *Psychological Medicine, 29,* 793 – 799.

Rosen, G.M., & Lohr, J. (1997). Can eye movements cure mental ailments? *National Council Against Health Fraud Newsletter: Quality in the Health Marketplace, 20,* 1.

Rosenman, S. (2002). Trauma and posttraumatic stress disorder in Australia: Findings in the population sample of the Australian National Survey of Mental Health and Wellbeing. *Australian and Nieu-Zeeland Journal of Psychiatry, 36,* 515 – 520.

Rosenthal, D.A., & Feldman, S.S. (1992). The nature and stability of ethnic identity in Chinese youth: Effects of length of residence in two cultural contexts. *Journal of Cross-Cultural Psychology, 23,* 214 – 227.

Roth, S., Dye, E., & Lebowitz, L. (1988). Group therapy for sexual-assault victims. *Psychotherapy, 25,* 82 – 93.

Rothbaum, B.O. (1995*). A controlled study of EMDR for PTSD.* Paper presented at the Association of Advanced Behavior Therapy, Washington, DC.

Rothbaum, B.O. (1997). A controlled study of eye movement desensitization and reprocessing in the treatment of post-traumatic stress disordered sexual assault victims. *Bulletin of the Menninger Clinic, 61,* 317 – 334.

Rothbaum, B.O., Foa, E.B., & Murdock, T. (1992). A prospective examination of post-traumatic stress disorder in rape victims. *Journal of Traumatic Stress, 5,* 455 – 475.

Rothbaum, B.O., Meadows, E.A., Resick, P.A., & Foy, D.W. (2000). Cognitive-behavioral therapy. In E.B. Foa, T.M. Keane, & M.J. Friedman, (Eds.), *Effective treatments for PTSD.* New York: Guilford Press.

Rothbaum, B.O., & Schwartz, A.C. (2002). Exposure therapy for posttraumatic stress disorder. *American Journal of Psychotherapy, 56,* 59 – 72.

Rousseau, C., Drapeau, A., & Platt, R. (1999). Family trauma and its association with emotional and behavioral problems and social adjustment in adolescent Cambodian refugees. *Child Abuse and Neglect, 23,* 1263 – 1273.

Russell, J.A. (1994). Is there universal recognition of emotion from facial expressions? A review of the cross-cultural studies. *Psychological Bulletin, 115,* 102 – 141.

Ruzek, J., & Watson, P. (2001). Early intervention to prevent PTSD and other trauma-related problems. *PTSD Research Quarterly, 12, 1 – 8.*

Sachinvala, N., Kling, A., Suffin, S., Lake, R., & Cohen, M. (2000). Increased regional cerebral perfusion by 99mTc hexamethyl propylene amine oxime single photon emission computed tomography in posttraumatic stress disorder. *Military Medicine, 165,* 473 – 479.

Sack, W.H., Clarke, G.N., & Seeley, J. (1995). Posttraumatic stress disorder across two generations of Cambodian refugees. *Journal of the American Academy of Child and Adolescent Psychiatry, 34,* 1160 – 1166.

Safer, M.A., Christianson, S., Autry, M.W., & Osterlund, K. (1998). Tunnel memory for traumatic events. *Applied Cognitive Psychology, 12,* 99 – 117.

Salkovskis, P.M., & Clarke, D.M. (1990). Affective responses to hyperventilation: A test of the cognitive model of panic. *Behaviour Research and Therapy, 28,* 51 – 61.

Sanderson, W.C., DiNardo, P.A., Rapee, R.M., & Barlow, D.H. (1990). Syndrome comorbidity in patients diagnosed with a DSM-III-R anxiety disorder. *Journal of Abnormal Psychology, 99,* 308 – 312.

Sar, V., Yargic, I., & Tutkun, H. (1996). Structured interview data on 35 cases of dissociative identity disorder in Turkey. *American Journal of Psychiatry, 153,* 1329 – 1333.

Sarraj, E.E., Punamaki, R.L., Salmi, S., & Summerfield, D. (1996). Experiences of torture and ill-treatment and posttraumatic stress disorder symptoms among Palestinian political prisoners. *Journal of Traumatic Stress, 9,* 595 – 606.

Scarvalone, P., Cloitre, M., & Difede, J. (1995). *Interpersonal process therapy for incest survivors: Preliminary outcome data.* Paper presented at the Annual Convention of the Society for Psychotherapy Research, Vancouver, British Columbia.

Scheck, M.M., Schaeffer, J.A., & Gillette, C. (1998). Brief psychological intervention with traumatized young women: The efficacy of eye movement desensitization and reprocessing. *Journal of Traumatic Stress, 11,* 25 – 44.

Scherer, K.R., & Wallbott, H.G. (1994). Evidence for universality and cultural variation of differential emotion response patterning. *Journal of Personality and Social Psychology, 66,* 310 – 328.

Schindler, F.E. (1980). Treatment by systematic desensitization of a recurring nightmare of a real life trauma. *Journal of Behavior Therapy and Experimental Psychiatry, 11,* 53 – 54.

Schlenger, W.E., Caddell, J.M., Ebert, L., Jordan, B.K., Rourke, K.M., Wilson, D., et al. (2002). Psychological reactions to terrorist attacks: Findings from the National Study of Americans' Reactions to September 11. *Journal of the American Medical Association, 288,* 581 – 588.

Schnurr, P.P., & Friedman, M.J. (1999). The National Center for PTSD: The past 10 years. *PTSD Research Quarterly, 10,* 1 – 7.

Schnurr, P.P., Friedman, M.J., & Rosenberg, S.D. (1993). Premilitary MMPI scores as predictors of combat-related PTSD symptoms. *American Journal of Psychiatry, 150,* 479 – 483.

Schnurr, P.P., Lunney, C.A., & Sengupta, A. (2004). Risk factors for the development versus maintenance of posttraumatic stress disorder. *Journal of Traumatic Stress, 17,* 85 – 95.

Schnurr, P.P., Rosenberg, S.D., & Friedman, M.J. (1993a). Change in MMPI scores from college to adulthood as a function of military service. *Journal of Abnormal Psychology, 102,* 288 – 296.

Schnurr, P.P., & Vielhauer, M.J. (1999). Personality as a risk factor for PTSD. In R. Yehuda (Ed.), *Risk Factors for Posttraumatic Stress Disorder* (pp 191 – 222). Washington, DC: American Psychiatric Association.

Schuff, N., Neylan, T.C., Lenoci, M.A., Du, A.T., Weiss, D.S., Marmar, C.R., et al. (2001). Decreased *N*-acetyl aspartate in the absence of atrophy in the hippocampus of posttraumatic stress disorder. *Biological Psychiatry, 50,* 952 –959.

Schuster, M.A., Stein, B.D., Jaycox, L.H., Collins, R.L., Marshall, G.N., Elliott, M.N., et al. (2001). A national survey of stress reactions after the September 11, 2001, terrorist attacks. *New England Journal of Medicine, 345,* 1507 – 1512.

Schwartz, J.M., Stoessel, P.W., Baxter, L.R., Martin, K.M., & Phelps, M.E. (1996). Systematic changes in cerebral glucose metabolic rate after successful behavior modification treatment of obsessive-compulsive disorder. *Archives of General Psychiatry, 53,* 109 – 113.

Schwarz, E.D., & Kowalski, J.M. (1991). Posttraumatic stress disorder after a school shooting: effects of symptom threshold selection and diagnosis by DSM-III, DSM-IIIR, or proposed DSM-IV. *American Journal of Psychiatry, 148,* 592 – 597.

Seedat, S., Warwick, J., van Heerden, B., Hugo, C., Zungu-Dirwayi, N., van Kradenburg, J., et al. (2004). Single photon emission computed tomography in posttraumatic stress disorder before and after treatment with a selective serotonin reuptake inhibitor. *Journal of Affective Disorders, 80,* 45 – 53.

Seeman, T.E., McEwen, B.S., Rowe, J.W., & Singer, B.H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences of the United States of America, 98,* 4470 – 4475.

Shalev, A.Y. (1992). Posttraumatic stress disorder among injured survivors of a terrorist attack. Predictive value of early intrusion and avoidance symptoms. *Journal of Nervous and Mental Disease, 180,* 505 – 509.

Shalev. A.Y. (2001). What is posttraumatic stress disorder? *Journal of Clinical Psychiatry, 62,* 4 – 10.

Shalev, A.Y., Freedman, S., Peri, T., Brandes, D., Sahar, T., Orr, S.P., et al. (1998). Prospective study of posttraumatic stress disorder and depression following trauma. *American Journal of Psychiatry, 155,* 630 – 637.

Shalev, A.Y., Peri, T., Canetti, L., & Schreiber. S. (1996). Predictors of PTSD in injured trauma survivors: A prospective study. *American Journal of Psychiatry, 153,* 219 – 225.

Shalev, A.Y., Yehuda, R., & McFarlane, A.C. (2000). *International Handbook of Human Response to Trauma.* New York: Kluwer/Plenum.

Shapiro, F. (1989). Efficacy of the Eye Movement Desensitization procedure in the treatment of traumatic memories. *Journal of Traumatic Stress, 2,* 199 – 223.

Shapiro, F. (1994). Alternative stimuli in the use of EMDR. *Journal of Behavior Therapy and Experimental Psychiatry, 25,* 89.

Shapiro, F. (1995). *Eye movement desensitization and reprocessing: Basic principles, protocols, and procedures.* New York: Guilford Press.

Sharan, P., Chaudary, G., Kavathekar, S.A., et al., (1996). Preliminary report of psychiatric disorders in survivors of a severe earthquake. *American Journal of Psychiatry, 153,* 556 – 558.

Sherman, J.J. (1998). Effects of psychotherapeutic treatments for PTSD: A meta-analysis of controlled clinical trials. *Journal of Traumatic Stress, 11,* 413 – 435.

Shin, L.M., Kosslyn, S.M., & McNally R.J., (1997). Visual imagery and perception in posttraumatic stress disorder: A positron emission tomography study. *Archives of General Psychiatry, 54,* 233 – 241.

Shin, L.M., Orr, S.P., Carson, M.A., Rauch, S.L., Macklin, M.L., Lasko, N.B., et al. (2004). Regional cerebral blood flow in the amygdala and medial prefrontal cortex during traumatic imagery in male and female Vietnam veterans with PTSD. *Archives of General Psychiatry, 61,* 168 – 176.

Shin, L.M., Rauch, S.L., & Pitman, R.K., (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Science, 1071,* 67 – 79.

Shore, J.H., Vollmer, W.M., & Tatum, E.L. (1989). Community patterns of posttraumatic stress disorders. *Journal of Nervous and Mental Disease, 177,* 681 – 685.

Silver, S.M., Brooks, A., & Obenchain, J. (1995). Treatment of Vietnam war veterans with PTSD: A comparison of Eye Movement Desensitization and Reprocessing, biofeedback, and relaxation training. *Journal of Traumatic Stress, 8,* 337 – 342.

Silver, R.C., Holman, E.A., McIntosh, D.N., Poulin, M., & Gil-Rivas, V. (2002). Nationwide longitudinal study of psychological responses to September 11. *Journal of the American Medical Association, 288,* 1235 – 1244.

Simpson, M. (1993). Traumatic stress and the bruising of the soul: The effects of torture and coercive interrogation. In J.P. Wilson & B. Raphael (Eds.), *International handbook of traumatic stress syndromes* (pp. 667 – 766). New York: Plenum Press.

Small, R., Lumley, J., Donohue, L., Potter, A., & Walderstrom, U. (2000). Midwife-led debriefing to reduce maternal depression following operative birth: A randomized controlled trial. *British Medical Journal, 321,* 1043 – 1047.

Solomon, Z., & Davidson, J.R.T. (1997). Trauma: Prevalence, impairment, service use, and cost. *Journal of Clinical Psychiatry, 58,* 5 – 11.

Solomon, Z., Miculincer, M., & Avitzur, E. (1988). Coping, locus of control, social support, and combat-related posttraumatic stress disorder: A prospective study. *Journal of Personality and Social Psychology, 55,* 279 – 285.

Somasundaram, D. (2001). War trauma and psychosocial problems: Patient attendees in Jaffna. *International Medical Journal, 8,* 193 – 197.

Somnier, F.E., & Genefke, I.K. (1986). Psychotherapy for victims of torture. *British Journal of Psychiatry, 149,* 323 – 329.

Sorenson, S., & Siegel, J. (1992). Gender, ethnicity, and sexual assault: Findings from a Los Angeles study. *Journal of Social Issues, 48,* 93 – 104.

Sorenson, S., & Telles, C. (1991). Self-reports of spousal violence in a Mexican American and non-Hispanic White population. *Violence and Victims, 6,* 3 – 15.

Southwick, S.M., Bremner, J.D., Rasmusson, A., Morgan, C.A., Arnsten, A., & Charney, D.S. (1999). Role of norepinephrine in the pathophysiology and treatment of posttraumatic stress disorder. *Biological Psychiatry, 46,* 1192 – 1204.

Southwick, S.M., Morgan, A., Nicolaou, A.L., & Charney D.S. (1997). Consistency of memory for combat-related traumatic events in veterans of Operation Desert Storm. *American Journal of Psychiatry, 154,* 173 – 177.

Southwick, S.M., Morgan, C.A., Vythilingam, M., Krystal, J.H., & Charney, D.S. (2003). Emerging neurobiological factors in stress resilience. *PTSD Research Quarterly, 14,* 1 – 8.

Spiegel, D. (1988). Dissociation and hypnosis in post-traumatic stress disorders. *Journal of Traumatic Stress, 1,* 17 – 33.

Spiegel, D. (1989). Hypnosis in the treatment of victims of sexual abuse. *Psychiatric Clinics of North America, 12,* 295 – 305.

Spiegel, D. (1991). Dissociation and trauma. In A. Tasman & S.M. Goldfinger (Eds.), *American psychiatric press, Review of psychiatry, 10,* 261 – 275. Washington, DC: American Psychiatric Press.

Spiro, A., Schnurr, P.P., & Aldwin, C.M. (1994). Combat-related posttraumatic stress disorder symptoms in older men. *Psychology and Aging, 9,* 17 – 26.

Squire, L.R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychology Revue, 99,* 195 – 231.

Stamm, B.H., & Friedman, M.J., (2000). Cuktural diversity in the appraisal and expression of trauma. In A.Y. Shalev, R. Yehuda, & C.A. McFarlane, (Eds.), *International handbook of human response to trauma.* New York: Kluwer/Plenum.

Steer, R.A., Beck, A.T., & Garrison, B. (1986). Applications of the Beck Depression Inventory. In N. Sartorius & T.A. Ban (Eds.), *Assessment of depression.* Geneva: World Health.

Steer, R.A., Beck, A.T., Riskind, J.H., & Brown, G. (1986). Differentiation of generalized anxiety and depression disorders by the Beck Depression Inventory. *Journal of Clinical Psychology, 42,* 475 – 478.

Steil, R., & Ehlers, A. (2000). Dysfunctional meaning of posttraumatic intrusions in chronic PTSD. *Behaviour Research and Therapy, 38,* 537 – 558.

Stein, M.B., Jang, K.L., Taylor, S., Vernon, P.A., & Livesley, W.J. (2002). Genetic and enivironmental influences on trauma exposure and posttraumatic stress disorder symptoms: A twin study. *American Journal of Psychiatry, 159,* 1675 – 1681.

Stein, M.B., & Kennedy, C. (2001). Major depressive and posttraumatic stress disorder comorbidity in female victims of intimate partner violence. *Journal of Affective Disorders, 66,* 133 – 138.

Stein, M.B., Kennedy, C.M., & Twamley, E.W. (2002). Neuropsychological function in female victims of intimate partner violence with and without posttraumatic stress disorder. *Biological Psychiatry, 52,* 1079 – 1088.

Stein, M.B., Koverola, C., Hanna, C., Torchia, M.G., & McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine, 27,* 951 – 959.

Stein, M.B., McQuaid, J.R., Pedrelli, P., Lenox, R.J., & McCahill, M.E. (2000). Posttraumatic stress disorder in the primary care medical setting. *General Hospital Psychiatry, 22,* 261 – 269.

Stein, M.B., Walker, J.R., Hazen, A.L., & Forde, D.R. (1997). Full and partial posttraumatic stress disorder: Findings from a community survey. *American Journal of Psychiatry, 154,* 1114 – 1119.

Struwig, F.W., & Stead, G.B. (2001). *Planning, designing, and reporting research.* Cape Town: Maskew Miller Longman.

Summerfield, D. (1999). A critique of seven assumptions behind psychological trauma programmes in war-affected areas. *Social Science and Medicine, 48,* 1449 – 1462.

Swartz, L. (1998). *Culture and mental health: A Southern African perspective.* Cape Town: Oxford University Press.

Taylor, S., Thordarson, D.S., Maxfield, L., Fedoroff, I.C., Lovell, K., & Ogrodniczuk, J. (2003). Comparative efficacy, speed, and adverse effects of three PTSD treatments: Exposure therapy, EMDR, and relaxation training. *Journal of Consulting and Clinical Psychology, 771,* 330 – 338.

Terr, L.C. (1991). Childhood traumas: An outline and overview. *American Journal of Psychiatry, 148,* 10 – 20.

Thompson, J.A., Charlton, P.F.C., Kerry, R., Lee, D., & Turner, S.W. (1995). An open trial of exposure therapy based on deconditioning for post-traumatic stress disorder. *British Journal of Clinical Psychiatry, 34,* 407 – 416.

Tichenor, V., Marmar, C.R., Weiss, D.S., Metzler, T.J., & Ronfeldt, H.M. (1996). The relationship of peritraumatic dissociation and posttraumatic stress: Findings in female Vietnam theatre veterans. *Journal of Consulting and Clinical Psychology, 64,* 1054 – 1059.

True, W.R., Rice, J., Eisen, S.A., Heath, A.C., Goldberg, J., Lyons, M.J., et al. (1993). A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Archives of General Psychiatry, 50,* 257 – 264.

Vaisrub, S. (1975). Da Costa syndrome revisited. *Journal of the American Medical Association, 232,* 164.

Van der Hart, O., Nijenhuis, E.R.S., & Steele, K. (2005). Dissociation: An insufficiently recognized major feature of complex posttraumatic stress disorder. *Journal of Traumatic Stress, 1*8, 413 – 423.

Van der Kolk, B.A., & Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories: Overview and exploratory study. *Journal of Traumatic Stress, 8,* 505 – 525.

Van der Kolk, B.A., & Van der Hart, O. (1991). The intrusive past: The flexibility of memory and the engraving of trauma. *American Imago, 48,* 425 – 454.

Van Etten, M.L., & Taylor, S. (1998). Comparative efficacy of treatments for posttraumatic stress disorder: A meta-analysis. *Clinical Psychology and Psychotherapy, 5,* 126 – 144.

Van Niekerk, J.F., Möller, A.T., & Nortje, C. (1999). Self-schemas in social phobia and panic disorder. *Psychological Reports, 84,* 843 – 854.

Van Steenwegen, D., Vervliet, B., Hermans, D., Beckers, T., Baeyens, F., & Eelen, P. (2006). Stronger renewal in human fear conditioning when tested with an acquisition retrieval cue than with an extinction retrieval cue. *Behavioral Research and Therapy, 44,* 1717 – 1725.

Van Zelst, W.H., De Beurs, E., Beekman, A.T.F., Deeg, D.J.H., & Van Dyck, R. (2003). Prevalence and risk factors of posttraumatic stress disorder in older adults. *Psychotherapy and Psychosomatics 72,* 333 – 342.

Vasquez-Nuttal, E., Romero-Garcia, I., & De Leon, B. (1987). Sex roles and perceptions of feminity and masculinity of Hispanic women: A review of the literature. *Psychology of Women Quarterly, 11,* 409 – 425.

Vasterling, J.J., Duke, L.M., Brailey, K., Constans, J.I., Allain, A.N. Jnr., & Sutker, P.B. (2002). Attention, learning, and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparisons. *Neuropsychology, 16,* 5 – 14.

Vaughan, K., Armstrong, M.S., Gold, R., O'Connor, N., Jenneke, W., & Tarrier, N. (1994). A trial of eye movement desensitization compared to image habituation training and applied muscle relaxation in post-traumatic stress disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 25,* 283 – 291.

Veronen, L.J., & Kilpatrick, D.G. (1982). *Stress inocculation training for victims of rape: Efficacy and differential findings.* Paper presented at the 16th Annual Convention of the Association for Behavior Therapy, Los Angeles, CA.

Vlahov, D., Galea, S., Resnick, H., Ahern, J., Boscarino, J.A., Bucuvalas, M., et al. (2002). Increased use of cigarettes, alcohol, and marijuana among Manhattan residents after the September 11th terrorist attacks. *American Journal of Epidemiology, 155,* 988 – 996.

Walker, J.J., & Nash, J.D. (1981). Group therapy in the treatment of Vietnam combat veterans. *International Journal of Group Psychotherapy, 31,* 379 – 389.

Wang, X., Gao, L., & Shinfuku, N. (2000). Longitudinal study of earthquake-related PTSD in a randomly selected community sample in North China. *American Journal of Psychiatry, 157,* 1260 – 1266.

Ward, G., & Carroll, M. (1997). Reality monitoring for sexual abuse memories. *Applied Cognitive Psychology, 11,* 293 – 304.

Warshaw, M.G., Fierman, E., & Pratt, L., (1993). Quality of life and dissociation in anxiety disorder patients with histories of trauma or PTSD. *American Journal of Psychiatry, 150,* 1512 – 1516.

Weathers, F.W., Blake, D.D., Krinsley, K.E., Haddad, W., Huska, J.A., & Keane, T.M. (1992. *The clinician-administered PTSD scale (CAPS): Reliability and construct validity.* Paper presented at the meeting of the Association for the Advancement of Behaviour Therapy. Boston, MA.

Weathers, F.W., Keane, T.M., & Davidson, J.R. (2001). Clinician-administered PTSD scale: A review of the first ten years of research. *Depression and Anxiety, 13,* 132 – 156.

Weathers, F.W., Ruscio, A.M., & Keane, T.M. (1999). Psychmetric properties of nine scoring rules for the clinician-administered posttraumatic stress disorder scale. *Psychological Assessment, 11,* 124 – 133.

Werner, P.D. (2001). Structured clinical interview for DSM-IV axis I disorders: Clinician version. In B.S. Plake & J.C. Impara (Eds.), The fourteenth mental measurements yearbook (pp. 1123 – 1125). Lincoln: NE: Buros Institute of Mental Measurements.

Westermeyer, J., Bouafuely, M., Neider, J., & Callies, A. (1989). Somatization among refugees: An epidemiological studey. *Psychosomatics, 30,* 34 - 43

Whisman, M. (1999). Marital dissatisfaction and psychiatric disorders: Results from the National Comorbidity Survey. *Journal of Abnormal Psychology, 108,* 701 – 706.

Wilson, D.L., Silver, S.M., Covi, W.G., & Foster, S. (1996). Eye movement desensitization and reprocessing: Effectiveness and autonomic correlates. *Journal of Behavior Therapy and Experimental Psychiatry, 27,* 219 – 229.

Wilson, S.A., Becker, L.A., & Tinker, R.H. (1995). Eye movement desensitization and reprocessing (EMDR) treatment for psychologically traumatized individuals. *Journal of Consulting and Clinical Psychology, 63,* 928 – 937.

Wilson, S.A., Becker, L.A., & Tinker, R.H. (1997). Fifteen month follow-up of eye movement desensitization and reprocessing (EMDR) treatment for posttraumatic stress disorder and psychological trauma. *Journal of Consulting and Clinical Psychology. 65,* 1047 – 1056.

Wolfe, J., & Kimerling, R. (1997). Gender issues in the assessment of posttraumatic stress disorder. In J. Wilson & T. Keane (Eds.), *Assessing psychological trauma and PTSD* (pp. 192 – 238). New York: Guilford Press.

Wolff, R. (1977). Systematic desensitization and negative practice to alter the after-effects of a rape attempt. *Journal of Behavior Therapy and Experimental Psychiatry, 8,* 423 – 425.

Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition.* Stanford: Stanford University Press.

Wyshak, G. (1994). The relation between change in reports of traumatic events and symptoms of psychiatric distress. *General Hospital Psychiatry, 16,* 290 – 297.

Yehuda, R. (2003). Adult neuroendocrine aspects of PTSD. *Psychiatric Annals, 33,* 30 – 36.

Yehuda, R., Kahana, B., Schmeidler, J., Southwick, S.M., Wilson, S., & Giller, E.L. (1995). Impact of cumulative lifetime trauma and recent stress on current posttraumatic stress disorder symptoms in Holocaust survivors. *American Journal of Psychiatry, 152,* 1815 – 1818.

Yehuda, R., & McFarlane, A.C. (1995). Conflict between current knowledge about posttraumatic stress disorder and its original conceptual base. *American Journal of Psychiatry, 152,* 1705 – 1713.

Yehuda, R., McFarlane, A.C., & Shalev, A.Y. (1998). Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. *Biological Psychiatry, 44,* 1305 – 1313.

Yehuda, R., Teicher, M.H., Trestman, R.L., Levengood, R.A., & Siever, L.J. (1996). Cortisol regulation in posttraumatic stress disorder and major depression: A chronobiological analysis. *Biological Psychiatry, 40,* 79 – 88.

Yule, W. (2000). Treatment of PTSD in children following RTAs. In E. Blanchard, & E. Hickling, (Eds.), *Road accidents and the mind.* (pp. 375 – 387). London: Elsevier.

Yule, W., Udwin, O., & Murdock, K. (1990). The "Jupiter"sinking: Effects on children's fears, depression and anxiety. *Journal of Child Psychology and Psychiatry, 31,* 1051 – 1061.

Zaidi, L.Y. (1994). Group treatment for adult male inpatients abused as children. *Journal of Traumatic Stress, 7,* 719 – 727.

Zatzick, D.F., Marmar, C.R., Weiss, D.R., Browner, W.S., Metzler, T.J., Golding, J.M., et al. (1997). Posttraumatic stress disorder and functioning and quality of life outcomes in a nationally representative sample of male Vietnam veterans. *American Journal of Psychiatry, 154,* 1690 – 1695.

Zatzick, D.F., Weiss, D.S., Marmar, C.R., Metzler, T.J., Wells, K.B., Golding, J.M., et al. (1997). Post-traumatic stress disorder and functioning and quality of life outcomes in female Vietnam veterans. *Military Medicine, 162,* 661 – 665.

Zayfert, C., Becker, C.B., Unger, D.L., & Shearer, D.K. (2002). Comorbid anxiety disorders in civilians seeking treatment for posttraumatic stress disorder. *Journal of Traumatic Stress, 15,* 31 – 38.

Zoellner, L.A., Alvarez-Conrad, J., & Foa, E.B. (2002). Peritraumatic dissociative experiences, trauma narratives, and trauma pathology. *Journal of Traumatic Stress, 15(1),* 49 – 57.