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Neurocognition in

Post-Traumatic Stress Disorder

By

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Abstract

The negative behavioural and emotional symptoms of Post-Traumatic Stress Disorder (PTSD) have been extensively reported in the literature. However, much less is known about the neuropsychological and neurobiological characteristics of the disorder.

This thesis consists of two papers, the first being a review which highlights the emerging picture of literature in the field of neuropsychology in PTSD, with particular reference to findings in those cognitive domains of general intellectual functioning, memory, attention and executive function.

Given that the findings associated within these domains are mixed, the second paper reports the outcome from a neuropsychological study of cognitive differences that was conducted to contribute to current knowledge in the area of neurocognition and visual memory in PTSD in particular. Trauma exposure, current PTSD, depressive and anxiety symptoms and performance on a range of neuropsychological tests were examined in tertiary care outpatients with PTSD (n=26), individuals who had been exposed to severe trauma but without current PTSD (n=26), and healthy controls (n=26).

In addition to previously reported deficits in verbal learning and fluency in PTSD, deficits in visual spatial memory were also found. These observable deficits in visual memory may reflect characteristic features of PTSD, such as reported difficulties in remembering certain aspects of traumatic events and the presence of visual flashbacks. It is uncertain whether these deficits represent a risk factor for PTSD, or a consequence of trauma, as suggested by research in animal models.

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Table of Contents

Part One: Literature Review Page Number

Page Number

Part Two: Empirical Paper

Literature Review

The Neuropsychology of Post-Traumatic Stress Disorder

Part One

This paper has been prepared for submission to Clinical Psychology Review and written in accordance with American Psychological Association guidelines

Running head: The Neuropsychology of Post-Traumatic Stress Disorder

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Abstract

The aim of this paper is to review the literature on the neuropsychology of Post-Traumatic Stress Disorder (PTSD) with relevance to neurocognitive literature on findings of impairment for neutral stimuli.

In order to provide a context within which the current literature can be considered, this review begins with a brief description of diagnostic criteria and the prevalence and co-morbidity of PTSD. Two key cognitive theories of PTSD are discussed and the success these models have in explaining PTSD symptomatology from empirical evidence is evaluated.

The factors influencing the development of PTSD including the role of intellectual functioning, autobiographical memory and, hippocampal and amygdala functioning is briefly noted. This is followed by the neuropsychological sequelae of trauma, specifically with reference to memory, attention and executive function. Lastly, clinical considerations and the link between PTSD and cognitive impairment as a consequence or cause are discussed before a final summary and conclusion.

1. Introduction

Since it first appeared as a formal diagnostic category in 1980, Post-Traumatic Stress Disorder (PTSD) has generated a vast amount of research that has highlighted the numerous consequences of trauma exposure. It has become progressively more apparent that in addition to negative emotional and behavioural symptoms, PTSD is also associated with deficits in neurobiological systems and some cognitive domains.

The aim of this paper is to review the current understanding of PTSD with respect to neuropsychology. For example, what aspects are known about and what is established as well as those issues that are unclear and unresolved. To achieve this, a brief description of PTSD is necessary. As such, the first section commences with a short introduction to PTSD including diagnostic criteria, prevalence data and co-morbidity with other disorders. The review will then describe how two specific psychological models account for PTSD and the role of risk, vulnerability and protective factors in the disorder with particular relevance to neuropsychological factors. Finally, we will consider what evidence there is for general impairment in cognitive domains in PTSD and what findings are inconclusive and, or, under researched. The focus here will be very much on neuropsychological tests findings from neutral stimuli and given the vast topic, this review is not intended to be exhaustive but rather highlight pertinent literature in the area.

The literature search criteria for this paper included accessing the following electronic databases: Psychinfo, Ovid, PubMed, Cochrane, Scopus, Informaworld and Sciencedirect. Keywords included in the search (both individually and in combination) were ‘cognitive’, ‘neuropsychological’, ‘memory’, ‘verbal memory’, ‘visual memory’, ‘attention’, ‘executive function’, ‘visual spatial memory’ and ‘visuo-construction memory’ each of them combined with ‘posttraumatic stress disorder’, ‘PTSD’, ‘trauma’, ‘traumatic stress’ and ‘post-traumatic stress’. In addition, reference lists from the identified articles were explored. The final articles were selected if they were written and published in the English language.

2. Post-Traumatic Stress Disorder

This section aims to describe the disorder and outline diagnostic criteria in relation to neurocognitive aspects of PTSD. Prevalence data will be highlighted and co-morbidity discussed.

2.1. Background.

Over the last few decades, great advances have been made in our understanding of the stress reactions following a traumatic event. However, a number of issues remain unclear. For example, why some individuals experience a number of behavioural, cognitive, emotional and neuropsychological difficulties and yet others do not. Investigators considering why this should be the case have tried to predict those at increased risk and articulate the processing involved in the development and maintenance of PTSD. In addition much research has been undertaken to address the neurobiological and neuropsychological sequelae of trauma.

Among the most studied populations are those that have experienced military combat, road traffic accidents or adult survivors of childhood sexual abuse. Consequently, significant advances have been made in our understanding of stress reactions and PTSD in adults, and the implications of these reactions in terms of behaviour and cognitive problems. To coincide with this the diagnostic criteria for PTSD has also evolved.

2.2. Diagnostic criteria of PTSD.

The Diagnostic and Statistical Manual DSM-IV-TR (APA, 2000) lists a number of detailed criteria that need to be present in order for a diagnosis of PTSD to be made. Those criteria particularly relevant to neuropsychology include three core constellations of re-experiencing the event in the form of nightmares, intrusive thoughts, hallucinations, images or flashbacks; persistent avoidance of stimuli associated with the traumatic event accompanied by feelings of detachment, psychogenic amnesia and

increased arousal, hypervigilance, difficulty concentrating and exaggerated startle response. Some individuals may not fulfil the total criteria specified in DSM-IV-TR (APA, 2000) for a diagnosis of PTSD but nonetheless suffer distressing symptoms. In this event, the patient is considered to have 'subsyndromal' PTSD (Blanchard & Hickling, 2004)

2.3. Prevalence of PTSD.

Although trauma is pivotal for PTSD aetiology not everyone who experiences a traumatic event goes on to develop the disorder. The estimated prevalence rates vary widely within the stringency of criteria applied and appear dependent on population and traumatic event. For example, in studies of specific groups who have experienced trauma, rates of PTSD range from 2.8% (Rosenman, 2002) to 25-30% (NICE, 2005).

Overall lifetime prevalence has been estimated at 7.8% (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995) with females more likely to develop symptoms than males in a ratio of 2:1 (Breslau, 2001). Exactly why this should be the case is uncertain and is despite men reporting greater trauma exposure (Frans, Rimmo, Berg, & Fredrikson, 2005). One general explanation proposed by Bryant and Harvey (2003) is a gender specific response bias or biological difference although gender alone is not considered to be a reliable predictor (Rosenman, 2002). The fact that not all individuals develop PTSD after trauma exposure and the differences in prevalence rates indicates that some individuals are more prone to develop PTSD than others.

2.4. Co-morbidity with other disorders.

Rates of co-morbidity in populations with PTSD are high. Kessler et al. (1995) reported that 88% of men and 79% of women with PTSD met the criteria for at least one other psychiatric diagnosis. The most common co-morbid diagnoses reported are depressive and anxiety disorders, substance misuse, and phobias (Jacobsen, Southwick, & Kosten, 2001; Kessler et al., 1995). It has been suggested that high co-

morbidity may result from wide use of DSM criteria that encourages multiple diagnoses or that perhaps, particularly in the case of depression, by the shared overlap in symptom measurement (Shalev, 2001).

As well as high co-morbidity, the impact of PTSD is also linked with altered cognitive functioning such as alterations in memory, attention, dissociation, affect and beliefs (Brewin & Holmes, 2003) in addition to social consequences (Kimble, Riggs, & Keane, 1999). Although some individuals recover spontaneously (Koren, Arnon, & Klein, 2001) others may suffer for many years, with lifespan difficulties including physical and mental health problems, and memory difficulties reported up to 40 years after the trauma (Bichescu, Schauer, Saleptsi, Neculau, Elbert, & Neuner, 2005).

As PTSD is associated with high prevalence and high co-morbidity rates it is of great importance to understand the disorder to better prevent and treat it. As such we now turn to consider prominent psychological theories of PTSD and evaluate how successful they are in accounting for the numerous neurocognitive features of PTSD that require explanation. Particular attention will be given to the disorganisation and incompleteness of the trauma memory; intrusive memories and the psychogenic amnesia that typically surround the event; dissociative responses and spontaneous flashbacks during which time there may be a strain on the cognitive capacity that can result in a reduced engagement with the present environment (Brewin, 2001), and excessive response to reminders of the trauma such as exaggerated startle response

3. Theoretical Models of PTSD

A number of theories of PTSD exist that attempt to account for the range of symptomatology detailed previously, while also explaining individual differences in response to trauma, for example psychobiological theories (Van der Kolk, 1988). However, it is the cognitive and cognitive science theories, implicated in reviews by Brewin (2001), Brewin and Holmes (2003), and Ozer, Best, Lipsey, and Wiess (2003) which can account for a greater degree of variation than behavioural, psychodynamic or

learning theories and can also be noticeably linked with prevalent treatment models used within clinical psychology practice.

Although, in the last two decades, there have been a number of cognitive theories that have expanded our knowledge of the psychology, epidemiology and biology of PTSD the focus here is on two recent theories; the cognitive model proposed by Ehlers and Clark (2000) and the Dual Representation Theory (DRT; Brewin, Dalgleish, & Joseph, 1996), together with the neuroscience update to the DRT (Brewin, 2001; 2008). These have been selected as they represent key developments in the understanding of PTSD and highlight two key neurocognitive areas namely memory and appraisal processes.

3.1. Ehlers and Clark's Cognitive Model.

In their cognitive model, Ehlers and Clark propose that individuals process the trauma in a way that causes an ongoing current serious threat that fits with the conceptualization of PTSD within DSM-IV-TR (APA, 2000) as an anxiety disorder. Ehlers and Clark identify two features that produce threat information. Firstly, negative appraisals of the trauma experience and secondly a disturbance in autobiographical memory leading to involuntary re-experiencing of the trauma.

Drawing on aspects of Foa and Rothbaum's (1998) emotional processing theory, Ehlers and Clark suggest that negative appraisals can be focussed on numerous areas, such as the traumatic event, PTSD symptoms such as numbing, and the individual's own future prospects. This variability in type of threat appraisal can lead to the variety of different emotional outcomes reported in PTSD (Ehlers & Clark, 2000).

A core process in this model is the unique character of the traumatic memory i.e., the inconsistency of trauma memory where individuals are unable to intentionally access information about some aspects, while experiencing involuntary memories in the form of intrusions and flashbacks. Ehlers and Clark suggest that a trauma memory is vivid and emotionally upsetting because it is fragmented (i.e., flashbulb). To the patient,

this memory feels like it is in the present and not like a memory of the past. The sense of current threat is a novel feature of this model and this element, along with some dysfunctional cognitions, enforces a sense of present and serious threat for the individual which provides evidence of the link to intrusive memories in PTSD (Ehlers, Hackmann, & Michael, 2004) and accounts for the maladaptive behaviours and the cognitive processing style that maintain PTSD.

3.1.2. Empirical evidence for the cognitive model.

With an emphasis on the organisation of memory, this theory is able to offer a good account of the development and maintenance of PTSD. Many features of the theory have been empirically supported, such as the appraisal component (Fairbrother & Rachman, 2006), peri-traumatic dissociation (Halligan, Michael, Clark, & Ehlers, 2003), different forms of processing and negative appraisals of the trauma (see Brewin & Holmes, 2003; Ehlers, Clark, Hackmann, McManus, & Fennell, 2005). However, the model fails to account for the exact reasons why the trauma memory is perceived in the present tense and not in the past. Another model that may account for this memory deficit was proposed by Brewin et al. (1996) Dual Representation Theory.

3.2. Dual Representation Theory (DRT; Brewin et al., 1996; Brewin, 2001; 2008).

The DRT (Brewin et al.,1996; Brewin 2001; 2008) is perhaps one of the most influential multi-level theories of PTSD. It builds on previous social cognitive (Horowitz, 1976; Janoff-Bulman, 1992) and information processing (Foa, Steketee, & Rothbaum, 1989) approaches and discriminates between two parallel memory systems. The first, a conscious experience of the trauma, is stored as verbally accessible memories (VAMs) and contains sensory features of the trauma, physiological and emotional reactions, and the perceived meaning of the event (Brewin et al., 1996).

Such memories may be detailed but highly selective. Where the traumatic event is overwhelming, the VAM is not sufficiently processed and repeated intrusive memories

can occur provoking strong emotional responses such as guilt and anger (Brewin et al., 1996).

The non-conscious processing that takes place during the trauma is proposed to be more extensive and results in the second memory type, situationally accessible memories (SAMs). These are automatically accessible through situational cues and store sensory, physiological and motor aspects of the trauma, and are problematic to regulate because the individual cannot always control their exposure to smells, sounds or sights that remind them of the trauma. SAMs contain information obtained from lower level perceptual processing of the trauma (e.g., minimally consciously processed visuo-spatial information) that causes arousal, flashbacks and, subsequent avoidance behaviour (Brewin et al., 1996).

The theory proposes that emotional memory processing occurs in an effort to reduce the negative secondary effects of trauma memory and essentially, the possible conflict between SAMs and VAMS. The authors suggest three endpoints of processing; the first being completion or integration, where discrepancies are resolved; the second is where the individual experiences heightened arousal, attention and memory problems, avoidance, possible depression and anxiety; lastly, emotional processing could lead to a premature inhibition of further processing with high levels of avoidance. This can account for late onset of PTSD as discrepancies are suppressed and not resolved.

3.2.1. Empirical Evidence for DRT.

Evidence from a variety of neuropsychology and cognitive literature supports the existence of separate memory systems involved in information processing pre and post trauma (Brewin, 2001; Brewin & Holmes, 2003). In addition, this theory is successful in accounting for a number of mechanisms involved and the range of phenomena associated with PTSD (Brewin et al., 1996).

Although DRT theory represents a step forward in our knowledge of trauma memories, it does not suggest how these memories are represented, whether VAMs and SAMs are found in normal memory, or account for pre-trauma risk factors, or post-trauma appraisal (Dalgleish, 1999). Pre-trauma risk factors are not integrated into the theory and the architecture of the two memory systems and their relation to existing memory systems is not clear (Dalgleish, 2004). Furthermore, it is not specific about whether trauma memory is perceived as being in the past, and does not relate this to the memory deficit observed in PTSD.

A complementary approach proposed by Brewin (2001) has been to consider the biological system underlying memory formation and has focused on two related areas. Firstly, the functioning of memory related brain structures under extreme stress and secondly, the role of neurohormones released in response to traumatic stress. The release of these stress hormones during exposure to trauma negatively affects the processing of the VAM because of their effect on the hippocampus.

Numerous authors (e.g., McNally, 1997; Shin, Rauch, & Pitman, 2005) have proposed that traumatic memories result from a disruption of hippocampal processing coupled with relatively high amygdaloid processing at the time of the traumatic event. Information that is processed in the amygdala during intense trauma may make poorly organised, indelible and highly fragmented memories. Brewin proposed that the information memory deficits at a time of extremely high levels of fear increase the likelihood of amygdala activation to subsequent reminders of the trauma and contribute to a sense of current threat (Brewin, 2001).

Brewin (2001) further posits that re-experiencing transfers memory information from SAM (which is not processed via the hippocampus) to VAM. The sensory memory of flashbacks can be recoded through focussed attention providing context of time and place and reducing the sense of current threat (Brewin, 2001). However, SAM's remain available to be activated at any time and without VAM representation to inhibit

amygdala activation, leaves the individual vulnerable to delayed onset PTSD for years (Brewin, 2001).

3.3. Summary of strengths and limitations of the theories.

Ehlers and Clark's cognitive model and the DRT overlap in that they both explain the persistence of PTSD, avoidance and re-experiencing phenomena and both assume that PTSD is the consequence of inefficient processing and, or, coping with a traumatic event. However, each model also presents its own conceptual difficulties and is unable to give a detailed and thorough account of why this processing is inefficient. Perhaps it is the role of numerous pre, peri- and post-traumatic factors that contribute to the development and maintenance of the disorder. The following section will focus on those risk vulnerability factors and individual differences that may contribute to and maintain the disorder considered most relevant to the topic area of this paper.

4. Factors influencing the development of PTSD

In a meta-analytic review Ozer et al. (2003) grouped risk factors of PTSD into three areas; historical and personal characteristics, trauma characteristics, and post trauma experience. Historical and personal characteristics include previous psychiatric history, previous experience of trauma, and gender. However, the findings for these areas have been modest or inconclusive with only small effect sizes revealed for personality, prior trauma and gender differences (Brewin, Andrews, & Valentine, 2000; Ozer et al., 2003). Although there is some degree of support for gender differences in that women present with higher rates of PTSD than men (Norris, Foster, & Weissharr, 2002) this has not translated into gender being recognised as a reliable predictor of who may ultimately develop PTSD after trauma (Rosenman, 2002).

Other characteristics pertinent to the neuropsychology of PTSD are reports that neurological soft signs; for example, subtle abnormalities of motor co-ordination, language and perception, including childhood attention deficit problems (Gurvits et al.,

2000), lower intellectual functioning (Vasterling, Duke, Brailey, Constans, Allain, & Sutker, (2002) and, or, a history of head injury (Mollica, Henderson, & Tor, 2002) may all moderate the relationship between trauma exposure and the development of PTSD.

According to Fairbrother and Rachman (2006) objective trauma severity has not been shown to correlate with symptom severity, highlighting the possible role of cognitive processes. Furthermore, rates of PTSD are known to differ according to trauma type as illustrated by one study that stated that accident victims reported less PTSD symptoms than victims of crime (Tarrier, Sommerfield, Pilgrim, & Faragher, 2000).

Other post trauma aspects such as social and personal resilience factors are also believed to present predictive factors (Gold, Engdahl, Eberly, Blake, Page, & Frueh, 2000; Holeva, Tarrier, & Wells, 2001). In general, it seems that acts directed at the individual generally result in greater problems with PTSD than natural disasters or impersonal events. This would suggest that factors related to psychological processes in the peri- and post-traumatic stage have a greater impact than stable prior characteristics such as prior trauma or history of psychopathology. However, it remains unclear how the numerous factors mentioned here may interrelate.

4.1. The role of intellectual functioning.

For some time there has been a question whether intellectual function was a risk factor for PTSD or if higher intelligence was a resilience factor (McNally, 2006). Few studies have measured the comprehensive and multiple aspects of Intelligence Quotient (IQ) sufficiently to make conclusions on the aspects of intelligence that may be impaired in PTSD. Those studies that have undertaken IQ assessments have suggested that PTSD diagnosis is related to lower estimated omnibus IQ scores (Brandes , Ben-Schachter, Gilboa, Bonne, Freedman, Shalev, 2002; Gurvits, Lasko, Schachter, Kuhne, Orr, & Pitman, 1993; Vasterling et al., 2002). In particular, a number of studies have suggested that global verbal dimensions of intellectual performance as measured by

standardised IQ tests, appear particularly sensitive in those diagnosed with PTSD (e.g., Gil, Calev, Greenburg, Kugelmass, & Lerer, 1990; Vasterling, Brailey, Constans, Borges, & Sutker, 1997). It should be noted that verbal IQ is strongly correlated to verbal memory (Cooper, 1995) and that certain neuropsychological tests are considered to measure 'pure' verbal short term memory (e.g., the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 1987). Whereas other tests with varying subdivisions are considered to measure verbal IQ as they also assess vocabulary, comprehension and information (e.g., the Wechsler Adult Intelligence Scale, WAIS-R; Wechsler, 1987). Most of the populations used in the studies mentioned previously have been war veterans, and it could be argued that those with lower levels of education were allocated to more combat related duties entailing a greater trauma exposure risk than those with a more formal education assigned to less combatant roles (Vasterling & Brailey 2005) limiting the conclusions that can be drawn regarding IQ as a risk factor or a resilience factor.

Other recent research has supported the assertion that lower pre-trauma intelligence increases the risk for developing PTSD symptoms, not that PTSD lowers performance on general intelligence tests (Breslau, Lucia, & Alvarado, 2006), therefore suggesting that higher IQ is a protective factor. For example, Gilbertson and colleagues (Gilbertson et al., 2006) conducted a comprehensive study on monozygotic twins discordant for combat exposure and concluded that full-scale IQ (as measured by the WAIS-R) represents a familial predisposition factor. However, co-morbid conditions may have contributed to these findings in that Gilbertson's sample demonstrated a heightened alcohol abuse history and the unique nature of the participants meant only a small sample was recruited (N = 19 monozygotic twins). Furthermore, premorbid risk factors can partially be accounted for by the possibility of a shared environment among brothers, including in utero environment, sociodemographic factors such as, lack of education, poverty, adverse parenting and disrupted families with more risk for traumatisation (Fairbank, Putnam, & Harris, 2007).

4.1.2. Autobiographical memory.

There have long been reports of a connection between low mood and memory recall in certain groups of patients (e.g., those suffering depression) and their tendency to produce 'overgeneral' autobiographical memories (for a review see Williams, 1996). Overgeneral autobiographical memory relates to a description of a category of events rather than to one particular personal memory episode from the past in response to a specific neutral cue word. So for example, Constans (2005) suggested when using the cue word 'candle' an individual with depression may respond with 'birthday parties' rather than linking the memory to one particularly birthday party.

People exposed to trauma, especially those with PTSD are frequently characterised by poor and overgeneral autobiographical memory (Harvey, Bryant, & Dang, 1998; McNally, Lasko, Macklin, & Pitman, 1995). The reasons why individuals with PTSD experience overgeneral memories is uncertain. Some have suggested that reduced autobiographical memory specificity might be used as a way of regulating affect (Hermans, Defranc, Raes, Williams, & Eelen, 2005). It could also be due to co-morbid depression but others have speculated that it may be related to an avoidant coping style or trauma history exposure (van Breeswijk & de Wilde, 2004). Specifically concerning trauma exposure, Williams (1996) proported that individuals with a history of negative life events (particularly in childhood), learn to regulate their mood by avoiding the negative emotions associated with the trauma. The continual use of this strategy makes retrieval process for personal memories less specified. In line with Williams' hypothesis, Brewin, Reynolds, and Tata (1999), reported that overgeneral memory bias was related to the extent of avoidance and intrusion of stressful memories. However, wider evidence to further support these findings is more mixed (Reas, Hermans, Decker, Williams, & Eelen, 2003).

4.1.3. Hippocampal and amygdala functioning.

Whilst difficulty retrieving specific autobiographical memories may reflect a strategy to regulate the severe distress provoked by the trauma memories retrieved, it might also indicate compromised structural and functional neuroanatomy and neurophysiology, particularly in the functioning of the hippocampus.

During threat situations, activity in numerous systems of the body allows the individual to assess and respond to the situation appropriately providing a protective mechanism that facilitates the 'flight or fight' response. However, in some individuals neurobiological responses to stress and fear may be maladaptive and contribute to PTSD (Southwick, Yehuda, & Morgan, 1995). Studies looking at the role of different anatomical memory systems in PTSD have mainly focused on the amygdala and the hippocampus. The amygdala is believed to be a subcortical, non-conscious system fundamental to the formation of emotional memories and threat related stimuli and having a critical role in the development of conditioned fear (Davis & Whalen, 2001). In contrast the hippocampus is important in contextual learning and involved with explicit memory recall, memory for episodic events (Wheeler & Buckner, 2004) and working memory (Squire, 1982).

The hippocampus is also responsible for the regulation of stress response, (Jacobson & Sapolsky, 1991) and animal studies in which stress was induced have indicated that stress exposure may lead to both behavioural changes and neurobiological alterations, including performance decrements on learning and memory tasks suggesting that chronic stress may affect the hippocampus through the excess release of neurohormones (Sapolsky, Armanini, Packan, Sutton, & Plotsky, 1990).

Findings from meta-analytic reviews (e.g., Karl, Schaefer, Malta, Dörfel, Rohleder, & Werner, 2006) indicate that trauma exposed adults with and without PTSD exhibit smaller hippocampal volume relative to control samples and adults with PTSD also exhibited significantly smaller anterior cingulate cortex, and smaller left amygdala when compared to non trauma exposed controls. Karl and colleagues concluded that these

findings suggest that hippocampal volumetric differences may be related to PTSD severity and that PTSD itself is associated with abnormalities in multiple frontal-limbic system structures (Karl et al., 2006).

In terms of risk or resilience factors, it may be that neurobiological pathways, via the central nervous system, present a vulnerability to stress in some individuals (Morgan et al., 2000). Support for this is provided by Gilbertson et al. (2002) who reported smaller hippocampi in non-exposed twins of PTSD patients suggesting a familial vulnerability factor rather than a product of the trauma exposure. It is clear, however, that a great deal of work remains to tease apart the relationship between neuroanatomical functioning and psychological symtomatology in PTSD.

5. Summary

Exposure to traumatic events is common. However, not all those who experience a trauma develop PTSD and it is essential to distinguish a normal response to a traumatic event from ongoing difficulties. Furthermore, PTSD remains somewhat of an anomaly amongst psychiatric disorders in that it requires a specific external trigger and is concerned with past, rather than future events. The impact of PTSD is considerable, high prevalence rates and potential long term suffering of those with PTSD justifies significant research attention.

Recent meta-analytic reviews have summarized the role of various predictive factors and established how pre-, peri- and post-trauma aspects may be especially important. This has treatment implications for the increasing number of patients who present to mental health services. Effective clinical treatment for PTSD requires a theoretical framework that incorporates and accounts for the range of problems implicated in PTSD to enhance our understanding and inform interventions.

We can make speculative distinctions between why some people develop PTSD after a trauma and not others and what increases risk. Individual differences in genetics and neurobiology along with environmental factors undoubtedly influence

neurobiological functioning and psychological development. The cognitive and neuroscientific theories outlined previously have clearly helped advance our knowledge in these areas. However, it should be noted that the neurobiological considerations presented in this review are extremely limited and simplistic and do not represent the complex relationship between behaviour and neurobiology.

Trauma can be understood in terms of heterogeneity of responses in memory systems, appraisals, peri-traumatic processing and post-traumatic interpretations. Multi-factor theories have helped increase our knowledge, in particular DRT and neuroscience model have been useful to further our understanding of how trauma memories may be encoded and stored.

Ehlers and Clark's model expands previous theories by highlighting the role of appraisals of coping strategies, thus acknowledging the bi-directional nature of the appraisal process and the process of dealing with traumatic memories. Both Ehlers and Clark's model and the DRT emphasise two aspects of clinical treatment, the modification of memories and changes to problematic appraisals. As our understanding of theoretical models has advanced so too has our knowledge of specific cognitive function. Increasingly, researchers use performance based neuropsychological tests to access certain cognitive dysfunctions that can be accounted for by specific brain lesions and, by using such tests they can attempt to support those models that relate PTSD to altered brain functioning. The following section will consider these cognitive domains and the deficits reported in the literature.

6. Neuropsychological sequelae of trauma

There is an abundance of research on how psychological trauma affects an individual's cognitive processing. In addition to intrusive thoughts and fragmented memories, the literature points to deficits in a variety of cognitive skills such as memory, learning ability, attention and aspects of executive functioning. Numerous studies have examined memory and attention, using emotional trauma related stimuli such as the

Stroop task, and have reported slower responses and attentional bias (Foa, Feske, Murdock, Kozak, & McCarthy, 1991; Litz et al., 1996). However, this review will focus on the literature around neuropsychological correlates of PTSD associated with emotionally neutral cognitive tasks. The reader is directed to helpful reviews addressing emotional and trauma-related cognitive symptoms such as information processing biases with emotional stimuli (Constans, 2005) and animal models of cognition and stress (Arnsten, 1998).

6.1. Cognitive Domains

In their review, Horner and Hammer (2002) noted that the most commonly researched areas (from trauma studies), and the main clinical presenting complaints by trauma survivors, were difficulties associated with cognitive impairments such as reduced memory and learning ability, attention capacity and skills which fall under 'executive function'. This review will describe the cognitive definitions of each domain before reviewing the literature on the deficits and, or, differences reported in these domains. Please note however, that it is not intended to review the literature on language and motor functioning in PTSD.

7. Literature on Cognitive Impairment in PTSD

7.1. General cognitive ability.

In terms of general cognitive ability, the term most frequently used i.e., IQ. is somewhat deceptive as IQ is not considered to be a unitary or static conception. Few studies have employed a comprehensive protocol to measure the multiple factors of IQ in a PTSD population. However, a number of studies have used the WAIS-R (Wechsler, 1981) test in populations with PTSD (e.g., Brandes et al., 2002; Gil et al., 1990; Gilberston, Gurvits, Lasko, & Pitman, 1997). From these studies two researchers have reported differences in general IQ, where participants with PTSD had significantly

lower overall IQ scores than non PTSD combat veterans (Gil et al., 1990; Gilberston et al., 1997).

In the Gil et al. (1990) mixed trauma study, 12 Israeli PTSD patients, a psychiatric control group (N = 12) and a healthy control group (N = 12) completed the WAIS-R tests and Gil noted that the two patient groups showed impaired performance on a variety of measures in comparison to the healthy control group, including IQ. However, the two patient groups did not significantly differ from each other, implying that distress from psychopathology may account for the effects, rather than something specific to PTSD. One interesting point regarding Gil's conclusion is that they highlighted a deterioration in IQ performance for the PTSD and psychiatric sample based on the comparison of two different assessments i.e., an unpublished Army Intelligence test (with little information on loading factors) that was used pre-trauma and the WAIS-R used post trauma. The comparison of these two different measures brings some doubt over their assertion for levels of IQ deterioration in their findings.

In another study, Brandes and colleagues (Brandes et al., 2002) used a larger sample consisting of 48 individuals who had survived motor vehicle accidents, terror attacks and interpersonal violence for their sample. They compared participants based on scores of either high or low PTSD symptomatology based only on DSM-IV-TR (APA, 2000) Criterion A, and assessed individuals using the WAIS-R within days of experiencing the traumatic event. They found that the high PTSD group showed lower IQ in relation to the low PTSD group. However, Brandes noted that the high PTSD group had a significantly lower level of education and higher anxiety than the low PTSD group and that the observed difference in IQ was explained by depressive symptoms.

Other studies using the WAIS-R have reported no significant differences of IQ in their samples (e.g., Bremner et al., 1993; Yehuda et al., 1995). Neurobiological theories of PTSD do not predict a decline in IQ with PTSD but evidence suggests IQ may be a premorbid risk factor. The association between IQ and PTSD may function through one of several different mechanisms, such as the subjective appraisal of threat

during the trauma such that that those with lower IQ are liable to overestimate the threat of traumatic situations (Macklin et al., 1998); individuals with higher IQ have better cognitive resources to enable them to cope with the emotional impact of traumatic experiences (Schnurr, Rosenberg, & Friedman, 1993) and those with lower IQ tend to have poorer health behaviours and access to mental health resources (Herrnstein & Murray, 1994).

The literature on intellectual functioning in PTSD would be strengthened if some of the methodological issues were resolved. For example, if deficits were studied more frequently in non veterans with appropriate control comparisons rather than for example, college students. In addition, many populations studied were a number of years post-trauma. For future research, longitudinal studies with acute PTSD populations across a variety of trauma types would be helpful in order to more fully understand the relationship between IQ and PTSD.

7.2. Memory, Attention and Executive Function.

Memory, attention and executive function are multifaceted concepts and therefore difficult to measure in a unitary fashion. Attentional processes are often mediated by elements of executive functioning, such as the ability to smoothly switch one's attention, and attention is often a necessary prerequisite for remembering. However, many studies report these as separate concepts when measuring skills in research contexts. It is within these separate domains that we move on to review the literature, via meta-analysis where possible. Many research populations are war veterans and so attempts have been made to identify mixed trauma participant groups for completeness. After a definition of the domain under consideration, the evidence for impairment will be reviewed followed by a brief comment regarding the link with PTSD symptomatology, consequences for maintenance of the disorder and treatment implications.

7.2.1 Memory and learning.

There is a considerable range of definitions for aspects of memory and learning. Generally speaking, learning can be considered to be the process of acquiring new information where memory is the consolidation and retention of acquired knowledge. Several types of memory have been proposed. Descriptions of short and long-term stores of information (Atkinson & Shiffrin, 1968) have given way to theories of working memory (Baddeley & Hitch, 1974) and the classification of the long-term memory concept into sub divisions.

Non-declarative memory is the ability to perform skilled action using objects or the body. This memory is not conscious and the information may come to mind even if there is no awareness or recollection of the prior occurance. The literature implies that non-declarative memory is related to the automatic priming of trauma-related stimuli and underlies our conditioned emotional responses or fear conditioning. As such, it may be responsible for the intrusive re-experienceing of the traumatic event in PTSD (Elzinga & Bremner, 2001). Declarative memory, or explicit memory, refers to memory for specific events in time. This memory is typically conscious and is influenced by a degree of attention and organisation.

Recall memory involves a search or retrieval process, followed by a recognition or decision based on appropriateness of the retrieved information, whereas recognition is essentially the ability to correctly remember something that has been encountered before i.e., a matching process (Eysenck & Keane, 1995). In PTSD, the memory disturbance most commonly reported is that concerned with impaired encoding, storage and or retrieval abilities.

Numerous studies have found differences between PTSD and control groups in memory functioning. In a recent meta-analysis Johnsen and Asbjørnsen (2008) reviewed 28 studies and reported that overall, PTSD participants had marked verbal memory impairment compared to healthy controls. They also point out that war veterans have demonstrated the greatest level of impairment when compared to

individuals exposed to physical and, or, sexual abuse. In addition, they reported that a greater effect size was observed dependent on the test procedures used, i.e., on the Wechsler Memory Scale Logical (WMS; Wechsler, 1981) and the Rey Auditory Verbal Learning Test (AVLT; Lezak, 1995; Rey, 1941) as compared to the CVLT (Delis et al., 1987).

A second meta-analysis by Brewin, Kleiner, Vasterling, & Field (2007) has examined memory for emotionally neutral information and confirmed that there is evidence for verbal memory deficits in individuals with PTSD. However, not all studies have found this deficit and there may be a number of factors affecting the findings of impaired memory performance. For example, study methodology, particularly small sample sizes, and different exclusion criteria between studies in terms of co-morbidity and substance misuse. Moreover, the neuropsychological tests used demonstrate different effect sizes and all tests have different validity and reliability information. Lastly, the influence of participant characteristics, such as when some studies use a student population**,** others use war veterans, others a more mixed trauma sample. Any or a combination of these factors have the potential to compromise and confound results, limiting the interpretation of the findings. A number of studies are discussed in the following section to highlight the numerous differences in findings in the literature.

In 1993, Bremner and colleagues (Bremner et al., 1993) used the WMS with 26 Vietnam veterans with combat related PTSD and 15 healthy community controls, matched for alcohol use and IQ. They found significant differences between the groups on sub tests across the verbal and visual domains on both immediate and delayed recall tasks.

Similiarly, Sachinvala et al. (2000) also observed significant differences between 36 male war veterans with chronic PTSD and controls where the group with PTSD performed poorly on all but two sub tests of a computerised memory task incorporating visually and verbally presented material, measuring aspects of memory, attention and executive function. In terms of a non veteran population, Jenkins, Langlais, Delis, and

Cohen (1998) found that rape victims with PTSD showed significantly worse delayed free recall, and also reported trends of poorer short delay recall and performance in list-learning than a control group.

Conversely, not all aspects of memory are compromised in PTSD and a number of authors have reported significance in some, but not all, specific component processes of memory. This is illustrated in a study by Yehuda et al. (1995) who found no differences for learning and immediate memory between participants with combat related PTSD and healthy controls, but did find statistical differences with regard to retention of material following exposure to an interference task. This suggests that people with PTSD may have fairly specific deficits in the monitoring and regulation of memory information. Furthermore, Vasterling, Constans, Brailey, and Sutker (1998) reported differences between PTSD and non PTSD gulf war veterans in showing heightened sensitivity to retroactive interference when initial learning is taken into account, more frequent intrusion on free recall tasks and poorer performance on tasks requiring learning of verbal stimuli.

Other researchers have not found any differences in either general memory tasks, verbal or visual memory or in any specific memory components. A study by Gurvits et al. (1993) using a war veteran population reported that 27 individuals with PTSD performed less well but not significantly, on the memory tasks compared to 15 combat veterans without PTSD. Interestingly, the researchers point out that PTSD participants demonstrated significant correlation between neuropsychological tests scores and more neurological soft signs than non-PTSD participants.

In a non veteran sample population, Stein, Kennedy, & Twamley (2002) reported no differences in their study on PTSD in survivors of childhood sexual abuse using verbal learning lists. Johnsen and Asbjørnsen (2008) in their meta-analysis noted that the effect size for memory deficits was stronger in war veterans than other trauma groups and yet Zalewski, Thompson, and Gottesman (1994) found no differences on tests of verbal or visual memory (immediate and delayed) between 241 Vietnam

veterans with PTSD, 241 veterans with anxiety disorder and 241 veterans without a psychiatric history, although trends in the data were reported and correlations with the severity of PTSD and lower scores. A strength of this study was that it benefited from a very large sample size, where all the participants may have been exposed to combat trauma of some description. But a limitation was the use of a self-report measure to ascertain PTSD symptoms rather than a structural diagnostic interview.

Overall, PTSD studies reporting positive findings of memory dysfunction, despite confounding factors such as the variance of neuropsychological test methodology, have revealed that a degree of memory impairment is consistently associated with PTSD. But it is clear given the variation of findings over numerous studies, that not all aspects of memory are equally compromised. The most pervasively impaired aspects appear to be, free recall (Johnsen & Asbjørnsen, 2008) and verbal memory (Brewin et al., 2007). However, much of the literature reports memory as a unitary construct and does not make a distinction between, or account for, specific memory components such as initial acquisition (Vasterling & Brailey, 2005), sensitivity to interference, and recognition. This makes direct comparisons difficult, highlighting a lack of uniformity across studies. Furthermore, Johnsen and Asbjørnsen (2008) noted that verbal impairment is greater in people with co-morbid depression and many studies differ in exclusion criteria for mood disorders, thus potentially confounding findings. In other studies, higher effect sizes are often reported when PTSD groups are compared with healthy controls (Johnsen & Asbjørnsen, 2008) but these groups are not always well matched.

A number of aspects remain unresolved and should be a focus for future research. For example, few studies have addressed the issue between subjective memory complaints and objective memory performance; rarely do studies link findings to illness duration or to self-reports of memory failure, and there are limited findings from longitudinal studies to discover if cognitive impairment changes over time. In addition, few studies speculate on the cause of underlying mechanisms of memory

impairment or include information on attentional problems that may influence performance on memory tasks.

One area greatly under-researched it that of visual memory. Brewin et al. (2007) noted the lack of significant findings on tests of visuo-spatial tasks and linked the DRT model with flashbacks and nightmares suggesting that the DRT theory predicted that PTSD should be associated with poorer verbal memory but not poor visuospatial memory. However, a number of researchers (e.g., Gurvits et al., 2002; Jelinek et al., 2006; Vasterling, Constans, Brailey, & Sutker, 1998) have observed statistically significant poorer performance in visuo-spatial memory by participants with PTSD compared to controls, therefore this area is worthy of further investigation.

The implications of these findings about memory and learning for clinical practice is that the clinician can provide tailored interventions to treat memory impairment and inform best practice in the knowledge that specific areas of memory impairment may differ between clients and findings may also inform any pharmacological and, or therapeutic approach.

7.2.2. *Attention.*

The domain of attention consists of a number of skills. Selective attention (or focused attention) refers to aspects of distractibility, mental tracking and vigilance. Sustained attention is the ability to maintain a consistent response during continuous and repetitive activity. Attentional switching refers to the capacity to switch the focus of attention smoothly between tasks having different cognitive requirements. Divided attention is the ability to focus and respond simultaneously to multiple tasks or multiple task demands (Wright & Ward, 2008).

The findings of attentional dysfunction in PTSD are rather mixed. In an early study, Sutker, Vasterling, Brailey and Allain (1995) investigated performance-based attentional tasks in 108 former Prisoners Of War (POWs). They report that mental tracking and attention span was statistically lower in those POWs who had also

experienced severe captivity weight loss in addition to PTSD symptoms. However, without the inclusion of a comparison control group determining the nature of the dysfunction is difficult as severe physical problems associated with being a POW (i.e., torture and, or, malnutrition) can impair cognitive functioning (Sutker, Allain, Johnson, & Butters, 1992).

Other literature of veterans includes the study by Sachinvala et al. (2000) who reported significant differences on all attention tasks on a computerised test known as the Cognitive Evaluation Protocol ® (CEP; McGuire et al., 2000). Sachinvala and colleagues referred to the attention tests in the form of 'simple', 'double' and 'reverse' domains but interestingly, they failed to find timing/reaction differences on the tasks, suggesting that PTSD participants were neither more careful nor impulsive than the control group. Overall, the study was designed to assess cognitive and functional capabilities in a sample of 36 war veterans and 18 controls. The researchers expected to find general cognitive impairment in the PTSD sample and reported significant findings in all cognitive domains. They also found a selective interaction between depression and memory but did not find an association between attention and depression. The authors suggested medication effects were unlikely to explain the findings. However detailed demographic information on the participants was sparse. It was reported that they were attending treatment and the PTSD was diagnosed as chronic, which may have other implications on the interpretation of the findings. In this respect, it is speculated that there may be a number of factors that could influence concentration and poor attention. These include poor sleep quality which may 'stretch' limited cognitive attentional resources. Another explanation might be if the sample experienced high levels of dissociation, ranging from day dreaming to full-blown dissociation which is also known to be prevalent in PTSD.

In another study, Vasterling et al. (1998) used an assortment of eight different measures of attention and found deficits on tasks of sustained attention, but not on measures on selective attention or flexibility of shifting attention in their sample of

veterans**.** This pattern implies limited ability to sustain attentional performance but not to selectively attend to it in the first instance. The authors suggested the observed impairments were consistent with models of PTSD that highlight the role of hyperarousal and implicate dysfunction of frontal-subcortical systems.

In contrast others (e.g., Golier et al., 1997; Yehuda et al., 1995) have not found evidence of a generalised attention deficit on any (initial, sustained or selective) measures between war veterans with PTSD. Although Yehuda concluded, in line with Vasterling et al. (1998), that PTSD patients may have fairly specific deficits in the monitoring and regulation of memory information rather than difficulties attending to it in the first place.

In a population of 15 rape victims, Jenkins et al. (2000) using a variety of tests (the Paced Auditory Serial Addition Test (PASAT; Levin et al., 1987); the Continuous Performance Test (CPT; Loong, 1988); WAIS-R Digit Span tests and the Trail Making Tests (Reitan, 1958) noted that attentional performance of the victims with PTSD was significantly impaired in comparison to 16 rape victims without PTSD and 16 non trauma exposed controls, on measures of divided and sustained attention, but not on measures of visual selective attention. Jenkins also controlled for potential confounding variables and noted that that depression played only a minor role in mediating deficits in sustained attention.

In common with other cognitive domains, findings of attentional deficits in PTSD are mixed and this may be due to the variance in tests and task demands (e.g., task duration, stimulus duration, response requirements). In addition, the delineation between attention and executive function tasks can be indistinct. Despite this, there is evidence to suggest that during tasks requiring sustained attention, those with PTSD perform more poorly than controls. This information is potentially important from a treatment perspective. If PTSD patients are characterised by a sustained attention bias, this has consequences for both exposure-based and cognitive based therapies. Many clinical researchers have adopted a threat stimuli paradigm to study the

attentional processes in patients with PTSD but further research using emotionally neutral stimuli is required in order to clarify the differences noted previously.

7.2.3. Executive Function.

Executive functioning refers to higher-level cognitive processes required to plan, organize, employ strategies, adapt to novel sequences, correct errors and inhibit irrelevant or maladaptive thoughts during day-to-day functioning. These processes require the successful integration of a number of skills, including initiation and motivation, attention, aspects of memory and executing plans and self-regulation, monitoring and the ability to be mentally flexible (Stuss & Benson, 1986).

Studies of executive function and other cognitive skills related to frontal lobe functioning are relatively limited and very few studies have focused on executive function alone. The ability to employ the strategies mentioned previously, are all critical for everyday functioning but the challenge for neuropsychological tests is to be sensitive enough to assess the sub-regions of the prefrontal cortex implicated in the neurobiological theories for executive functioning. Given this inherent complexity, most tests rely on the assessment of mental flexibility to determine executive function deficits.

In terms of research, a number of studies have associated PTSD with deficits in a variety of cognitive operations associated with mental flexibility tasks (e.g., Beckham, et al., 1998; Gilbertson et al., 2001; Koenen et al., 2001; Koso & Hansen 2006; Uddo et al.,1993; Vasterling et al., 1998).

Executive neuropsychological tasks are sensitive to problems in complex solving problems, information processing and flexibility of abstract thought (Bryson, Whelahan, & Bell 2001). Tests commonly used include the Wisconsin Card Sorting Test (WCST; Berg, 1948; Heaton, Chelune, Talley, Kay, & Curtiss, 1993) to measure executive memory and flexibility; the Trail Making Test (Reitan, 1958) to measure cognitive flexibility; the Tower of London Test (ToL; Krikorian, Bartok, & Gay, 1994) to measure

intentionality and the Stroop Color and Word Test (SCWT; Stroop, 1935) to measure inhibition.

Beckham et al. (1998) found that combat veterans with PTSD performed significantly worse than veterans without PTSD on the Trail Making Test. Also using the Trails Test, Koso and Hansen (2006) examined the performance of 20 Bosnian male combat veterans with PTSD, and reported a significantly slower performance than a non PTSD control group. Unfortunately, Koso and Hansen's PTSD sample suffered from a heavy percentage of co-morbid psychiatric diagnoses and over half were on psychotropic medication thus limiting the conclusions that can be drawn from the association to PTSD rather than to other confounds.

A more recent study by Kanagaratnam & Asbjørnsen (2007) focused entirely on executive functioning in a sample of 22 individuals with chronic PTSD following exposure to political violence. The test protocol included the WCST; the ToL, Trail Making, and the Stroop test. They observed impaired performance on executive memory for the PTSD in comparison to a war-exposed control group. However they failed to find any significance in the executive components of intentionality and inhibition between the groups.

Similar mixed findings have been reported by Barrett, Green, Morris, Giles and Croft (1996). Using the WCST as measure of cognitive flexibility and set shifting they reported that veterans with PTSD performed significantly worse than veterans without PTSD, although they noted that these effects were not accounted for by PTSD alone, but by concurrent anxiety and depression. Yet other studies have reported no significant findings in any task of executive functioning (e.g., Dalton, Pederson, Blom, & Beysner, 1986; Gurvits et al., 1993).

The limited literature currently available on executive functioning suggests that PTSD symptoms may be related to processing difficulties and impairment in executive memory. Given deficits in mental flexibility this could also have an impact on the processing of traumatic memories delaying recovery. Overall, this area is presently

under researched and requires further extended and systematic study in populations with PTSD.

8. Clinical Considerations

The usefulness of research examining neurocognitive functioning in PTSD will depend on how successfully the findings are incorporated into prevention and early intervention treatment. DSM-IV-TR (APA, 2000) criteria incorporates memory and attentional deficits in to its diagnostic criteria and neuropsychological assessment is generally routine where brain injury is suspected after a traumatic incident (e.g., a road traffic accident). Although there are mixed findings concerning neurocognitive deficits and PTSD, meta-analytic reviews point to memory impairment, particularly in levels of free recall (Johnsen & Asbjørnsen, 2008) and verbal memory (Brewin et al., 2007). These memory difficulties along with potentially impaired sustained attention and executive memory problems indicate a processing style that might have implications for the development, maintenance and clinical treatment of post-traumatic symptoms.

In terms of therapeutic treatments for those individuals who suffer PTSD, it is suggested that interventions should also target the reduction of symptoms of major depression which is highly co-morbid with PTSD and has been related to response suppression problems and shown to influence memory, attention and executive functions in PTSD.

Another possible explanation for deficits in cognitive performance in PTSD is that individuals may not be sufficiently motivated or engaged in the tests to perform to their full potential. Many studies do not routinely include measures of motivation in the test protocol. However, there is little evidence in the literature of the general and global deficits that one would expect to see if participants were not putting in full effort to the tasks. An alternative explanation could be that the high correlation of depression with a PTSD population may reflect executive function type deficits regarding initiation. Secondary gain issues have also been addressed in the form of compensation claiming

participants or treatment seeking samples and results have been incredibly similar regardless of participant motivation (e.g., Beckham et al., 1998; Vasterling et al., 2002). Furthermore, PTSD is associated with increased arousal and enhanced attention in specific contexts (i.e., trauma related stimuli) thereby negating the possibility of lack of motivation or effort. Additionally, the pattern of cognitive deficits is in line with neurobiological conceptualisations of PTSD in terms of dysfunction within the hippocampus (Bremner, 2001) and also corresponds to observations from electrophysiological and neuroimaging research (Shin et al., 2005).

9. PTSD and cognitive impairment - consequence or cause?

The mechanism by which the cognitive difficulties mentioned previously occur is unclear. Whether they develop as a consequence of PTSD, or if they occur as a consequence of the adverse effects of extreme stress causing brain damage remains an unanswered question. Many studies in this area include trauma survivors with and without PTSD, but few compare trauma exposed PTSD, trauma exposed non PTSD and normal control groups. Additionally, Horner and Hammer (2002) have argued that co-morbidity in PTSD samples makes conclusions regarding cognitive functioning and its association with PTSD difficult to disentangle, further complicating the picture. As PTSD is commonly part of a very complex clinical picture, further studies on samples with common co-morbidity, and trauma exposure is required to gain insight into the mechanisms of the disorder.

10. Summary

In summary, the literature reviewed here suggests that some neurocognitive deficits may be associated with PTSD. The findings are largely indicative of impaired verbal memory with some studies also finding differences in attention and elements of executive function, especially cognitive flexibility. However, a number of factors limit the generalisability of these findings: i) the small sample sizes often used; ii) a failure to

adequately control for co-morbidity; iii) a specific focus on war veterans; and iv) a considerable variety in methodological approaches and neurocognitive tests used.

11. Conclusion

This paper has described the diagnosis and context of PTSD, discussed two key cognitive theories and explored neurocognitive literature on findings of neuropsychological tests using neutral stimuli. The psychological theories offered a framework to aid our understanding of PTSD by accounting for the heterogeneous nature of responses and outcomes. The DRT model and the complementary neuroscience approach has been pivotal in synthesising earlier approaches and conceptualising two types of trauma memory, whilst Ehlers and Clark's cognitive model highlights the paradox of PTSD where anxiety is associated with an event that took place in the past.

Several themes have emerged through this review, notably that cognitive deficits are not global or limited to tasks using only emotional stimuli. Neutral stimuli cognitive tests report significant findings of deficits within the domains of attention, memory and executive function. Abnormalities are being discovered in specific neural systems and a pattern of complex interaction between these systems is emerging. It is clear that neuropsychological research into trauma and PTSD in particular, has much to offer in furthering our understanding of the relationship between neurobiology, cognitive difficulties and PTSD. Difficulties such as post trauma memory impairment, poorly integrated memories, psychogenic amnesia, subjective reports of everyday memory problems, attentional and executive functioning need to be understood better and only continued and careful investigation can accomplish this.

Empirical Paper

Cognitive Differences in Post-Traumatic Stress Disorder

Part Two

by

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Abstract

PTSD has been found to be associated with verbal memory impairment, but findings in other cognitive domains, particularly visual memory are rare and less conclusive. It is unclear if these are PTSD-related or due to coexisting psychological symptoms or trauma history.

Neuropsychological test performance, PTSD symptoms and mood were examined in 26 individuals with PTSD, 26 trauma exposed individuals without PTSD, and 26 controls.

The PTSD sample showed significant impairment of visual spatial recall for complex figuration, independent of depressive symptoms. Differences were observed in aspects of verbal memory and executive function but not attention.

Deficits in visual memory may reflect characteristic features of PTSD, such as flashbacks and have important implications for current understanding of fear-learning

Key words:

PTSD; Post-traumatic stress disorder; cognitive; verbal memory; visual memory; attention; executive function; CVLT, SVLT, Rey Osterrieth.

Introduction

1.1. Post-Traumatic Stress Disorder (PTSD)

PTSD is a disorder that can occur in some individuals following exposure to one or more traumatic events where they experienced intense fear, helplessness or horror. It is classified as an anxiety disorder and characterised by symptoms of persistent re-experiencing of the event(s) (e.g., flashbacks, nightmares), persistent avoidance of stimuli associated with the trauma, numbing of general responsiveness, (e.g., anhedonia, reduced affect) and increased arousal (e.g., insomnia) (DSM-IV-TR; American Psychiatric Association, 2000). PTSD has also been linked with psychogenic amnesia and empirical reports of memory dysfunctions, particularly poorer memory and learning (Yehuda, Golier, Tischler, Stavitsky, & Harvey, 2005) and these memory dysfunctions are at the centre of current PTSD models (for example, Brewin, 2008; Ehlers & Clark, 2000).

There also appear to be prominent individual differences as indicated by the fact that not all studies have reported significant findings (e.g., Jelicic, Geraerts, & Merckelback, 2008; Twamley, Hami, & Stein, 2004)**.** Some individual differences and risk factors that may contribute to the development of PTSD and possible cognitive difficulties include the severity of the trauma, level of mood and anger problems and intellectual functioning (Brewin, Andrews, & Valentine, 2000).

In addition, clinicians and researchers are increasingly becoming familiar with patients' description of problems with attending to and recalling basic information whilst at the same time, reporting distressing, intermittent and recurrent intrusive memories of the trauma. This dichotomy of both decreased and enhanced memory functioning in PTSD has not gone unnoticed and has generated a great deal of interest in the unique characteristics of memory in PTSD. For example, in memories with emotional meaning such as those related to the trauma, electroencephalographic activity has demonstrated those with PTSD show biased processing towards trauma related stimuli and reduced processing of neutral material (Karl, Malta, & Maercker, 2006 for review; Semple, et al., 1996; Vrana, Roodman, & Beckham, 1995).

If intrusive memories and elevated arousal in PTSD reflect a general interference with cognitive processing as suggested by Kolb & Whishaw (2003), it has been hypothesized that beyond trauma memories, impairment can be seen in general cognitive ability. For example, studies have noted that individuals with PTSD show cognitive impairment in the domains of attention (Gilbertson, Gurvits, Lasko, & Pitman, 1997; Sachinvala et al., 2000; Vasterling, Duke, Brailey, Constans, Allain, & Sutker, 2002), executive function (Beckham, Crawford, & Feldman, 1998), initial acquisition of information (Vasterling, Constans, Brailey, & Sutker, 1998) and learning and memory (Bremner et al 1993; Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001; Jelineck, 2006; Moradi, Doost, Taghavi, Yule, & Dalgliesh, 1999; Uddo, Vasterling, Brailey, & Sutker, 1993; Vasterling, Brailey, & Sutker, 2000). These will now be considered in more detail.

1.2. Verbal Memory Dysfunction in PTSD

The literature on memory in PTSD suggests that memory impairment is modality specific, with a pronounced deficit in verbal memory (Brewin, 2001; Brewin, Kleiner, Vasterling, & Field, 2007; Horner & Hamner, 2002; Vasterling & Braily, 2005).

A recent meta-analytic review by Johnsen and Asbjørnsen (2008) supported this finding as they reported a consistent impairment of verbal memory in patients with PTSD as compared to controls. Stronger effects were reported for war veterans rather than those exposed to physical and sexual abuse (Johnsen & Asbjørnsen, 2008). However, this analysis was limited to findings on one memory function i.e., free recall, whereas the inclusion of the complementary sub-components of memory performance (encoding, storage, and retrieval) would have offered a more complete memory profile. For example using a test such as the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 2000) which includes specific measures of memory function like list learning, total recall across all trails, long and short delay cued and free recall, vulnerability to interference, recognition performance and forced choice recognition, which also to identifies potential

insufficient effort (Delis et al., 2000), and error type (repetitions, intrusions, and false positives) enables various aspects of memory function to be evaluated separately.

Data recorded included list learning, total recall across all trails, long and short delay cued and free recall, vulnerability to interference, recognition performance and forced choice recognition (also to determine lack of effort), and error type (repetitions, intrusions, and false positives).

Brewin et al. (2007) has also examined memory for emotionally neutral information by meta-analysis. They examined 27 studies for the moderator variables that have been implicated as potentially accounting for group differences in varying types of trauma. Brewin and colleagues concluded, that despite extensive variety between the studies, individuals with PTSD show a small to moderate association between verbal memory deficits and PTSD. In addition, and contrary to the findings from Johnsen and Asbjørnsen's (2008) analysis, Brewin noted that the association was not specific to certain populations in that civilians exposed to state persecution or terror demonstrated the greatest magnitude of difference. Although this does offer some clarity in an area of research that has mixed results, the complexity of analysis meant that the effect of different memory neuropsychological tests, specific memory components or the control groups within the different types of traumas were not considered.

Overall, the extent of verbal memory dysfunction has varied considerably among studies and not all aspects of memory are equally compromised in PTSD. For example, Yehuda et al. (1995) reported normal ability for immediate memory, cumulative learning, and active interference from previous learning and concluded that patients with PTSD have specific deficits in monitoring and regulation of memory.

More recently, Vasterling and Brailey (2005) have suggested that PTSD is characterised by less proficient initial acquisition of information and heightened sensitivity to interference. Most studies have typically restricted measurement of memory to free recall formats. Although there is less consistent evidence of impairment in recognition over time, Isaac, Cushway, and Jones (2006) suggested the distinction between initial acquisition and

retention is important regarding underlying neuroanotomical features of brain function and links to PTSD symptomatology. For example, verbal memory impairments have been linked both to increased arousal state and intrusive re-experiencing phenomena (Kolb & Whishaw, 2003). Memory retention is more closely linked to hippocampal functioning, acquisition skills during verbal fluency tests have been linked to the hypoactivation (Matsuo et al., 2003) and altered functional integrity of the prefrontal cortex and the ability to spontaneously utilize strategic processes (Vasterling & Brailey, 2005).

Furthermore, many studies have not reported on the more qualitative aspects underlying verbal memory impairments such as error types. It is helpful to consider what type of errors are made on neuropsychological tests as incorrect answers may provide more information about specific memory functioning, particularly in relation to everyday activities and cognitive flexibility. Using such tests as the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 2000) which includes specific measures of error type, means some features of memory function can be evaluated separately. This test also identifies potential insufficient effort (Delis et al., 2000).

1.3. Visual Memory

Given the plethora of studies on verbal memory, there is surprisingly little research on visual memory in PTSD. Recently, Gilbertson and colleagues (Gilbertston et al., 2007) highlighted the growing picture in animal literature of the hippocampus as a crucial area for the processing of contextual information as it relates to fear-mediated learning. In humans, Incisa della Rocchetta and colleagues proposed that the hippocampus is crucial to recall of spatial location, particularly when the individual is focussing on environmental feature(s) (Incisa della Rocchetta, Samson, Ehrlé, Denos, Hasboun, & Baulac, 2004). It is, therefore, valuable to conduct visual memory assessment, as abilities such as remembering patterns and spatial positions are important functionally relevant skills that underpin everyday activities, such as navigating in an unfamiliar environment, relocating an item previously put

down (Mapou, 1992), driving a car or for people employed in architecture, driving, art and graphic design (Danckwerts & Leathem, 2003; Shum, Harris, & O'Gorman, 2000).

In those studies that have examined visual memory, findings have been mixed. For example, Gilbertson et al. (2001) and Gurvits, Carson et al. (2002) did not find any group differences on a complex figure-drawing task in their sample. Whereas others (Gurvits, Lasko, Repak, Metzger, Orr, & Pitman, 2002; Jelinek et al., 2006; Stein, Kennedy, & Twamley, 2002; Vasterling, Constans, Brailey, & Sutker 1998), have reported that PTSD diagnosed participants performed more poorly on visuo-construction and visuo-spatial memory ability than controls or trauma exposed participants.

However, most studies that explore visual memory often select a fairly simple uni-dimensional single assessment test that generally involves a reproduction component that greatly relies on the participant's motor skills and construction ability. Therefore, any observed impairment may be due to a physiological, organisational, visuo-spatial or a visuo-perceptual problem or a combination of these, and potentially confound the results (Loring & Papanicolaou, 1987). Importantly, Stein et al. (2002) observed that the differences in their sample were not due to the organization of the task (i.e., aspects of executive function).

In addition, Shum, Harris and O'Gorman (2000) noted that most visual tests do not allow an examination of visual memory functions such as degree of new learning, speed, recognition or retention of information. It is likely, therefore, that researchers are unable to obtain information on any subtle visual impairment in a PTSD population. Using a test instrument such as the Shum Visual Learning Test (SVLT; Shum, O'Gorman, & Eadie, 1999) where the individual's ability to recognize visual patterns is not dependent upon re-constructing the figure would address this.

The familiarity of the visual material presented is also an important factor. Heilbronner (1992) suggested that increased familiarity heightens the chances of the material being dependent on language related skills (i.e., verbalisation), rather than on visual spatial abilities; therefore, on language-related rather then visual-related areas of the brain (Heilbronner, 1992; Lee, Loring, & Thompson, 1989). To address this, Shum et al. (1999)

proposed that Chinese characters present visuo-spatial relationships in the form of dashes, lines, strokes and dots and are unfamiliar to people whose language is not Chinese or who have not studied the language. This element of unfamiliarity and a recognition rather than reconstruction process, without the need for administrator judgement in marking the task, provides a potentially useful measure of memory. Additionally, Danckwerts and Leathem (2003) specifically recommend the Shum Chinese Figure task for a PTSD population as it dissociates between aspects of learning, speed, recognition and retention of information, thereby offering the potential to reveal precise impairment.

1.4. Attention and Executive Function

In a similar vein to the literature on memory deficits in PTSD, the findings in executive function and attention dysfunction in PTSD is equally mixed. For example, Yehuda et al. (1995) found that PTSD patients performed within normal limits on an attention task until an interference condition was introduced, then the performance of PTSD patients, specifically their capacity for retention, diminished. This finding suggests that memory defects in PTSD may reflect underlying disturbances in the monitoring and regulation of memory, rather than attention to the task in the first instance. However, many self-reported complaints of memory difficulties highlight attention difficulties as well as problems with recall (Wolfe & Charney, 1991). As individuals can experience memory difficulties that are primary, or secondary to attention problems it is useful to include a measure of attention in any neuropsychological protocol to attempt to clarify this point.

In terms of executive functioning, the literature links PTSD to deficits in a number of cognitive operations associated with executive functioning (Beckham, et al., 1998; Gilbertson et al., 2001; Kanagaratnam & Asbjørnsen, 2007; Uddo et al., 1993). PTSD symptoms such as severe arousal may interfere with cognitive flexibility (McFarlane, 2000). But again, as with many neurological test findings in PTSD, other reports of deficits are more mixed (Johnsen, Kanagaratnam, & Asbjørnsen, 2007) or inconsistent regarding whether executive functioning difficulties could be accounted for by depression (Johnsen et al., 2007;

Leskin & White, 2007). Overall, it is unclear if individuals with PTSD exhibit executive function deficits and, if so, if they are independent of depressive symptoms. Therefore, a simple measure of cognitive flexibility should be included in study protocols.

1.5. Everyday Memory Function

There is a modest amount of research into the increasingly reported subjective difficulties of everyday functional memory (Danckwerts & Leathem, 2003). As noted previously, patients are complaining of poor functional memory, while memories of their traumatic past remain vivid and prominent (Yehuda, Golier, Halligan, & Harvey, 2004). Obtaining a reliable measure of the functional impact and degree of such difficulties presents a dilemma. Traditional neuropsychological tests seem unable to specifically detect the relationship between test performance and behaviour in everyday settings (ecological validity), confounded either by methodological inconsistencies or tests that do not provide a comprehensive examination of memory. This may be because tests have been designed for those with brain lesions or alternatively, because PTSD is related to performance fluctuations which may not be picked out by tests. A memory questionnaire is therefore a useful adjunct to traditional tests to improve ecological validity.

1.6. Methodological Considerations

Despite extensive research, the evidence for cognitive dysfunction in PTSD patients remains inconclusive. Some differences may be explained by the variation in methodological approaches. Perhaps one of the most significant issues is determining whether the cognitive deficits are features of PTSD, a result of the trauma or other confounding variables. For example, given the high prevalence of depression, anxiety disorders and substance abuse (see Brady, Killeen, Brewerton, & Lucerine, 2000, for a review) many studies have not addressed co-morbidity (e.g., Gilbertson et al., 1997; Sachinvala et al., 2000; Vasterling et al., 2000). It is important to be aware of these potential confounds as others have reported that depression in PTSD is related to cognitive impairment (Johnsen et al., 2007).

In addition, small sample sizes are prevalent and do not supply adequate power to reveal differences among variables if there are medium or small effects, and could lead to significant positive findings in samples that are not typical of the population in question. Other studies have not matched groups on important demographic characteristics such as age and gender (Uddo et al., 1993), and few studies have included measures of response bias or motivation, issues that could certainly influence test performance.

Another research approach has been to focus on specific populations such as war veterans (Barrett, Green, Morris, Giles, & Croft, 1996; Semple et al., 1996; Uddo et al., 1993), with fewer studies presenting data from mixed civilian populations. Others have used student populations (Twamley et al., 2004) who may differ in significant ways to the clinically diagnosed PTSD population in terms of level of distress, co-morbid diagnoses and premorbid IQ.

Given these limitations it is difficult to conclude whether the positive findings reported are related to PTSD, demographic variables, co-morbidity or indeed, if the impairment is more directly associated to the stress of trauma exposure. This latter point is important as evidence for the possibility that the cognitive changes of PTSD may be due to the stress effects of trauma exposure are being highlighted in the literature on neuroendocrine functioning. Some authors are suggesting stress-related glucocorticoid alterations and increased variability in cortisol release could underlie changes in cognitive functioning, either as a cause or as an effect of hippocampal functioning (Sapolksy, 2002; Yehuda, 2001).

1.7. Research Aims

Verbal memory deficits in PTSD have been extensively studied but visual memory has been less intensively investigated. The current research aims to explore the recognition of complex visual patterns (with and without a construction element using the Shum Visual Learning Test (SVLT), a recently developed computer task.

No single study can hope to fully address the many complicating factors highlighted previously. However, this paper will attempt to account for some of the methodological issues noted, for example, where studies have used extremely small sample sizes.

Given the inconsistencies reported, a measure of verbal memory, attention and executive functioning will be included in the neuropsychological assessment. Further, by recruiting a mixed sample of participants exposed to trauma with and without PTSD, and a control group, it is hoped to identify deficits that can be accounted for by PTSD or by trauma exposure. Finally, confounding factors of trauma severity, depressive symptoms and anxiety, along with a measure of subjective experience of everyday memory failure will be considered.

1.8. Research Questions

1. Do traumatised participants demonstrate impaired performance in memory, attention, and executive functioning on neuropsychology tests in comparison to controls?
2. Is the degree of impairment related to the severity of PTSD symptomatology?
3. Do co-morbid depressive symptoms and, or, anxiety account for any of the impairments?
4. Will reports of failure in everyday memory function be significantly greater in the PTSD sample?

Method

2.1. Participants

2.1.1. Recruitment.

Participants were recruited from a number of sources. Some were tertiary care outpatients from a local city hospital. These patients were screened from an original patient list of over 90 patients. Initial identification of potential participants was undertaken by their Psychiatrist at the hospital and screening criteria involved identifying those with experience

of a traumatic life event more than 6 months previously, were aged between 18-75, spoke English as a first language, and indicated a willingness to undertake research. After screening, 33 individuals were invited by the author to take part in the study, 15 individuals responded.

Following the same screening criteria listed above, the author examined data relating to over 70 individuals registered in a Motor Vehicle Accident (MVA) survivor database maintained by the University of Southampton and invited 45 individuals to take part in the study, 15 people responded to the invitation and 11 people were invited to take part in this study. In total over 160 individuals from the hospital and the MVA data base were screened for suitability to take part in the study.

To expand the participant population, the study information was also sent (with permission) to 24 local GP surgeries, a combat veterans support group, a military base and a number of posters were put up around the University campus. A letter of invitation was also published in two local evening newspapers. This process resulted in a response from 32 individuals and after initial screening, 27 individuals were invited to take part in the study.

The control group were specifically targeted for recruitment based on their age and gender (i.e., 'cherry picked' to match the PTSD group). These individuals were identified opportunistically i.e., via colleagues, relatives, acquaintances, friends and posters displayed around the University campus. Individuals were invited to take part in the study if they had not experienced a significant traumatic life event in accordance with the dominant categories of traumatic events listed on the Posttraumatic Diagnostic Scale (Foa, 1995), were not being treated for a mood or anxiety disorder and fulfilled the inclusion criteria detailed above. The majority of the recruitment process was shared with another Clinical Psychologist and was conducted over a period of approximately five months.

Following the initial screening process of individuals willing to take part in research, a further set of exclusion criteria particular to this study was specified. This included knowledge of an Asian language (to minimise familiarity with the Shum Visual Learning Test; SVLT) and health issues including a traumatic or acquired brain injury or history of head

trauma; epilepsy; neurological disease or loss of consciousness for more than 10 minutes; recreational drug use or alcohol abuse or dependency; uncorrected hearing or sight loss, and, or, a psychotic disorder. The criteria were imposed because of possible effects on neuropsychological measures. From a total of 83 people, 5 people were not tested because they fulfilled one or more of the exclusion criteria although these individuals did take part in the research conducted by another Clinical Psychologist.

2.1.2. Final sample.

In the final sample, participants were aged between 25-75 (mean 48 years, *SD* = 14.63) and the gender ratio was 39 women to 29 men with 26 participants in each of the 3 groups, 78 people in total.

2.2. Design

This study employed a between groups design using 3 groups: i) a clinical group who met the diagnostic criteria for PTSD; ii) a trauma exposed group who did not meet PTSD criteria; and iii) a non-trauma exposed control group. For the CVLT and SVLT learning components there is a mixed design; between and within (repeated measures). PTSD symptomatology was identified by current symptoms as indicated by the Posttraumatic Diagnostic Scale (PDS; Foa, 1995). The dependent variables were attention, memory and executive function scores. All the neuropsychological tests used have normative data however, a control group was included to increase the robustness of study.

2.3. Ethics

Since this study involved NHS patients, ethical approval was obtained from the National Ethics Research Service (NRES) (Appendix A) and the University of Southampton Ethics Committee (Appendix B). Data Protection Act compliance (Appendix C), Research Governance (Appendix D) and clinical insurance (Appendix E) was obtained.

All participants gave written informed consent (Appendix F) and were advised of their right to withdraw at any time. Guidelines provided by The British Psychological Society concerning ethical principles were followed (Code of Conduct, Ethical Principles and Guidelines). Participants were reimbursed for their time with a gift voucher. Application for ethical review was shared with another Clinical Psychologist.

2.4. Procedure

The protocol for this study was merged with two other studies on PTSD. During any neuropsychological test delay recall periods for this study, participants completed questionnaires from the other studies. Prior to the test session, an information sheet (Appendix G) and the Everyday Memory Questionnaire (EMQ; Sunderland, Harris, & Gleave, 1984) was mailed to participants.

2.4.1. Clinical evaluation.

Participants who had experienced a traumatic event in accordance with the dominant categories of traumatic events of the PDS (Foa, 1995) were allocated to one of two groups based on their response. The PDS is a self-report scale consisting of 49 items used to determine the presence of PTSD symptoms in accordance with DSM-IV-TR (APA, 2000), A - F criteria. An endorsement was required on whether the experience of the traumatic event involved injury or threat to life and, or, feelings of being terrified or helpless. The PDS provided an impairment of functioning over the previous 4 weeks and symptom severity score (range 0-51) and severity ratings are classified as 1-10 mild, 11-20 moderate, 21-35 moderate to severe, 36-51 severe (Foa, 1995). Information regarding the time lapse since the trauma and whether it was a single (Type 1) or repeated (Type 2) experience was obtained.

Participants meeting the complete PDS PTSD criteria (i.e., meeting criteria A, B, C and D (symptom severity scores ranged from 13-45) were allocated to the PTSD group. Participants who did not fulfil the diagnostic criteria were allocated to the Trauma Exposed

(TE) group (symptom severity scores ranged from 3-10). The PDS was verbally administered and scored by another Clinical Psychologist prior to the test session. The PDS has test-retest reliability of $r = 0.83$ and validity of $r = 0.75$ (Foa, Cashman, Jaycox, & Perry, 1997)

All participants were evaluated on the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) for the presence of anxiety and depressive symptoms. Participants also gave their age, education, hand preference, marital status, history of psychiatric diagnoses, medication and alcohol intake.

2.4.2. Neuropsychological assessment.

The standardized and validated neuropsychological tests were selected to answer the research questions based on ease of administration, availability of the tests and time constraints. The protocol followed the same presentation for each participant and lasted around 2 hours, including a break; the author was vigilant to potential participant fatigue. Participants were asked regarding intrusive memories and flashbacks and requested to notify the author if they experienced these symptoms during the course of testing, no participant reported either symptom. Tests were conducted and scored according to relevant criteria. Administration time, test-retest reliability and validity data is reported.

2.4.2.1. General intellectual functioning.

The National Adult Reading Test (NART; Nelson & Willison, 1991) assessed ability to pronounce 50 phonetically irregular words and took approximately 3 minutes to administer. Although developed as a measure of premorbid IQ in cognitively impaired individuals, the value of the NART lies with its high correlation between reading ability and IQ in a normal population and is unaffected by psychiatric disorders (Crawford, Parker, Stewart, Besson, & De Lacey, 1989). It has test-retest reliability of $r = 0.85$ and validity $r = 0.85$ (Crawford, Stewart, Cochrane, Parker, & Besson, 1989). In common with other PTSD research (e.g.,

Dileo, Brewer, Hopwood, Anderson, & Creamer, 2008; Buckley, Blanchard, & Hickling, 2002) the NART was used to establish the comparability of IQ between groups in this study.

2.4.2.2. Verbal memory.

The California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 2000) was developed to provide a brief clinical assessment of processes and strategies involved in verbal learning and memory performance (Delis, Kramer, Kaplan, & Ober, 1987). The CVLT reports test-retest reliability of $r = 0.86$ (Delis et al., 1987) and validity of $r = 0.88$ (Delis et al., 2000; Crosson, Novack, Trennery, & Craig, 1988). The CVLT took approximately 42 minutes to administer, including delay time.

2.4.2.3. Visual spatial memory.

Visual spatial memory was examined using the Rey-Osterrieth Complex Figure (Corwin & Bylsma, 1993; Osterrieth, 1944; Rey, 1941). The Rey provides measures of perceptual organisation, visuo-constructional ability and visuo-spatial memory. Test-retest reliability is reported at $r = 0.76$ (Delaney, Prevery, Cramer, & Mattson, 1988) and validity at $r = 0.88$ (Meyers & Meyers, 1995).

Total administration time was approximately 36 minutes including immediate and delay period. The figures were scored following guidelines by Taylor (1959) and Duley et al. (1993). The maximum score is 36 for each figure. The performance of every participant on the copy trial was good with 99% achieving a score of 32 or over. Therefore, only the scores for immediate and delayed recall were used for analysis. A random sample of figures were double marked by a colleague as a reliability check, agreement was 98.2%.

2.4.2.4. Shum Visual Learning Test.

A measure of visual memory was used using the Shum Visual Learning Test (SVLT) (Shum et al., 1999). The SVLT, a newly developed computer based task (Version 1.0, 2006, updated from the Shum et al. (1999) paper version) was designed to assess components of

visual memory without a construction component, which potentially contaminates results (Shum et al., 1999). The SVLT assessed an individual's ability to recognise visual patterns using Chinese characters as target stimuli. Without knowledge of the Chinese language, the unfamiliarity of the characters and their complexity provided a 'pure' examination of visual memory (Heilbronner, 1992; Lee et al., 1989; Shum et al., 1999). Test-retest reliability is statistically significant at $r = 0.82$ and validity is reported as $r = 0.72$ (Shum et al., 1999). Age (but not gender and education) has been found to affect SVLT test performance (Shum et al., 1999).

Target stimuli for Set A (Appendix H) and Set B (Appendix I), were 10 unfamiliar Chinese characters. Each character was yellow, approximately 4cm x 4cm and presented for 2 seconds on a blue computer screen. For each recognition trial participants were shown 20 characters 2 at a time, a target and a distractor. The distractors (Appendix J for Set A, Appendix K for Set B) have an added, deleted or a relocated stroke to the target stimulus.

Participants made a response within 5 seconds, even if uncertain, using the left or right arrow keys on the computer keyboard. The task consisted of six trials, where three learning trials contain Set A stimuli, followed by an interference and recall trial of Set B stimuli, and a 20-minute delay retention trial with Set A stimuli. Administration time, including delay period was 27 minutes. Measures of response time, correct hits, overall learning score, learning index, retention after interference, and delayed retention index were recorded. The scoring output takes into account hit rate and false positive rate (Shum et al., 1999).

2.4.2.5. Attention and Executive Function.

The Trail Making test (Reitan, 1958; 1992) is a two-part pen and paper test measuring complex visual scanning (Shum, McFarland, & Bain, 1990), attention, flexibility of cognition and motor speed (Lezak, 1995). Test-retest reliability is reported at $r = 0.80$ (Spreen & Strauss, 1991) and validity $r = 0.59$ (Delis, Kaplan, & Kramer, 2001). Administration time depends on how quickly the participant completes the task. Most finish both parts within 3½

minutes. Higher test times suggest poorer performance, whilst lower times reflect better performance. The raw score of timed seconds was used for analysis.

Word generation ability and mental flexibility (an aspect of executive function) was examined using the Controlled Oral Word Association (COWA), 'FAS' and 'animals fruits and vegetables' (Benton, Hamsher, & Sivan, 1994; Spreen & Strauss, 1998). Test-rest reliability is reported at $r = 0.71$ (Snow, Tierney, Zorzitto, Fisher, & Reid, 1988) and validity $r = 0.83$ (Delis et al. 2001). Participants have 60 seconds to name as many words as possible that begin with the letter or belong to the category. Raw scores of age, gender and education were adjusted for analysis (Lezak, 1995). Administration of the COWA takes 6 minutes.

Digit Symbol - Coding - (WAIS III) is a substitution task and measures speed of psychomotor processing and sustained attention by transcribing a nonsense symbol that corresponds to a particular digit. Test-retest reliability is $r = 0.88$ (Wechsler, 1981), validity is $r = 0.70$ (Joy, Kaplan, & Fein, 2004). Participants had 90 seconds to complete the task and raw scores of correctly drawn symbols were used for analysis.

2.4.3. Questionnaires

2.4.3.1. The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983).

The HADS is a 14-question self-report measure of anxiety and depressive symptoms. Each question has four possible responses and scores range from 0 to 3 depending on the presence and severity of symptoms experienced during the previous 7 days. Two separate scores, one for anxiety and one for depression (ranging from 0 to 21) were obtained for each participant. Raw scores were used in the data analysis. Administration time is around 3 minutes. The HADS has test-retest reliability of $r = 0.94$ (Herrmann, 1997) and validity of $r = 0.80$ (Bejelland, Dahl, Haug, & Neckelmann, 2002).

2.4.3.2. The Everyday Memory Questionnaire (EMQ; Sunderland et al., 1984).

The EMQ contains 27 questions about common memory lapses encountered in everyday living. Reliability is reported as $r = 0.85$ and validity $r = 0.92$ (Royle & Lincoln, 2008). The participants rate themselves on a 1-9 point scale, evaluating the frequency experienced for each memory failure. A score of 1 equates to 'not at all in the last 3 months' and a score of 9 equates to 'more than once a day'. Total scores ranged from 27 to 243. Higher scores represent greater perceived memory impairment. The EMQ was completed prior to the test session to ensure that performance on the neuropsychology tests did not influence EMQ responses.

2.5. Debriefing

On completion of the session participants were thanked and debriefed (Appendix L). Participants were reminded to contact their GP (or Psychiatrist) if they experienced any distress as a result of their trauma. In addition their attention was drawn to the helpline telephone numbers on the information sheet. Three individuals requested a summary of their personal neuropsychology test results to be sent to their GP or Psychiatrist and a further 8 participants requested a summary of the research findings.

Results

3.1. Statistical Analysis

Statistical analysis was performed on SPSS ® for Windows, Release 15, (2007). A priori power calculations were conducted based on Cohen (1992). In order to provide sufficient power to detect a differences between groups, given a small to medium effect size (alpha =.05, power =.8) there would need to be at least 52 participants. However, Bremner et al. (1993) reported a large effect size ($d < 1.0$) in his study of patients with PTSD (total N = 41). In contrast, Stein et al. (2002) report a small effect size ($d < .10$) in their study of domestic violence and PTSD (total N = 48). Therefore, it was predicted that in a one-way Analysis of Variance (ANOVA) design with three groups, it would be necessary to recruit a

minimum of 23 participants for each group to obtain a medium effect size at α = .05 and a power of .8.

3.2. Data Screening

A significance level was set at an α = .05 for analysis and all tests were one-tailed with the exception of the analysis conducted to ascertain the impact of mood. Data was checked for normal distribution and homogeneity of variance using Kolmorgorov-Smirnov and Levine's statistical tests. Variables that were not normally distributed were transformed using log transformations. Transformation normalised the EMQ data and therefore analysis was conducted on the transformed scores. Transformation did not normalise the data for the List B, short delay cued recall and all error measures (repetitions, intrusions and false positives). A decision was made to use ANOVA on untransformed scores as assumptions of normality can be violated with minor effects due to the robustness of the ANOVA test (Field, 2009; Howell, 2000).

3.3. Stages of Analysis

The main analysis of the data was carried out in a number of stages. Precise statistical test information is detailed in the relevant section. First, group socio-demographic characteristics were analysed. In a second stage of analysis, group differences on the neuropsychological test scores were analysed. Finally, the impact of mood, PTSD severity, self reported everyday memory and medication on the neuropsychological tests scores was explored.

3.4. Group Characteristics and Evaluation of Potential Confounds

Information on clinical characteristics was obtained from demographic questionnaires. The groups were matched by age and gender. One-way ANOVA and Bonferroni-corrected post hoc *t*-tests were conducted to compare means of age for the all groups. No significant age differences were found (see Table 1). On the nominal/categorical data of gender,

handedness, marital status, medication and diagnosis a Chi square test was conducted. A *t*-test was used to check for group differences between the PTSD and TE groups for Type 1 or Type 2 trauma occurrence. The PTSD group reported significantly more Type 2 trauma than the TE group.

Differences in years of education, level of education and IQ were tested by ANOVA, no significant differences were found. As intelligence level may reflect a risk factor in PTSD measures, NART (raw score, number of words correct) and years spent in education were used to obtain an estimate of aptitude for each participant.

Other data obtained included marital status, weekly consumption of alcohol and handedness, which were tested, by ANOVA or Chi square test. No statistical difference was found for marital status or weekly alcohol consumption between the three groups. A number of individuals in the TE and PTSD groups carried co-morbid diagnoses and were taking medication.

All participants completed the HADS (Zigmond & Snaith, 1983). ANOVA analysis demonstrated a significant group effect for anxiety. Post hoc Tukey tests revealed a significant difference between the Control and PTSD group ($p = .001$) and the TE and PTSD group ($p = .001$). However, there were no significant differences between the Control and TE groups in reported levels of anxiety.

The HADS score for depression also revealed a significant group difference. Post hoc testing verified significant differences between the Control and PTSD groups ($p = .001$) and the TE and PTSD groups ($p = .001$). As with the anxiety measure, there were no group differences between the Control and TE groups for depression. Therefore, potential confounds identified for analysis of impact on the data were medication, anxiety and depression scores. Data for the above variables are illustrated in Table 1.

Table 1

Demographic and Clinical Characteristics of Participants

	C *M*(S.D.) (A)	TE *M*(S.D.) (B)	PTSD *M*(S.D.) (C)	F/χ^2	df	p [a]	Post Hoc Tukey [b]
Age	48.19 (14.88)	47.46 (14.57)	47.27 (11.0)	0.22	2,77	.80	
Gender Male	N = 9 (34.6%)	N = 10 (38.5%)	N = 10 (38.5%)	0.11	2	.95	
Female	N = 17 (65.4%)	N =16 (61.5%)	N = 16 (61.5%)				
Right Handed	N = 26 (100%)	N = 25 (96.2%)	N = 21 (80.8%)	7.58	2	.02	A > C
Education (*years*)	13.23 (2.58)	13.85 (2.62)	13.62 (2.55)	0.38	2,77	.69	
University education	N = 8 (30.8%)	N = 11 (42.3%)	N = 10 (38.5%)	0.96	2,77	.58	
NART * Score *(correct)*	34.96 (6.82)	37.5 (5.33)	35.62 (7.23)	1.07	2,77	.35	
Married or cohabiting	N = 21 (80.8%)	N = 18 (69.2%)	N = 16 (61.5%)	4.81	6	.57	
Alcohol (*weekly units*)	3.85 (2.68)	4.58 (5.05)	3.08 (4.38)	0.85	2,77	.43	
Co-morbid diagnoses	0	N = 6 (23.1%)	N = 16 (61.5%)	27.50	4	$\leq$.001 [a]	C > A, B
Psychiatric Medication	0	N = 4 (15.4%)	N = 17 (65.4%)	6.45	1	$\leq$.001 [a]	C > B
HADS** Anxiety	5.58 (3.79)	6.85 (3.72)	13.15 (4.35)	27.24	2,77	$\leq$.001 [a]	C > A, B
Depression	2.54 (2.76)	4.38 (3.51)	8.81(5.37)	16.58	2,77	$\leq$.001 [a]	C > A, B

[a]. Statistically significant p <.001 [b] A, Control; B, Trauma Exposed; C, PTSD; groups differ significantly ($p \leq .05$).

*National Adult Reading Test ** Hospital Anxiety and Depression Scale

The PDS questionnaire provided data on each participant concerning the severity of symptoms they had experienced. Table 2 illustrates the mean scores from the TE and PTSD group for the three PDS subscales of re-experiencing, avoidance and arousal and the level of individuals who experienced a Type 2 trauma. Information on the time lapse since the trauma(s) occurred was also obtained. Twenty participants in the PTSD and 17 participants in the TE group reported the trauma took place more than 5 years previously.

Table 2

PDS Severity Sub Scale Scores and Type of Trauma for TE and PTSD groups; N = 26 per group.

	TE	PTSD	t/ χ^2	Df	p [a]
PDS Severity Sub Scale	*M*(S.D.)	*M*(S.D.)			
Score					
Re-experiencing	0.42 (0.76)	7.08 (3.48)	9.51	27.35	$\leq$.001 [a]
Avoidance	1.04 (1.73)	9.96 (4.58)	9.28	31.99	$\leq$.001 [a]
Arousal	1.38 (2.57)	9.81 (3.41)	10.03	46.49	$\leq$.05
Type 2 Trauma	1.58 (0.51)	1.92 (0.27)	7.58	38.41	$\leq$.004

[a] Statistically significant p $\leq$.001

3.5. Group Differences in Neuropsychological Tests

The following section contains the analysis relating to the memory tasks and the tests conducted for attention and executive functions. To limit the number of statistical tests performed, a MANOVA was conduced for learning effects, i.e., trials 1, 2, 3, 4 and 5 of the CVLT and trials 1, 2 and 3 of the SVLT. Only when the MANOVA was statistically significant were ANOVA and corrected Tukey post hoc tests conducted to localise the effects of the individual components. The tables highlight the mean and standard deviation of raw scores or seconds if indicated, for all tasks along with the relevant statistical F and P value of the data for analysis along with effect size and observed power.

3.5.1. Memory - Verbal

3.5.1.1. CVLT.

For the CVLT, a MANOVA, with trial (CVLT 1-5) as the within subjects factor and group as the between subjects factor, revealed a significant main effect for trial $F(4,72) = 206.18$, $p < .001$, partial $\eta^2 = .92$) and group $F(1,75) = 5.82$, $p = .004$ partial $\eta^2 = .13$) but no trial by group interaction $F(8,146) = 1.46$, n.s. Following up the main effect for group, Bonferoni-corrected post hoc *t*-tests revealed that the PTSD group showed significantly lower performance than non-exposed controls ($p = .003$) but not TE participants. The two control groups did not differ significantly in performance. When controlled for depression the group effect was no longer significant ($F(1,74) = 2.30$, n.s.). In order to follow up the significant effect for trial, simple contrasts were computed. They revealed that word recall was significantly higher in the last trial than previous trials, which indicates a significant learning effect across groups. Table 3 shows means and SD per group and results of the one-way ANOVAs per subtest.

Table 3 - *Neuropsychological Data - Verbal Memory - CVLT Scores*

Verbal Memory – CVLT	C *M* (S.D.) (A)	TE *M* (S.D.) (B)	PTSD *M* (S.D.) (C)	F/χ^2	df	p [a]	Tukey [b]	*d*	Observed Power
Trial 1 (initial learning)	8.0 (2.0)	6.81 (1.89)	6.58 (2.27)	3.51	2,77	≤.035 [a]	C < A		
Trial 2	10.54 (2.23)	9.92 (2.33)	9.42 (2.34)	1.53	2,77	.22			
Trail 3	12.65 (2.33)	11.69 (2.89)	10.27 (2.75)	5.25	2,77	≤.007 [a]	C < A		
Trail 4	13.62 (1.85)	12.58 (2.65)	11.19 (2.78)	6.31	2,77	≤.003 [a]	C < A		
Trail 5	14.23 (1.63)	13.35 (2.13)	12.07 (2.19)	7.61	2,77	≤.001 [a]	C < A		
Total over trials 1-5	59.19 (8.85)	54.38 (10.19)	49.54 (11.15)	5.93	2,77	≤.004 [a]	C < A	.15	.95
List B	6.84 (1.78)	5.65 (1.49)	5.88 (1.96)	3.36	2,77	≤.04 [a]	B < A	.52	.95
Recall									
Short delay free recall	11.81 (2.86)	11.58 (2.97)	10.23 (3.48)	1.95	2,77	.15		.70	.99
Short delay cued recall	12.92 (2.12)	12.15 (2.92)	11.04 (3.23)	2.73	2,77	.07		.77	.99
Long delay free recall	12.38 (2.84)	11.73 (2.91)	10.88 (.63)	1.64	2,77	.20		.61	.99
Long delay cued recall	12.81 (2.48)	12.31 (2.66)	11.15 (3.47)	2.21	2,77	.12		.79	.99
Recognition									
Long delay recognition	15.19 (1.17)	14.69 (1.12)	14.15 (2.11)	2.22	2,77	.06		.42	.92
Long delay choice	15.96 (0.2)	15.92 (0.1)	15.92 (0.27)	2.98	2,77	.36		.01	.95
Errors									
Repetitions - all trials	2.12 (3.88)	2.58 (1.86)	5.31 (4.22)	5.41	2.77	≤ .006[a]	C > A, B	.04	.95
Intrusions all trials	1.11 (1.34)	1.19 (2.09)	3.04 (3.82)	4.09	2.77	≤.02 [a]	C > A, B	.07	.99
False positives	1.19 (2.95)	0.96 (1.51)	2.61 (4.17)	2.31	2.77	.11		.02	.95

[a] Statistically significant p ≤.05. [b] A, Control; B, Trauma Exposed; C, PTSD; groups differ significantly (p ≤.05).

3.5.1.2. CVLT learning curve.

Data of the three groups learning curve over the CVLT trials was plotted and Figure 1 illustrates a learning curve pattern. It can be seen that the PTSD group demonstrate a similar pattern to the other groups although they have lower mean recall per trial.

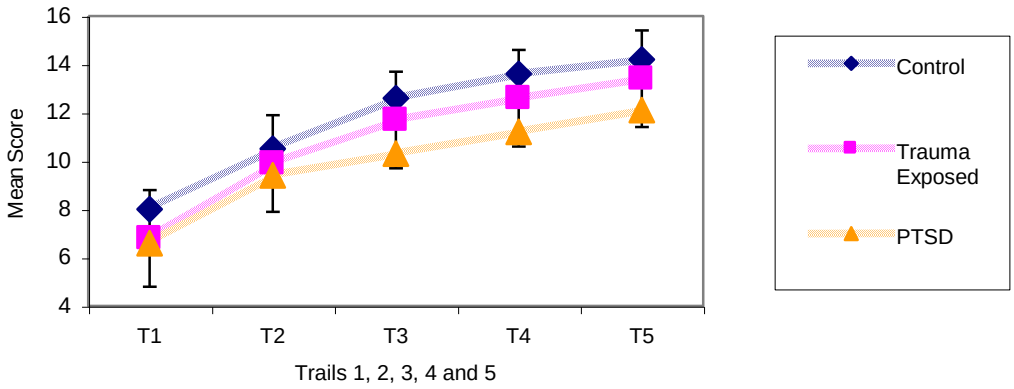

Figure 1. CVLT Trial 1-5 Mean Scores

3.5.1.3. CVLT - List B.

List B is the interference trial presented on completion of the five trials of List A. One-way ANOVA analysis revealed a significant difference between the three groups. Post hoc Tukey tests revealed that the Control group had significantly greater recall of List B words than the TE exposed group ($p = .04$) but not in comparison to the PTSD group.

3.5.1.4. CVLT - cued and free recall.

This element of the CVLT consisted of 4 components of recall tasks, short delay cued and free recall and long delay cued and free recall. One-way ANOVA analysis was not significant for any condition.

3.5.1.5. CVLT - recognition and forced choice.

ANOVA data analysis was not significant for long delay forced choice and missed significance for long delay recognition.

3.5.1.6. CVLT- errors.

One-way ANOVAs were conducted on error data. For the number of repetitions over all trials there was a significant difference between groups. Bonferoni-corrected (at alpha level) Tukey post hoc comparisons revealed that the PTSD group indicated a significantly higher number of repetitions recorded by the PTSD group in comparison to the control group ($p = .002$) and TE group ($p = .009$). A significant finding was also observed for the number of intrusions for all trials. Post hoc comparisons revealed that the PTSD group experienced significantly more intrusions than either the control ($p = .03$) or the TE group ($p = .04$). There was no significant difference between the number of false positive responses reported between the groups. This analysis is reported in Table 3. When controlled for depression the group effect was no longer significant ($F(1,74) = .14$ n.s.). This suggests that the increase error performance of the PTSD group, can be attributed to higher levels of depression.

3.5.1.7. Summary of CVLT data analysis.

Post hoc testing on the CVLT trials 1, 3, 4, 5, and the total scores of Trials 1-5 indicate the PTSD demonstrated a significantly poorer performance than participants in the Control group. The PTSD group were found to have experienced a greater number of word repetitions and intrusions over all trials than the Control and TE groups. Interestingly, the TE group were observed to recall fewer items on the intrusion trail of List B than the Control group. However, in the overall analysis, after controlling for depression there was no longer a group effect for any condition.

3.5.2. Memory - Visual

3.5.2.1. Shum Visual Learning Test.

For the Shum computer task, a MANOVA with trial (SVLT 1-3) as within subjects factor and group as between subjects factor revealed only a significant main effect for trial

($F(2,74) = 69.99$, $p < .001$, partial $\eta 2 = .65$) but no effect for group $F(1,75) = 0.48$, n.s.) and no trial by group interaction $F(4,150) = 0.84$, n.s. Post hoc simple contrasts revealed that the last trial was significantly higher than the two previous trials, which indicates a learning effect across groups. Table 4 shows means and SD per group and results of the one-way ANOVAs per subtest. Of note is that the controls showed a tendency to a higher learning index than both trauma exposed groups. ANOVAs were conducted to check for differences in the Total Trial Score, Recognition after Interference, Long Delay Recognition and the Learning Index and no significant group differences were found. Table 4 illustrates the visual memory data.

3.5.2.2. Rey Osterrieth Complex Figure Task.

One-way ANOVA analysis indicated a significant group difference in the recall of the Rey Osterrieth Immediate and Delayed tasks. Post hoc Tukey tests revealed a difference between the Control and PTSD ($p = .04$) and the TE and PTSD group ($p = .04$). There was no significant difference between the Control and TE groups ($p = .34$). For the delayed recall task there was a significant group difference between the Control and the PTSD group ($p = .02$) and the TE and the PTSD group ($p = .001$). There were no group differences between the Control and the TE group ($p = .39$). After controlling for anxiety and depression by ANCOVA analysis, the Rey Immediate Recall condition significance persisted for depression $F(2,72) = 3.86$, $p = .05$ but not for anxiety, $F(2,72) = .58$ n.s. Similarly, in the Delayed Recall condition when controlling for depression, $F(2,72) = 3.09$, $p = .05$, significance was maintained but not for anxiety, $F(2,72) = .40$, n.s. This suggests that findings in immediate and delayed recall memory are independent of depressive symptoms for the PTSD group.

Table 4

Neuropsychological Data - Visual Memory - SVLT and The Rey Osterrieth Complex Figure Test

Visual Memory	C *M* (S.D.) (A)	TE *M* (S.D.) (B)	PTSD *M (*S.D.) (C)	F/χ^2	df	p [a]	Tukey [b]	*d*	Observed Power
SVLT									
Total Trails 1–3	18.69 (4.87)	19.88 (3.47)	18.96 (4.11)	.58	2,77	.56		.51	.98
Recognition after interference	06.0 (0.19)	05.85 (.30)	05.96 (.22)	.79	2,77	.45		.06	.07
Long delay recognition	06.35 (.39)	06.57 (.27)	05.85 (.36)	.29	2,77	.74		.30	.64
Learning Index	01.84 (.66)	01.52 (.61)	01.52 (.54)	2.39	2,77	.09		.15	.20
Rey Osterrieth Complex Figure									
Recall *(immediate)*	21.31 (6.15)	24.04 (7.82)	16.21 (8.21)	8.42	2,77	$\leq$.001 [a]	C < A, B	3.24	1
Recall (*delayed)*	20.90 (6.21)	23.63 (7.53)	15.35 (8.36)	7.39	2,77	$\leq$.001 [a]	C < A, B	3.45	1

[a] Statistically significant p <.001.

[b] A, Control; B, Trauma Exposed; C, PTSD; groups differ significantly (p $\leq$.05).

3.5.3. Attention

In the domain of attention participants completed two tasks the Trail Making Test A and the Digit Symbol copy task. The results from both of these tests were not significant. Table 5 illustrates these data.

3.5.4. Executive Function

Elements of executive function were tested using the Part B of the Trail Making Test, the FAS and Animals tasks. On univariate analysis the FAS test was significant. Post hoc Tukey test proved significant group differences between Control and PTSD ($p = .007$) and TE and PTSD ($p = .04$). There were no group differences between the Control and TE groups ($p = .78$). ANCOVA was not significant for depression, $F(2,74) = 7.13$, n.s. or anxiety $F(2,74) = .19$, n.s. This suggests the levels of depression and anxiety on this PTSD group influences their ability on the FAS task of verbal fluency (lexical).

The Trail Making Test, Part B demonstrated a non-significant trend whilst the verbal fluency test of Animals was not significant for group differences. These data are highlighted in Table 5.

Table 5

Neuropsychological Data - Attention and Executive Function

Attention and Executive Function	C *M* (S.D.) (A)	TE *M* (S.D.) (B)	PTSD *M* (S.D.) (C)	F	df	p [a]	Tukey [b]	*d*	Observed Power
Attention									
Trail Making - Test A (*seconds*)	28.54 (12.06)	28.00 (9.69)	32.27 (13.55)	2,77	.99	.37		1.90	1
Digit symbol (*seconds*)	57.27 (13.24)	52.73 (12.96)	50.50 (12.65)	2,77	1.84	.17		2.82	1
Executive Function									
Trail Making - Test B (*seconds)*	51.96 (20.95)	53.62 (20.99)	67.08 (30.04)	2,77	3.0	.06		6.77	1
FAS Test	53.04 (10.01)	51.04 (11.01)	43.79 (11.05)	2,77	5.4	$\leq$.006[a]	C < A, B	3.97	1
Animals	60.23 (11.61)	58.92 (11.55)	56.96 (11.32)	2,77	.53	.59		1.34	1

[a] Statistically significant p $\leq$.05.

[b] A, Control; B, Trauma Exposed; C, PTSD; groups differ significantly (p $\leq$.05).

3.6. Group Differences in Questionnaires

3.6.1. Everyday Memory Questionnaire (EMQ; Sunderland et al., 1984).

Having log transformed data, one-way ANOVA univariate statistics indicated a significant difference between the groups. Post hoc Tukey tests showed group differences between the Control and PTSD group (p = .001) and the TE and PTSD group (p = .001). There were no differences between the Control and TE groups (p = .99). ANCOVA analysis demonstrated a lack of significance when controlling for depression F(2,72), = 2.15, n.s), but did show significance for anxiety F(2,72), = 5.25, p = .02) . This result suggests that depression has an impact on the individual when self-reporting their memory concerns, whereas levels of anxiety do not. These data are illustrated in Table 6.

Table 6

The Everyday Memory Questionnaire (EMQ) Data

EMQ	C *M*(S.D.) (A)	TE *M*.(S.D.) (B)	PTSD *M*(S.D.) (C)	F	df	p [a]	Tukey [b]	*d*	Observed Power
Raw Scores	61.96 (4.42)	64.08 (5.12)	109.62 (7.38)	2,77	21,57	.001	C > A, B	21.98	1

[a] Statistically significant p <.001.

[b] A, Control; B, Trauma Exposed; C, PTSD; groups differ significantly (p <.001).

3.7. Correlation Analysis – PTSD severity

To ascertain the relationship between the severity of PTSD (as measured by the PDS), scores of the EMQ, and to limit the number of statistical tests performed, correlations were examined for the PTSD sample on those tasks where group differences had been detected. Participant's HADS scores were controlled for using 1st order partial correlation (r = .592, p = .001; value of variance R^2 = 8.3%).

For the CVLT, the severity of PTSD was negatively correlated with the total learning score, Trials 1-5 (r = -.209, p = .03). This suggests a relationship between a high score on the PDS symptom severity for PTSD and a low score on total learning across trials 1-5. No other correlations were found on the CVLT data. No correlations were found on the Rey Complex Figure immediate and delayed conditions or for the EMQ. For the FAS task a significant negative correlation was observed (r = - 341, p = .001). This suggests that the higher the PDS symptom severity score the lower the individual's score for this test.

3.8. Influence of Medication

A number of individuals were taking medication and the significant result on the Chi square analysis justifies further control for medication. T-tests were conducted for the PTSD and TE groups, comparing medicated vs. un-medicated, on those neuropsychological tests that proved significant, i.e., the CVLT for all trials, the FAS, the Rey Osterrieth Complex Figure immediate and delayed recall. There were no significant results for the CVLT total lists, t(50), = 1.64, n.s. However, there was a difference for the FAS, t(50) = 2.38, p = .02), the Rey immediate recall, t(50) = 3.53, p = . 001) and the Rey delayed recall, t(50) = 3.76 = p.001) indicating that possibly medication had an effect on neuropsychological test performance as those not taking medication had greater recall.

Discussion

4.1. Aims of the Study

The purpose of this study was to investigate neuropsychological functioning in different domains in PTSD using standardised clinical assessment tools. The study utilized a recently developed computerised visual memory task that, as far as can be ascertained, has not been evaluated before in a PTSD population.

By allocating three groups of participants, the study aimed to determine if differences in neuropsychological functioning are observed between individuals with PTSD

symptomatology, trauma exposed participants without PTSD and non-trauma exposed controls. The potential impact of selected confounding variables was addressed. For example, adequate statistical power and mood disorders. In addition, the mixed trauma, non treatment-seeking sample were matched for age and gender and no significant differences were observed on a measure of IQ between the groups.

4.2. Research Findings

4.2.1. Influence of depression and anxiety

Many of the participants in the PTSD sample in this study had moderate to high self-reported symptoms of depression and anxiety as indicated by their HADS scores. PTSD is associated with high co-morbidity of psychiatric disorders and there is evidence to suggest that depression influences tasks requiring cognitive effortful processing and can influence memory encoding and organisational strategies (Veiel, 1997). Of particular relevance is a study by Otto, Bruder, Fava, and Delis (1994) who noted that that depressed participants without PTSD performed below the normal range on the CVLT.

In the findings of this study, covariance analysis demonstrated that anxiety and, or, depression negatively influenced cognitive performance, specifically in domains measuring aspects of executive function and verbal memory tasks. Thus any cognitive deficits in these areas may be attributed to mood disorder. Conversely, the strength of mood disorder did not entirely account for visual memory deficits where cognitive performance was found to be independent of depression (but not anxiety). The implication of these findings more clearly highlights the potential association between PTSD, visual memory deficits and fear learning and is discussed in a later section.

Interestingly, Miller and Chapman (2001) have advised against the use of ANCOVA and recommend caution when interpreting covariance data when the covariate (i.e., measures of depression and anxiety levels) differ between experimental groups (i.e., PTSD vs. non PTSD groups), as controlling for differences in this instance can

underestimate the actual significance level of the dependent measure. Nevertheless, the findings of this study cannot rule out the impact of mood disorder on cognitive function and the use of ANCOVA is in common with other studies that have attempted to ascertain the influence of depression and anxiety (e.g., Johnsen et al., 2007).

The relationship between PTSD and depression in cognitive test performance suggests the possibility that intrusive memories may engage task related resources and leave fewer cognitive resources available for the individual with PTSD to complete neurocognitive tests. This suggestion follows the specific findings of depression and its relationship to PTSD as reported by Johnsen et al. (2007) and Vasterling et al. (1998). Furthermore, the probable overlap in neuropsychological findings in depression and PTSD is not unexpected given that the limbic system is implicated in emotion processing. There is undoubtedly a need for the interacting effects of PTSD and mood disorder in particular, to be clarified. It is therefore considered meaningful to construct a group of participants with PTSD where anxiety and depression has been controlled. An alternative approach to research i.e., to examine sub-groups of PTSD diagnosed individuals with and without depression and anxiety would undoubtedly represent a challenge to obtain adequate sample sizes to conduct appropriate analysis. Moreover, those studies that have included clinical comparison samples have reported mixed findings and less than clear patterns of PTSD versus other diagnoses and cognitive impairment (Gil et al., 1990; Zalewski et al., 1994). Whilst some studies have revealed no evidence of correlation between cognitive performance and depression in PTSD samples (e.g., Golier, Yehuda, Lupine, Harvey, Grossman, & Elkin, 2002; Stein et al., 2002); other studies specifically examining different groups of non-depressed and depressed participants with PTSD have failed to find differences on cognitive measures between the groups (e.g., Vasterling, Rogers, & Kaplan, 2000; Vasterling et al., 2002).

In respect to clinical implications, findings in the literature remain sufficiently mixed to demonstrate a continued lack of clarity. The employment of strategies to examine the

influence of co-morbidity whilst taking into account the associated degree of cognitive impairment in PTSD remains paramount in clinical practice and is an area of research where methodological issues and the interpretation of findings need to be considered in context.

4.2.2. Cognitive impairment in PTSD.

In line with the predictions, the PTSD group demonstrated significantly lower scores on the individual and total list learning scores for the CVLT and higher repetition and intrusion errors were made in comparison to the control groups. Participants with PTSD also showed significant impairment of immediate and delayed visual spatial memory recall for complex figuration as compared to the control groups. In addition, the PTSD group performed significantly worse on the FAS test of verbal fluency compared to the other groups.

However, there were no significant differences in measures of free and cued recall on the CVLT measure of verbal memory across all groups. There were also no group differences on measures of visual memory based on the SVLT Chinese figure task. However, there was a significant learning effect across all groups and the control demonstrated a trend towards a higher learning index than both the trauma-exposed groups.

No significant findings were observed in the Trail Making Part A, Digit Symbol Coding (tests used to assess attention), Trail Making Part B or the Animal verbal fluency task (tests of executive function).

In the PTSD group, the severity of PTSD was found to negatively correlate with one measure of verbal memory, the CVLT measure of total list learning, and one measure of executive function, the FAS. This finding implies that patients with higher degrees of symptom severity perform less proficiently on tasks assessing list learning and verbal fluency.

In terms of the Everyday Memory Questionnaire, as predicted, the PTSD group differed significantly in comparison to the other groups on higher self-rated scores of failure in everyday memory. The degree of memory impairment on CVLT list scores and repetition errors were accounted for by mood disorder symptoms.

The exception to this was the finding on visual spatial recall for complex figuration, where the PTSD group performed significantly poorer compared to the control groups, independent of mood measures. Contrary to the prediction no deficits in the domain of attention were observed. In the following sections, the findings will be considered in more detail by cognitive domain.

4.3. Unique visual memory impairment in PTSD?

The findings on visual memory in this study are mixed. On one hand, in a visual task that taps into perceptual organisation, visuo-constructional ability and visuo-spatial memory (Rey-Osterrieth) revealed a significantly impaired performance in PTSD that seems to be unique as it cannot be accounted for by comorbid depression. On the other hand, in a new visual task that was hypothesized to tap into complex visual pattern recognition and visual memory, no effects were found. Below these contradictory findings are discussed.

Contrary to expectation, the general performance of the PTSD group did not differ significantly from the other groups in aspects of visual memory as measured by the SVLT. All groups demonstrated a learning effect and one interesting feature that emerged was the control group's higher learning index when compared to both the TE and PTSD groups. There may be a number of reasons for the slower learning rate in the PTSD and TE groups. In the traumatic brain injury (TBI) literature, there are reports of slower learning in individuals, with TBI both on the SVLT task (Shum et al., 2000) and other visual patterned stimuli (Hannay, Levin, & Grossman, 1979; Reid & Kelly, 1993). Further, Eadie and Shum (1995) suggest that the SVLT is sensitive to localised damage to the right cerebral hemisphere. However, brain injury was an exclusion criterion for this study, therefore the

absence of specific lesions could explain why the SVLT was not statistically significant in this PTSD population.

Individuals with PTSD were not found to be different to controls on the retention after interference and delayed retention indices of the SVLT. These findings are unexpected as patients with PTSD have been consistently found to be susceptible to deficits in retroactive interference (e.g., Vasterling et al., 1998; 2000). Although the SVLT is sensitive to right cerebral hemisphere damage, it is speculated that the test may not be sensitive enough to detect group differences in this particular sample, either due to statistical power (although visual memory differences were noted in the Complex Figure Task) or because of the complexity and subtlety of neurological deficits in a mixed trauma PTSD population. Alternatively, perhaps PTSD influences different aspects of visual memory and the Complex Figure and the SVLT measures discrete but separate areas. Overall, the computerised SVLT methodology is an interesting tool that has the potential to advance neuropsychological measurements and refine the assessment of visual memory function.

Concerning the Rey Complex Figure task, the significant results from this study support the findings of others (Bremner et al.,1993; Vasterling et al., 1998) and suggest that patients with PTSD present a pattern of cognitive dysfunction in the performance on complex figure tasks that is characterised by alterations in both delayed and immediate recall. Furthermore, these results were independent of depressive symptoms and this has implications for understanding the importance of visual memory for fear learning.

The findings may be in line with studies that relate visuo-spatial deficits in PTSD to (potentially pre-trauma) hippocampal dysfunction (Gilbertson et al., 2007). The hippocampus, a brain structure in the medial temporal lobe with anatomic connections to the amydgala and prefrontal regions, manages and integrates explicit memory functions in the appropriate spatial and temporal context that are under conscious recall. It is also particularly sensitive to the encoding of the context associated with a traumatic experience. It is because of the hippocampus that a stimulus can become a source of conditioned fear,

as can all the objects surrounding it, and the location in which the trauma occurred. It has been shown that patients with hippocampal damage show impairment in both the immediate and delayed recall of the Rey-Osterrieth Complex Figure (Bohbot, Kalina, Stepankova, Spackova, Petrides, & Nadel, 1998) suggesting that the right hippocampus is linked to learning in visuo-spatial memory tasks.

Given that PTSD has been conceptualized as a failure to naturally extinguish fear responses conditioned during trauma exposure (Pitman & Delahanty, 2005), especially hippocampus-dependent impairments in contextual fear learning, the significantly impaired performance by the PTSD group on the Complex Figure visual spatial reconstruction may implicate visuospatial impairment is a risk factor for PTSD. This is supported by findings that flashback memories are associated with selectively impaired performance on visuo-spatial tasks (Hellawell & Brewin, 2002) and lends support to Brewin's neuroscience model (Brewin, 2001) and is supported by the above-mentioned findings of Gilbertson et al. (2007).

4.4. Verbal Memory

Consistent with previous findings from researchers who have used the CVLT (Gilberston et al., 2001; Lindauer, Olff, van Meigel, Carlier, & Gersons, 2006; Sachinvala et al., 2000) and recent meta-analyses (Brewin et al., 2007; Johnsen & Asbjørnsen, 2008), this study observed that the PTSD group has significantly lower scores on individual and total list recall scores for the CVLT and higher repetition and intrusion errors were made in comparison to the control groups. There were no differences in level of IQ as measured by the NART across all groups, suggesting the deficits are not attributable to IQ. However, after co-variance analysis these differences were attributed to the high levels of depression suffered by the PTSD group supporting the findings of Johnsen et al. (2007).

Although Brewin's meta-analytic findings report verbal memory is consistently worse than visual memory, and that these differences could not be explained by potential

confounding factors, he does not comment on whether the neurological deficits predate the trauma or if they are a result of PTSD (Brewin et al., 2007). This point is discussed in a later section.

In this study, no impairments were observed in either the short or long delay free recall, or the short or long delay cued recall, recognition trials or interference trials. The findings are contrary to the positive results reported by Jelinek et al. (2006). Although similar in diagnostic methodology (i.e., they used the PDS to ascertain PTSD symptoms and a mixed trauma sample) Jelinek used different neuropsychological measures to the CVLT. Johnsen and Asbjørnsen (2008) note in their meta-analysis that studies using the Wechsler Memory Scale (Wechsler, 1987) and the Rey Auditory Verbal Learning Test (Lezak, 1985; Rey, 1941) displayed stronger effect sizes than the CVLT. One implication of this is the CVLT might not have detected subtle deficits if they were present in this sample.

An advantage of using the CVLT is that the long delay forced choice task can also be used as a measure of motivation. Scores were very high on all groups in this subsection, and this can be interpreted as showing a high level of engagement and good motivation throughout the task and groups. Overall, the findings suggest that the groups were using effective learning or organizational strategies (Sternberg & Tulving, 1977) during encoding but depressive symptoms significantly influenced their performance. These findings concur with those reported by Johnsen et al. (2008) who focused on verbal memory in refugees using the CVLT, and reported significant differences on learning and recall and attributed the differences to depressive symptoms.

4.5. Attention

The study did not find any deficits in any of the measures of attention. There are limited studies that focus specifically on attention in PTSD as tested by neutral (i.e. non trauma-related) neuropsychological tasks. Most studies encompass attention tasks

alongside other cognitive tests (e.g., Lindauer et al., 2006; Sachinvala et al., 2000; Stein et al., 2002; Vasterling et al., 2002). As poor concentration is a characteristic of PTSD and in agreement with other findings (e.g., Sutker, Vasterling, Brailey, & Allain, 1995) it was expected that there may be some differences on working memory and sustained attention similar to those reported by others (e.g., Jenkins, Langlais, Delis, & Cohen, 2000; Vasterling et al., 1998; 2002). However, no impairment was observed between any of the groups on visual scanning and sustained attention, perhaps the tests selected lacked the necessary complexity to detect significant deficits. As the CVLT contained a measure of motivation it is unlikely that this was a key issue. It may be that PTSD patients can concentrate in a focused way, but overall their resources are limited unless they allocate them specifically and for a limited time, which these tests may not have picked up.

The use of Event-Related Potential (ERP) studies provides an opportunity to understand attentional processing in PTSD more thoroughly. A meta-analytic review of ERP studies (Karl et al., 2006) using the oddball paradigm (in which participants detect, amongst a series of standard stimuli, an infrequent deviant one) suggested that novel distracting stimulus may play a role in directing attention allocation in PTSD. This is consistent with the clinical presentation of PTSD and with information-processing biases towards vague or potentially threatening stimuli. Attentional processes were narrowly measured in this study; it would be useful if future research assessed the numerous domains of attention (e.g., Mirsky, Anthony, Duncan, Ahern, & Kellan, 1991) to obtain a more complete profile and gain additional information from ERP or neuroimaging data.

4.6. Executive Functioning

In agreement with other research this study found impaired measures of lexical verbal fluency (e.g., Uddo, et al., 1993; Yehuda et al., 1995). Interestingly there were no differences between the groups for semantic fluency indicating the PTSD group found lexical fluency more difficult than semantic fluency. On the other hand, these tests load on

to many aspects of executive function for example, verbal knowledge, expressive language, reading-writing and abstract cognitive operations (Sbordone, 2000). Therefore we are unable to assume specific functional deficits in this sample population.

The findings on cognitive set shifting (Part B, Trail Making) missed significance. Gilbertson et al. (2001) and Stein et al. (2002) have published significant data using this test whilst others (e.g., Gil, Calev, Greenberg, Kugelmass, & Lerer, 1990; Levy, 1988) have reported no differences. It is unclear why the disparity across studies exists, although it is reasonable to suspect that individual characteristics within the sample contribute heavily in this regard. As conventional executive function tests are inclined to be basic and underspecified in terms of the cognitive processes that they engage (Burgess, 1997) they may lack the sensitivity to detect dysfunction in different groups. Continuing research may help fractionate the components of the executive system in PTSD, identify the complexity of functions and verify the neuroanatomical correlates.

4.7. Self Reported Memory Failure

Patients with PTSD symptomatology reported significantly greater difficulty with memory failure in tasks of everyday function as measured by the EMQ questionnaire. Findings suggest that subjective perception of memory failure reflects objective memory impairment in CVLT list learning and visual spatial memory. However, there is also a lack of consistency between self-report of memory failure and test performance for recall and recognition memory. This could be because laboratory tests are inadequate indicators of everyday memory problems, or it may be linked to the absence of attention deficits in dedicated tests but that everyday memory may well be dependent on lack of attentional resources. Another possibility is that subjective memory difficulties may be provoked by a variety of factors including depression or anxiety. These findings have important functional consequences but the relationship between tests and everyday memory problems in real life is far from clear. Impairments reported by patients need further investigation to aid our

understanding, not only for memory assessment per se, but in order to develop compensatory memory techniques for those patients who lack appropriate strategies.

4.8. Sample Characteristics

The reasons for the lack of findings in the domains of attention and particularly in the CVLT verbal memory long delay cued and both free recall conditions is uncertain. Indeed, the findings on all aspects of verbal memory, particularly when controlling for depression, is not in line with the findings of recent meta-analytic reviews (e.g., Brewin et al., 2007; Johnsen & Asbjørnsen, 2008). The following section discusses, perhaps the most obvious explanation for the differences between the findings reported and those in the literature, which is concerning the characteristics of the sample.

The sample demographic characteristics of participants were representative of a cross section of people reflecting a diversity of recruitment. There were no statistical group differences on education, IQ level, marital status or alcohol intake. As expected, the groups differed significantly on measures of anxiety, depression and medication.

Deficits reported in attention, learning and memory in other PTSD literature (for example, Gilberston et al., 2001; Sachinvala et al., 2000; Vasterling et al., 1998; 2000) reveal some differences from the sample groups they recruited in comparison to this study. This sample showed heterogeneity of traumatic experiences within the PTSD group, the studies mentioned previously have investigated homogeneous trauma samples of predominantly male war veterans. They may have more severe comorbidity or greater impairment than this sample and possibly negative issues that impact after the trauma, such as lack of social support and additional life stress (Brewin et al., 2000). Thus limiting the generalisability of their findings to populations other than war veterans.

Another factor is the age range (50-55 years) of the above studies; this study reports a slightly younger mean age of 48 years. Further, the average education range reported

by the previous literature was between 10-12 years, whereas in this study the average number of years in education is slightly higher at 13.66 years.

4.9. PTSD Severity

Some of the cognitive impairments (i.e., total list learning and the FAS) did negatively correlate with PTSD severity supporting the earlier findings of Lindauer et al. (2006); Sachinvala et al. (2002) and Vasterling et al. (1998). This suggests that patients with higher degrees of symptom severity perform less proficiently on tasks assessing list learning and verbal fluency. Those researchers that do investigate a correlation between symptom severity and cognitive deficits have consistently reported a link with symptom severity and impairment although the specific domains or test area findings are varied. Moreover, these associations do not explain whether a similar link exists between the stressor severity and the specific cognitive difficulties.

4.10. PTSD or Trauma Exposure and Cognitive Impairment

To disentangle whether it is PTSD or trauma exposure that can cause cognitive problems, valuable evidence is provided by Gilbertson et al. (2006) in a study of combat veterans and their identical non-combat exposed twin. By comparing the performance of veterans with and without PTSD the consequences of combat exposure and inherited risk factors (that may increase or decrease the disorder), Gilbertson was able to determine that the identical co-twin of PTSD veterans, who did not have combat exposure or PTSD themselves, showed a similar pattern in poor verbal memory. They proposed that better neuropsychological functioning in these areas provided pre-existing resilience when veterans were later faced with traumatic events.

In this sample, high co-morbidity means that some cognitive difficulties may be related to other psychiatric disorders and the absence of significant global deficits further complicates any interpretation. Not all forms of trauma or PTSD are identical in the kinds

of neuropsychological symptoms they bring on and many different types of trauma can result in PTSD. Co-morbidity confounding factors of the disorder may change over time (McNally et al., 1997). If this is the case then the duration between the traumatic event and testing may be critical and physiological factors such as sensitivity of the sympathetic nervous system and atypical cortisol release may influence test performance.

4.11. Methodological Limitations and Strengths

One limitation of this study was the reliance upon self-reported symptoms on the PDS to establish PTSD symptomatology. Whist this measure has good reliability and validity and has been used in other studies (e.g., Sachinvala et al., 2000) the most common measure used in the literature is the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). Furthermore, this study did not exclude participants with co-morbid psychiatric conditions and those taking medication. Whilst this may limit the conclusions that can be drawn, it reflects a realistic clinical sample.

This study draws on strengths including the use of clinically reliable assessment tools, the addition of a new visual memory test along with the statistical control of symptoms of depression and anxiety. It also considered the evaluation of self-report memory data and the inclusion of a trauma exposed group without PTSD.

4.12. Future Research

The preceding discussion suggests possible directions for further research in the neuropsychological differences in PTSD within the specific cognitive domains. Of course, many of the limitations of PTSD research stems from the very real difficulties in recruiting sufficient numbers of the relatively rare patients who are free of potentially confounding variables. Nevertheless, a more detailed examination of specific cognitive domains would also help to clarify remaining questions. For example, attentional problems have been indicated by some studies but not others. Additionally, the inclusion of a larger

neuropsychological test sequence to examine specific areas of executive function may produce more definitive results and a more consistent analysis of motivational factors would be helpful to reconcile the differences in the literature. Finally, a combined neuroscience focus on the mechanisms that lead to the expression of cognitive differences will no doubt enhance our understanding of the pathways that lead to PTSD.

4.13. Conclusion

In agreement with other literature, deficits in certain areas of cognitive function and not in others have been observed in this sample. Considering the tremendous variation in factors leading to PTSD it would be extraordinary if all forms of the disorder were associated with a consistent pattern of cognitive abnormalities.

Given the immense problems capturing multifaceted cognitive deficits, future research would benefit from multi professional involvement and controlled quantification of PTSD and related neurocognitive difficulties. This should remain a desirable goal for both clinicians and researchers.

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