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The relationship between symptoms of perceived trauma and verbal learning and memory deficits

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The Relationship Between Symptoms of Perceived Trauma and Verbal Learning and Memory Deficits

By

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Abstract

Recent empirical studies on individuals with Posttraumatic Stress Disorder (PTSD) report an association between PTSD and deficits in short-term verbal memory. While the previous studies utilised a global score of PTSD, the present study assessed the association of individual symptoms as a result of perceived traumatic events with verbal learning and memory deficits. In addition, the severity of the traumatic events was examined as well as gender, age, IQ, education and previous alcohol abuse and drug use. A sample of 148 adults reported to have experienced a traumatic event such as sexual or physical abuse as children, war veterans, or other severe traumas were recruited from agencies and communities in Western Australia. Participants were administered an adaptation of the Trauma Symptom Inventory (TSI: Briere, 1995) to assess perceived symptoms associated with PTSD: intrusive experiences, defensive avoidance, anxious arousal, dissociation, depression and anger. The vocabulary subscale of the Wechsler Adult Intelligence Scale-Revised (WAIS- R; Wechsler, 1981) was used as the indicator for the level of IQ, while alcohol abuse was assessed using the Alcohol Use Disorders Identification Test (AUDIT; Babor, de la Fuenta & Saunders, 1992). Verbal learning and memory was assessed using the Rey Auditory Verbal Learning Test (RAVLT; Spreen & Strauss, 1991).

A series of four hierarchical multiple regressions using sets of demographic, trauma severity and trauma symptom variables predicted four measures from the RAVLT: Trial 1, sum of Trials 1-5 for verbal learning and for verbal memory immediate and delayed recall trials.

Firstly, it was found that the demographic factors of gender, age, IQ, education, alcohol and drug use accounted significantly for 24 –32% of the variance for predicting verbal learning and 17 –24% for verbal memory. Secondly, when the set of trauma severity factors (ie number of traumas, distress and duration) were included in step 2 of the hierarchical multiple regressions, a significant increase was found of 4-6% of the variance in predicting verbal learning and 2-4 % for predicting verbal memory. Thirdly, by partially out the set of demographic variables and the set of trauma severity variables, the set of trauma symptoms significantly increased the prediction by 2-3% of the variance for verbal learning and 5-6% of the verbal memory. Specifically, the symptoms

of dissociation and anxious arousal contributed significantly to the prediction of immediate recall, while anxious arousal was the only significant trauma symptom for predicting delayed recall.

Verbal learning and memory deficits may have serious implication in a number of settings in particular, children's early academic performance, for those seeking therapy, and in the court-room. Assessing dissociative symptoms associated with trauma may be a useful strategy for assisting individuals in the treatment of trauma intervention. One further recommendation is made to assess specific trauma symptoms rather than a global PTSD score.

DECLARATION

" I certify that this thesis does not incorporate, without acknowledgment, any material previously submitted for a degree or diploma in any institution of higher education and that, to the best of my knowledge and belief, it does not contain any material previously published or written by another person except where due reference is made in the text."

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A special note of thanks go to my two children Lara and Paul who supported me, even though they didn't quiet understand what I was going through. I hope that my process has not put them off doing a thesis in their future studies!

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Background

Research with people exposed to extraordinary stressors has expanded in recent years, but little has focused on perceived trauma and symptoms associated with verbal learning and memory deficits. In the last two decades, numerous studies have been conducted on the physiological and psychological sequelae of Posttraumatic Stress Disorder (PTSD) (Rosen & Field, 1988). Recently, empirical research suggests that cognitive impairment in the form of verbal memory deficit is associated with PTSD in Vietnam veterans (Bremner, Scott, Delaney, et al. 1993; Uddo, Vasterling, Brailey & Sutker, 1993; Yehuda, Keefe, Harvey, Vasterling, et al. 1995); adult survivors of childhood abuse (Bremner, Randall, Scott, Capelli, et al. 1995; Stein, Hann, Vaerum & Koverola; 1999); rape victims, (Jenkins, Langlais, Delis & Cohen, 1998) and in Persian Gulf War veterans (Vasterling, Brailey, Constans & Sutker, 1998).

The essential feature of PTSD is the presence of a traumatic event. According to the Diagnostic and Statistical Manual of Mental Disorders 4th ed. (DSM-IV; American Psychiatric Association, 1994) a traumatic event (criterion A) for PTSD requires that a) the individual perceives the event as life threatening *or* a threat to the physical integrity of self or other and b) that the event invokes intense fear, helplessness or horror.

Clinical and research evidence shows that individuals can vary in the severity of their reaction to trauma ie. to each specific criterion of PTSD as set out by the diagnostic criteria from the DSM-IV (American Psychiatric Association, 1994). This raises the question as to which of the specific symptoms or group of symptoms may be associated

with verbal deficits. Research in this area has grouped individuals into PTSD and non-PTSD groups for preliminary investigation, however such groups would not be considered homogeneous as to symptomatology.

The early work on the role of the stressor in PTSD linked specific symptoms to the type of trauma, such as "war syndrome", "rape syndrome", or "battered wife syndrome". However, it is currently accepted that response to trauma has a generalised pattern regardless of the type of trauma (see below for trauma and PTSD symptoms). The literature on the types of traumas and subsequent development of PTSD exists from a range of groups: war veterans (Sutker, Uddo-Crane, & Allain, 1991; Yehuda & McFarlane, 1995) prisoners of war (Basoglu et al., 1994); subjects of terrorist attacks (Brooks & McKinlay, 1992); refugees (Carlson & Rosser-Hogan, 1991); holocaust survivors (Danieli, 1981); abused children (Craine, Henson, Colliver, & MacLean, 1988; Donaldson & Gardner, 1985; Greenwald & Leitenberg, 1990; Lindberg & Distad, 1985); sexual assault victims (National Victim Centre, 1993); victims of crime (ie house-breaking, robbery, assault) (Simpson, Morley, & Baldwin, 1996); victims of domestic violence (Astin, Lawrence & Foy, 1993; Kilpatrick & Williams, 1998; Mezey & Kaplan, 1997); witnesses to domestic violence (Kilpatrick & Williams, 1998), motor vehicle accident victims (Blanchar, Hickling, Taylor, Loos, 1995; Kuch, Cox, & Evans, 1996), medical patients (de Girolamo & McFarlane, in press) and victims of natural disasters (McFarlane & Papay, 1992).

History of the Diagnosis of PTSD

War veterans' reaction to combat was described in the early part of the century as "soldier's heart", "war neurosis", "shell shock" and "combat fatigue". The impact of the physiological arousal and psychological reactions to combat in some soldiers in World

Wars I and II led Kardiner (1941) to formulate the term "physioneurosis". The symptoms of physioneurosis included the physiological response of vigilance and sensitivity to the environment as threatening as if still present on the battlefield. Patients continued to experience symptoms after leaving combat and there appeared to be a lowered threshold to stimulation leading to a fright response. The psychological responses consisted of an altered sense of self, chronic irritability, startle reactions and explosive aggression. It was not until research with Vietnam veterans and the synthesis of the different syndromes that the concept of PTSD was refined and the DSM –III (American Psychiatric Association, 1980) first described the disorder. Numerous studies on war veterans have documented PTSD as a result of the war experience, including delayed onset PTSD (Van Dyke, Zilberg, McKinnon, 1985; Bonwick & Morris, 1996). As the DSM evolved, further modifications have been made. Other long term psychological disorders as a result of the war experiences often co-exist with PTSD, including alcohol abuse, generalised anxiety, panic disorder, major depression, domestic violence and interpersonal problems (Davidson, Kudler, Saunders, & Smith, 1990; Matsakis 1996).

Researchers often find a history of trauma in clients of mental health services (Brown & Anderson, 1991). In a study conducted by Jacobson and Richardson (1987) on 100 consecutive admissions to a psychiatric ward, 57% of cases reported that they had been victims of either sexual or physical abuse before admission. Mueser, Goodman, Trumbetta, Rosenberg, Osher, Vidaver, Auciello, and Foy (1998) reported exposure to at least 1 traumatic event in 98% of cases with severe mental illness in a sample of 275 patients. The rate of PTSD was 43%. Of particular interest however, only 3 of the 119 patients with PTSD (2%) had this diagnosis in their medical charts. The study suggested

that trauma symptoms and PTSD may be a commonly comorbid disorder with severe mental illness.

The Role of Trauma in the Diagnosis of PTSD

According to the DSM-III (American Psychiatric Association, 1980), a traumatic event (or the stressor of criterion A) was categorised as outside the range of usual human experience. The revised third edition of the DSM (American Psychiatric Association, 1987) continued with the same definition of a traumatic event. However, with the improvement of methodologies to assess the prevalence of traumatic events, it was found that this definition of a traumatic event was inadequate. Changes were established with the DSM-IV (American Psychiatric Association, 1994) that the traumatic event must be perceived by the individual as life threatening or a threat to the physical integrity of self or other, and that the event invokes intense fear, helplessness or horror.

Three major characteristics of PTSD have remained the same across the three recent editions of the DSM (ie re-experiencing, avoidance, and increased arousal) with the recent alteration that re-experiencing can be triggered by internal as well as external cues. The DSM-III-R dropped the acute/chronic distinction of PTSD. However, this has been reinstated in the DSM-IV with the acute stage of a duration now of less than three months rather than 6 months and the chronic classification greater than 6 months. The DSM-IV (1994) has introduced the new diagnosis of acute stress disorder (ASD) which applies to the immediate short term response to trauma of less than 4 weeks. If the trauma response symptoms are experienced after one-month, assessment for PTSD criteria is needed.

The immediate psychological response to trauma is with behavioural agitation, emotional experiences of anxiety, panic, numbing, cognitive disorganisation, and defensive processing such as dissociation (Shalev, 1996). These processes during the trauma and hours after the trauma develop to protect an individual from overwhelming affect. They are referred to as peritraumatic responses (Marmar, et al. 1994).

Acute stress disorder focuses on two types of symptoms, dissociation and emotional reactions during the four weeks after the traumatic event. The dissociative symptoms are present either while experiencing or immediately after experiencing the distressing event. At least three symptoms are required for the criterion B for a diagnosis of ASD and these include: 1) sense of detachment, numbing or absence of emotional responsiveness; 2) diminished awareness of surroundings; 3) derealisation; 4) depersonalisation; 5) lack of memory for significant aspects of the trauma. Criterion C requires at least three of the following: sudden fear or anxiety, hyperarousal, somatic symptoms, intrusive thoughts of the trauma, sleep problems, anger, despair or social withdrawal. In addition to the symptoms, a disturbance in social or occupational activities or the immobilisation of seeking assistance is also experienced.

The early symptoms abate with time (Foa, Steketee & Rothbaum, 1989). If an individual dissociates during the acute stage, it is considered to be a critical ingredient in later developing PTSD (van der Kolk, 1996; van der Kolk & McFarlane, 1996; van der Kolk, van der Hart & Marmar, 1996). There are a number of factors that may influence the persistence of symptoms from acute stress reaction to PTSD or other psychological disorders such as panic attack and major depression. These include biological factors, developmental level at the time of abuse, the severity and chronicity of the stressor, social context, and previous and subsequent life events (Carlson, 1997). In addition,

Smith and North (1993) have reported that deliberate and intended acts causing human distress, such as rape and assault, may have more psychological impact than natural disasters. However, it must be noted that adjustment to trauma is a natural process of human behaviour, and not all traumatic events develop into PTSD. On the other hand, individuals affected by PTSD have long term responses to the trauma, which may differ according to whether the trauma was a discrete event (ie.one event), or multiple events or situations. In general, chronic trauma symptoms may persist for years and often a life-time (Andrews, Crino, Hunt, Lampe & Page, 1996; Archibald & Tuddenham, 1965; Falk, Hersen & Van Hasselt, 1994; Fleming, et al. 1999; Zlotnick, Warshaw, Shea, Allsworth, Pearlstein & Keller, 1999).

Symptoms of Trauma and PTSD

According to the DSM-IV, individuals exposed to a traumatic event may have a constellation of symptoms that include; a) re-experiencing of intrusive recollections of the event involving images, dreams, flashbacks, thoughts or perceptions that relive the trauma, b) avoiding the stimuli associated with the trauma through numbing (dissociation) involving an inability to recall important aspects of the trauma, detached feelings, and avoidance of places, people, thoughts and activities related to the traumatic event and, c) an increased arousal that was not present before the event, that is, difficulty falling or staying asleep, hypervigilance, heightened responses, difficulty concentrating, irritability or outbursts of anger. A diagnosis of PTSD requires the presence of at least one or more symptom of re-experiencing, three or more symptoms of avoidance and two or more symptoms of increased arousal, all of which were not present before the trauma.

PTSD criteria are also classified according to the duration of symptoms: those lasting between more than one month and three months are classified as *acute PTSD*;

those lasting more than three months are classified as *chronic* PTSD. The onset of symptoms can also be delayed from up to 6 months to 30 years following the traumatic event.

Intrusive Re-Experience After a distressing event, most people become absorbed and engrossed with the event, re-experiencing the event over and over in their mind involuntarily. The intrusions may be experienced as flashbacks in the form of images or thoughts or nightmares. These intrusions are often accompanied by extreme distress both emotionally and physiologically, which can make the individual feel immobilised and unaware of the environment. According to Horowitz (1976), the purpose of the intrusive process is to provide meaning and integrate the emotions of the event. Most people develop tolerance for the emotions, while others are unable to integrate the event and develop avoidance and hyperarousal associated with PTSD. When traumatic events remain unprocessed, with time the intrusive experiences have adverse long-term biological changes with a host of symptoms such as avoidance, anxious arousal, more generalised triggers or reminders of the trauma. Briere (1995) notes that high levels of intrusive experiences may also be linked with previous psychological trauma.

Defensive Avoidance is a coping mechanism to manage and deal with the intrusive experiences as a form of protection to the individual to prevent being re-exposed to the traumatic event. Carlson (1997) describes avoidance in terms of cognitive, affective, behavioural and physiological responses. Since the trauma brings back the associated pain and the element of threat or danger, it is a natural process to try and avoid the distress. Cognitive avoidance includes consciously thought stopping or distortion of the event such that the event can no longer be remembered. Emotional numbing can be classed as affective avoidance whereby strong feelings are lost. Avoiding places, people

or watching television that resemble aspects of the trauma are common behavioural avoidance strategies.

Anxious Arousal is part of the reaction of the physiological autonomic system to a traumatic event. Trembling, nervousness, jumpiness and exaggerated startle responses or angry outbursts with problems falling asleep or staying asleep are typical behavioural symptoms of individuals experiencing anxious arousal. Cognitive symptoms of anxious arousal include excessive worrying of what might happen and fears of threat to self or others. The loss of trust in others and /or the security of their environment may also occur.

Assessment inventories and clinical interviews evaluating responses to traumatic events focus on the three classes of symptoms of intrusive experience, defensive avoidance and anxious arousal for a diagnosis of PTSD. Most research studies have focused on a global diagnosis of PTSD. In addition, other trauma symptoms associated with PTSD as per the DSM-IV include dissociation and anger and are considered important as possibly relevant for learning and memory problems. Although the symptom of depression is not included as part of the criteria of PTSD in the DSM-IV, a large proportion of participants in the studies of PTSD and memory also presented with depression. The research studies have been inconsistent, with some excluding these subjects with comorbidities while others included these subjects as variants of PTSD. Since depression has been reported to affect memory (See review by Burt, Zembar & Niederehe, 1995), it was considered important to include it as part of the trauma symptoms in relation to verbal memory.

Depression symptoms are common in individuals who experienced a traumatic event. Although depression is not included in the PTSD criteria of the DSM-IV,

according to Carlson (1997), depression may be a primary as well as a secondary symptom of PTSD. Typical features of depression include: depressed mood and depressive cognitions, including feelings of sadness and unhappiness, a perception of worthlessness, inadequacy and a view of the future as hopeless, with a tendency to have thoughts about death and dying.

Dissociation is part of the re-experiencing and avoidance criteria of PTSD symptoms (DSM-IV, 1994). Perry, Pollard, Blakley, Baker and Vigilante (1995) and Carlson (1997) strongly argue that this phenomena be assessed as a separate symptom. According to Perry et al. (1995), dissociation refers to disengaging from stimuli in the external world and attending to an "internal" world. Behaviours such as daydreaming, fantasy, depersonalisation, (ie. distortions in perceptions of the self), derealisation (ie. distortions in perceptions of objects or environment) and amnesia (ie. inability to recall events associated with the distressing experience) are examples of dissociation.

From the turn of the century, dissociation has been historically linked with trauma. The well documented works of Myers, Charcot, de la Tourette, Janet and later Freud and Breuer have helped develop the contemporary view of trauma and dissociative processes (van der Kolk, van der Hart & Marmar, 1996). However, during the development of the DSM-III, researchers and clinicians working on the classification of PTSD worked separately from those working on classifying dissociative disorders. According to van der Kolk, Weisaith & van der Hart (1996), there was no connection between the working groups on dissociation and trauma and hence the creation of a separate classification for dissociation.

Anger, aggression and irritability have been reported in association with experiencing a traumatic event. Victims of abuse or trauma have difficulty

understanding any meaning of the event or the injustice of the event, which can the lead to feelings of deep anger. Kardiner (1941) was the first to define the symptoms of trauma from war veterans and he documented explosive aggressive behaviours that were not part of the pre-war behaviour. He describes the aggression as "not deliberate, or pre-meditated.... ..always impulsive ,episodic and alternates with moods of extreme tenderness" (p. 97) (van der Kolk, 1996). Further research on anger as a response to trauma has been well documented with veterans with PTSD (Chemtob, Hamada, Roitblat & Muraoka, 1993); towards partners (Carrol, Rueger, Foy, & Donahoe, 1985); with anger related problems at work (Knight, Keane, Fairbank, Caddell & Zimering, 1984); and criminal assault (Lee & Rosenthal, 1983; Riggs, Dancu, Gershuny, Greenberg & Foa, 1992; Yassen & Glass, 1984).

According to Carlson (1997), behaviours, feelings and thoughts of aggression need to be clarified for different treatment strategies if they are in response to the initial, secondary or associated response to trauma. Aggressive behaviours may result from flashbacks, or from an inability to reduce tension and or as the result of a socially learned behaviour. The inability to regulate affect has been observed with abused infants and children who develop a disorganised pattern of attachment and are unable to use the caregiver to soothe them. Problems associated with dysregulation of affect include learning disabilities and aggressive behaviour towards self and others (van der Kolk, Pelcovitz, Roth, Mandel, Mcfarlane & Herman, 1996). The experience of anger is often described by victims of trauma as not under their control (Briere, 1995).

The expression of anger can take many forms of physical or verbal aggression towards someone or the self. Cultural, social, and biological factors influence the expression of anger. From clinical observations, it appears that males are more likely to

express anger towards others, while females are more likely to express self- directed aggression to gain some control in their lives (Carlson, 1997).

Prevalence of Trauma and PTSD

The prevalence of at least one lifetime traumatic event (TE) in community studies range from 39% to 87% of cases. Any comparison of rates of traumatic events need to be taken with caution because of the changes in criterion A for PTSD from the DSM-III-R (1987) to the DSM-IV(1994). The small number of relevant studies is summarised in Table 1.

Table 1.

The Prevalence of Traumatic Events (TE)

Studies	No of Participants	At Least one TE in their Life Span	Other
Breslau, et al. (1991)	1007 civilians in a Health organisation	39%	
Norris, (1992)	1000	69%	21% TE in the last 12 months
Resnick, et al. (1993)	4009AmericanWomen (> 18yrs)	69%	36% 1 out of 4 criminal events in life time
Kilpatrick, et al. (1987)	391 in South Carolina	75% victims of crime	24% Rape 13% Attempted Rape 18%Com Molestation 5% Attempted " 4% Sexual Assault 10%Aggrevated " 6% Robbery 45% Burglary
Kilpatrick, et al. (1992)	528 > 15yrs 400 treatment for PTSD, 128 non-treatment	87% at least one	64 % more than one event in their life time 75% Experienced their first traumatic event before 18yrs

The prevalence of PTSD in the general population is reported to range from 1% to 7%, but to be higher in specific high risk groups such as victims of rape, Vietnam veterans, firefighters and others who experience extremely stressful events. For such groups, the rates of PTSD can range from 20% to 40% (Breslau, Davis & Andreski, 1991; Card, 1987; Davidson & Fairbank, 1993). A number of studies on the prevalence of PTSD are summarised in Table 2. Similarly, the rates of developing PTSD need to take into account the sensitivity and specificity of the specific assessment instruments used as well as the changes in diagnostic criteria over time.

Table 2.

The Prevalence of Posttraumatic Stress Disorder in General Population Samples and Following Specific Traumas.

Study	No of Participants & Type of Participants	Developed PTSD
Helzer, et al (1987)	Epidemiologic Catchment Area (ECA)	After the trauma: Males 15% Females 16% Life time PTSD Males .5% Females 1.6%
Breslau, et al., (1991)	1007 Urban population	24% Life time prevalence of 9.3%
Kessler, et al (in press)	8,098 National Comorbidity Survey	General population 6.5% Females 48.4% Males 10.7%
Kulka, et al. (1990)	Vietnam Veterans	15% Current PTSD 30% Lifetime PTSD 11% Partial symptoms
Southwick, et al. (1993)	Desert Storm Veterans	9% 6 months after their return from the Persian Gulf
De Girolamo & McFarlane (in press)	35 studies of Vietnam Veterans	Range from 2% to 70%
Kilpatrick, et.al. (1987)	Crime Victims	19-75%
Yehuda, et al (1995)	Prisoners of war	47-50%Persistent and Chronic PTSD
Kilpatrick, et al. (1987)	Rape Victims in a community sample	57%
Kilpatrick, et al. (1992)	Rape Victims Non-victims	31% 5%
Rothbaum, et al, (1992)	Rape Victims	94% 2 weeks post rape symptoms 50% 12 weeks post rape
Resnick, et al. (1993)	4,009 AmericanWomen (> 18yrs)	17.9% Lifetime history of PTSD
McFarlane, (1992)	469 firefighters	16% Immediately following exposure < half of these symptoms gone 42 mths
Shore, et al. (1986)	Volcanic Eruption of Mt Saint Helen	3.6% Two Year follow-up 0%

Stress and Trauma

"Stress" has been used in the literature widely with a broad definition and it is important to differentiate conceptually between stress and trauma. For example, can one regard chronic stress as traumatic, or do the chronic effects of trauma constitute stress? From the literature above on the incidence of trauma and PTSD, it is clear that PTSD can occur after relatively ordinary events as well as extraordinary events under DSM-IV criteria. Some events can be perceived by some individuals as threatening or dangerous, while the same events may be perceived as difficult and challenging for others and as uneventful for still others. Examples of such events may include childbirth, a motor vehicle accident, or being trapped in an elevator. In addition, PTSD symptoms recover with time in the majority of people who have experienced a very distressing event.

The literature on stress over the decades has focused on the original homeostatic theory of H. Selye (1956). Selye defined stress in terms of a biological response he called the general adaption syndrome to a condition in which expectations were not congruent to the present or anticipated perceptions of the internal or external environment. Compensatory responses are part of the homeostatic system whether stress is beneficial (eustress) or harmful (distress). The general adaption syndrome consists of 3 phases: the alarm reaction, resistance and exhaustion.

Following from Selye's work, modern psychophysiological approaches to medicine examine the relationship between emotional dysfunction due to distress as a risk factor for medical illnesses. Hall (1999) reports that chronic stress or long term exposure to a stressor or perceived stressor involves a shift from anabolic (building up) processes to catabolic processes (breaking down) of the bodily functions and can

contribute to reproductive disorders, gastrointestinal problems, coronary-arterial disease, changes in immunocompetence and other problems. Stress, chronic stress and traumatic stress have distinct features. Chronic stress may have long term psychological impairment, but is differentiated from traumatic stress by a number of factors. Stress becomes traumatic when the response to an event is psychologically damaging such that cognitive beliefs are challenged, affect is narrowed, and habituation occurs before the stressor is relieved or that habituation will continue after the stressful event (Shalev, 1996). Traumatic stressful events resulting in the acute reaction phase may lead to the development of PTSD symptoms, which involve a complex process including neurobiological changes with a persistent hyperarousal condition, changes in cognitive schema as a result of the event which further affect social and interpersonal interactions with others such as trust and security. The trauma symptoms in turn require a specific number of symptoms and persistence of the psychological symptoms before a diagnosis of PTSD is made.

Stress and Memory

Since the turn of the century, research conducted into the area of the effects of stress has included its impact on memory. Studies on animals exposed to inescapable shock have indicated an association between stress and altered memory, including short-term memory deficits as well as decreased initiation of behaviour and symptoms of emotional disruption (Drugan, Ryan, Minor, & Maier, 1984; Seligman & Maier, 1967). For examples, inescapable shock (IS) is an environmental stimulus that can induce persistent biochemical changes in the brain and longstanding behavioural changes.

Sapolsky, Packan and Vale, (1988) hypothesised that high levels of glucocorticoids released during stress may have neurotoxic effects on the hippocampus

(See review by Keenan & Kuhn, 1999). This structure of the brain is involved in learning and memory, particularly declarative or explicit memory and spatial learning. Starkman, Gebarski, Berent, and Schteingart (1992) reported significant positive correlations between hippocampal formation volume and scores on a verbal memory test. The hippocampus also plays an integral role in the control of autonomic and vegetative functions of adrenocorticotropin secretions (Eichenbaum & Otto, 1992). Studies with patients with Cushing's syndrome, who have an increased activity of the hypothalamus-pituitary adrenal (HPA) axis with high production of cortisol, have reported deficits in concentration and memory problems in 66-83% of cases (Starkman, Schteingart & Schork, 1986). Cortisol is measured because it is one of the steroids from the adrenal cortex responsible for 95% of all the glucocorticoid activity. More recently, de Quervain, Roozendaal, Nitsch, McGaugh & Hock (2000) report consistent findings with elevated cortisol levels affecting declarative memory using a free-recall of verbal material, but not the overall cognitive function or the recognition memory. The participants received cortisone tables (25 mg) or placebo. Such results suggest that elevated glucocorticoids levels may have a negative impact in stressful conditions such as examinations, job interviews, combat and as witness in a courtroom.

A longitudinal study of healthy elderly people administered a battery of tests assessing memory attention and language in relation to measures of cortisol. Scores on measures of explicit memory and selective attention were significantly negatively correlated with high basal cortisol levels (Lupien, Lecours, Lussier, Schwartz, Nair & Meaney, 1994). Other studies with patients diagnosed with major depression (Rubinow, Post, Savard & Gold, 1984) and Alzheimer's disease (Issa, Rowe, Gauthier & Meaney, 1990), who also have elevated cortisol levels, have found similar associations between

cortisol and cognitive impairment in memory, but a strong causal relationship has been elusive. Kirschbaum, Wolf, May, Wippich and Hellhammer (1996) conducted two studies linking cortisol with impaired memory function in humans. The first study conducted exposed participants to a laboratory stress followed by a declarative memory test. The second study investigated the effects of administrating cortisol (10 mg) alone (ie without stress) compared to a control placebo group to examine procedural, declarative memory and spatial thinking one hour after administration. The results showed impaired performance in declarative memory and spatial thinking tasks but not in the procedural memory tests. Data from the MacArthur Studies on Successful Aging have also validated these findings. Seeman, McEwen, Singer, Albert and Rowe (1997) found that women who experienced a decrease of cortisol levels demonstrated improvements in memory performance, but this was not found amongst the men. The study suggests plasticity of the hippocampus in relation to cortisol and memory function. McEwen (1999) reviews the literature on animal models and humans in relation to stress and hippocampal plasticity. The two areas of the hippocampus affected by stress and involve structural plasticity are: 1) atrophy of dendrites in the CA3 region following repeated stress, 2) dentate gyrus granule neurons, which suppress neurogenesis, with both acute and chronic stress. Apart from the glucocorticoids affecting the plasticity of hypocampal neurons, excitatory amino acids and N-methyl-D-aspartate (NMDA) receptors are involved. McEwen speculates that PTSD and recurrent depressive illness may have distinct pathways of glucocorticoid hormones depending on the individual's experiences and reactivities. However, little is known of the neurochemical changes induced by trauma that may have occurred 10 to 20 years ago. The increase in neurochemical, autonomic and HPA reactivity may involve neuronal loss and atrophy of

the hippocampal structures. So far little is known of the causal mechanisms from adaptive plasticity of the hippocampus to permanent damage and atrophy. However, there is some suggestion that Cushing's syndrome maybe reversible with drugs. Recently a study by Starkman, et al. (1999) using transsphenoidal microadneomesctomy to decrease cortisol levels have shown reverses in human hippocampal atrophy and volume. Participants in this study who were younger had a greater increase in hippocampal formation volume than older participants. The age range of the 22 participants was 24 to 54 years. McEwen suggests that where atrophy leads to neuronal loss, the use of therapeutic drugs in the early stages of the trauma or recurrent depression may aid in the reduction of hippocampal damage and thus memory impairment.

Studies of stress resulting from trauma symptoms that lead to the diagnosis of PTSD have recently also resulted in increased interest with regards to possible biological changes. There is still questioning as to whether the immediate reaction at the time of the trauma is related to the long-term neurobiological and psychological changes or if there is an additional effect due to the factors associated with PTSD. PTSD is now understood to be a maladaptive process rather than a normal response to trauma because of these changes (van der Kolk, 1996)

Biological Changes with Trauma and PTSD and the Association with Memory

Since the inclusion of PTSD in the DSM, more recent studies have shown a relationship between the experience of trauma and changes in physiological and neurobiological systems. Biological findings have emerged to indicate that cases with trauma symptoms show changes that are quite different from the stress-response of earlier animal models and stress in humans. In addition, the biological changes seen with trauma victims who have developed trauma symptoms of PTSD are different from those

observed in other associated mood or anxiety associated disorders (Yehuda & McFarlane, 1995). Charney, Deutch, Krystal, Southwick and Davis (1993) proposed that a number of brain structures become dysfunctional due to the pathophysiology of PTSD. These include important structures of the limbic system, the amygdala and the hippocampus and also the locus coeruleus. In addition to the adaptation to stress, a number of physiological responses are activated as part of the enduring symptoms in PTSD. These changes affect the noradrenergic, dopamine, opiate, and corticotropin releasing factor neurochemical systems. Catecholamine and other neurotransmitter systems interact in a complex regulatory and compensatory manner in response to the effects of a traumatic experience (van der Kolk, Greenberg, Boyd, & Krystal, 1985).

Since the early documentation of the symptoms related to war veterans by Kardiner (1941), the changes of the psychophysiological response to reminders of the trauma as well as other neutral stimuli such as a loud noise have been reported. The central nervous system (CNS) becomes more sensitive and an autonomic reaction to "normal" non-threatening stimuli with fear is part of the known psychophysiological changes (Rosen & Fields, 1988; McFall, Murburg, Roszell & Veith, 1989). Experimental studies on the autonomic, sympatho-adrenal, and hypothalamo-pituitary-adrenal (HPA) systems have been conducted using white noise and combat sounds with veterans with PTSD and without PTSD. The results show that PTSD veterans have significantly higher subjective distress, skin conductance, heart rate, plasma catecholamines, ACTH and cortisol levels at baseline. In addition, the PTSD veterans exhibited an exaggerated responses to combat sounds in comparison to the control group on skin conductance, heart rate, plasma epinephrine and norepinephrine, but not ACTH (Liberzon, Abelson, Flagel & Young, 1999). Earlier studies have also shown the heightened autonomic response with

a diagnosis of PTSD (See review by McFall, et al. 1989). However, Liberzon, et al. also attempted to integrate the psychophysiological responses and the neuroendocrine levels with exposure to combat sounds, but this hypothesis was not supported.

More recently, the autonomic nervous system of combat veterans with PTSD and veterans suffering from PTSD and concurrent panic attack and a control group without PTSD or panic attack have been tested with sodium lactate infusion to test the reactions of their autonomic nervous system. The PTSD group without panic attacks reported flashbacks that were accompanied by high anxiety symptoms and low cortisol levels. The control groups did not differ on any of the measures, indicating that the changes found in the PTSD only group may be because of an extreme autonomic response (Jensen, Keller, Peskind, et al. 1997).

The hormones released during stress play a specific role and modulate the response of adapting to stress. Cortisol regulates hormones such as glucocorticoids and catecholamines through a feedback loop to the hippocampus, hypothalamus and the pituitary. However, with exposure to a traumatic event or perceived traumatic situation and the subsequent development of PTSD, the intrusive thoughts and experiences have been shown to be accompanied by changes to specific neuroendocrine hormones such as norepinephrine (NE), glucocorticoids, serotonin and endogenous opioids (Charney, et al. 1993; van der Kolk, 1996).

Neurobiological studies of trauma and PTSD victims have examined combat veterans, as well as other populations including women and children. A typical profile of PTSD shows lower basal cortisol levels, higher glucocorticoid receptor numbers, and enhanced sensitivity to exogenous steroids (Golier & Yehuda, 1998). Furthermore, a study of rape victims and the immediate response measures of cortisol and 3-methoxy-4-

hydroxyphenylglycol (MHPG) who subsequently developed a PTSD diagnosis showed that women with a prior history of physical or sexual assault had attenuated cortisol levels compared to women without such a history. The MHPG levels appeared to be associated with injury related to the rape and active avoidance (Yehuda, Resnick, Schmeidler, Yang & Pitman, 1998)

However, Stein, Yehuda, Koverola and Hanna (1997) examined responses of women with early experiences of sexual abuse in childhood or adolescence to the dexamethasone suppression test (DST). The DST further tests the efficiency of the hypothalamic-pituitary adrenal (HPA) axis in shutting off a normal stress response. The results showed that compared to non-abused women, women with a previous history of severe sexual abuse had significantly enhanced suppression of plasma cortisol in response to 0.5mg of dexamethasone. The authors report similar findings with veterans with combat related PTSD. Other populations studied with similar findings include Holocaust survivors (Yehuda, Kahana, Dinder-Brynes, Southwick, Mason & Giller, 1995). However, the HPA response to the dexamethasone suppression test is different with acute stress and major depressive disorder; cortisol is high suggesting that the cortisol receptors are not sensitive. Of particular interest, Lemieux and Coe (1995) examined women with a history of PTSD related to childhood sexual abuse and found that, compared to the control groups, levels of norepinephrine, epinephrine, dopamine and cortisol were significantly elevated. Since these findings differed from the combat veterans with PTSD, the discrepancy was explained in terms of gender difference or age at onset of the trauma or physiological variation. Methodological issues need to be addressed as well, particularly when making comparisons across studies since some

studies measure salivary cortisol levels, others plasma levels, while others measure 24hr urinary cortisol excretions.

Arousal vs Dissociation

These differences may be accounted for by individual differences in the coping response to the extreme trauma. Perry, Pollard, Blakley, Baker and Vigilante (1995) describe an adaption style to trauma as a continuum from arousal to dissociation. The neurobiology of hyperarousal response is different to the neurobiology of dissociation. Perry et al. associates the neurobiology and phenomenology of dissociation to the "defeat" reaction described in animals. From the animal models, an important difference between hyperarousal and dissociation is the response of the central nervous system to the release of acetylcholine with dissociation or the freeze or defeat response. The *parasympathetic* nerves to the heart, the vagus nerve, cause acetylcholine to be released, decreasing the rate in the sino-atrial (S-A) node and also decreasing the excitability of the atrioventricular (A-V) junctional fibres. A decrease in blood pressure is experienced, sometimes causing fainting. The hyperarousal response is due to the release of norepinephrine, a *sympathetic* stimulation effect, which has the opposite effect on the heart to the vagal stimulation. First, it increases the rate of the S-A nodal discharge and also increases excitability of all portions of the heart, with increased contractions of both the atrium and ventricle. Maximal stimulation can increase the heartbeat to three times the normal rate in humans (Guyton, 1969). According to Perry, et al. extreme psychological responses to trauma may evoke arousal and/or dissociative symptoms as a result of the physiological response of the sympathetic and parasympathetic systems. Studies of PTSD victims measuring levels of norepinephrine and cortisol have found that the ratio of norepinephrine to cortisol levels have been twice as high when compared to

groups of patients with bipolar manic depression or paranoid schizophrenia. No studies have been cited to measure a PTSD diagnosis in relation to norepinephrine and acetylcholine levels.

However, the function of the central cholinergic systems and their role in cognition has increasingly become an area of interest because of the association of acetylcholine and Alzheimer's disease. People who die with the disease have been demonstrated to have decreased cholinergic markers in the cerebral cortex. Studies have shown that acetylcholine can either improve memory or impair memory, depending on the acetylcholine receptor subtypes. For example, one particular type of acetylcholine receptor, the M1 muscarinic subtype, may impair memory, while a M2 receptor antagonist improves memory (Everitt & Robbins, 1997).

Changes in brain structures using magnetic resonance imaging (MRI) have been reported with lower hippocampal volume in patients with PTSD linked to combat trauma (Bremner, Randall, Scott, Bronen, et al. 1995; Gurvits, Shenton, Hokama & Ohta, 1996) and adult survivors of childhood abuse (Bremner, Randall, Vermetten, et al. 1997; Stein, Koverola, et al. 1997). Another study using functional magnetic resonance imaging by Kato, Erhard et al. (1998) found the hippocampal region to display a long-term sustained response to an auditory word memory task. This was twice as long as the duration in the auditory areas and Broca's area.

With advances in such techniques as magnetic resonance imaging, a recent study by Sheline, Sanghavi, Mintum and Godo (1999) found subjects with a recurrent history of depression to have smaller hippocampal volumes compared to the control group. In addition, smaller amygdala core nuclei volumes correlated with hippocampal volumes. The study suggests that repeated stress during recurrent depressive episodes may cause

insult to the hippocampal cells affecting the overall volume. So far these studies do not differentiate the biological changes for different psychological diagnostic classifications. Since depression is often part of PTSD symptoms, and PTSD is not diagnosed in many individuals presenting with mental disorders such as major depression, it only seems logical to examine specific symptoms, rather than diagnoses, which cluster symptoms together. Similarly examining symptoms would address the inconsistency found with neurohormonal levels in PTSD groups. The three classes of specific trauma symptoms associated with the diagnosis of PTSD, intrusive experience, defensive avoidance, anxious arousal, together with levels of dissociation, depression and anger may contribute in understanding the underlying neurobiology and relationships with learning and memory.

These biological changes associated with trauma and PTSD are different from the stress response, with high cortisol levels being directly associated with memory impairment. Research on neurobiological changes so far have extended to maltreated children with a diagnosis of PTSD and associated with adverse brain development and alterations in the biological stress system (Boring, Frustaci & Ryan, 1999; De Bellis, Baum, et al. 1999)

Studies examining stress, trauma and PTSD associated with learning and memory deficits have continued to focus on biological changes and have not provided an integrated psychological theoretical framework in an effort to understand cognitive impairment and trauma.

A Theoretical Framework

Jones and Barlow (1992) attempted to integrate PTSD theory with cognitive processes. Their theoretical model of the etiology and maintenance of the symptoms of

PTSD directly links the possible inherited biological vulnerabilities to PTSD and the psychological dispositions of the individual to explain the cognitive distortion component of the aetiology of PTSD. However, Jones and Barlow's model focuses on cognitive distortions (i.e., attributions) rather than cognitive deficits, and fails to explain trauma symptoms associated with verbal memory deficits.

From the cognitive literature, Baddeley's (1986) Working Memory Model provides further understanding of the process by which symptoms of trauma may affect memory systems. He conceptualised working memory as a system for temporary short-term maintenance and managing of information needed to perform a variety of cognitive tasks. The working memory model comprises sub-systems of a "central executive" (CE) and two sensory processing systems; the phonological loop (PL) which processes auditory information, and the visuospatial sketchpad (VSSP) which processes visual and spatial information. (See Figure 1.) The CE functions to help maintain and control the information by active attention. The other subsystems help to maintain the information as a guide for adaptive action. However, if the "central executive" becomes dysfunctional, as may be the case with individuals with PTSD or trauma symptoms, then active attention for the short-term maintenance of information may be disrupted. The working memory system may provide some clues about the way that specific trauma symptoms may impact on dysfunctional verbal learning and memory. Since the slave systems have a limited capacity for processing information, concurrent visuospatial processing, such as intrusive re-experiencing symptoms of trauma or auditory processing such as rehearsal, may then interfere with short-term memory. (See model in Appendix A.)

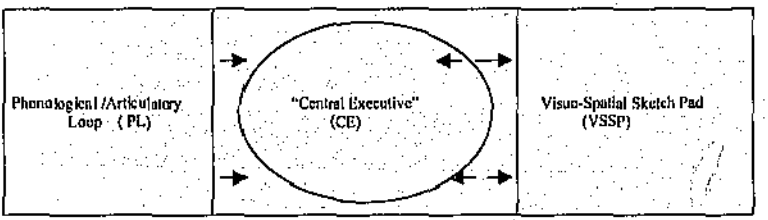

Figure 1. Working Memory Model from Baddeley, (1986).

Cognitive Processes After Trauma and PTSD

Memory problems as a result of trauma and PTSD may be due to changes in the neurobiology and brain structures responsible for learning, recall and emotional control and expression. Research into the aftermath of trauma and PTSD in relation to cognitive disruption has focused on two main areas: 1) cognitive processing biases, and 2) cognitive distortions.

Cognitive Processing Biases and Emotions

The relationship between cognitive processing and emotions has involved a long-standing debate; from the view that emotions are not dependent on cognition, (Zajonc, 1980) to the other extreme in which cognitive appraisal precedes emotions (Lazarus, 1982). However, an important factor in all these models is the differentiation between *conscious* strategic processes and *non-conscious* automatic processes. The automatic processes are considered to be involved with emotions, eg spreading activation, in relation to mood-memory network theory (Bower, 1992). A number of experimental studies in the last two decades have examined emotion and selective encoding associated with depression, anxiety and PTSD. Patterns that are quite distinct in processing

personally relevant emotional information have been found in individuals with anxiety, depression, and PTSD in comparison to matched control groups.

Interference Stroop Studies with Emotional Disorders

These patterns have developed from studies using interference tasks such as the modified Stroop colour-naming task. In this, the colours of the words are presented in blocks of neutral, positive, negative and emotionally disturbing or fear specific words. The reports have shown that individuals with high anxiety take more time to name the colour ink for threat-related stimulus words (Mathews & MacLeod, 1985; Watts, McKenna, Sharrock & Trezise, 1986). Similar patterns have been reported with people with high levels of depression for depression relevant words (Gotlib & McCann, 1984; Williams & Naulty, 1986). However a different pattern emerged in a study including a group of 20 patients with a) nine patients with a combined diagnosis of anxiety and depression and 11 patients with generalised anxiety only, and b) a control group. Patients with only generalised anxiety showed more colour-naming interference for anxiety words than neutral words using both supraliminal and subliminal exposure conditions. Interestingly the individuals with both anxiety and major depression did not show interference with negative words even though high levels of anxiety were diagnosed (Bradley, Mogg, Millar & White, 1995). Depression with anxiety seems to counteract the bias for negative information in preconscious processes. This is possibly due to depression being associated with amotivation, a slowing down in decisions and lacking in cognitive initiatives (Hertel & Hardin, 1990) or simply that individuals suffering from depression have a higher priority to focus on the self and internal perceptual events (Ingram, 1990) rather than future events.

Cognitive processing of emotional information in PTSD using a modified Stroop procedure have been conducted with combat veterans (Kaspi, McNally & Amir, 1995; McNally, English & Lipke, 1993; McNally, Kaspi, Reimann, & Zeitlin, 1990), rape victims (Cassiday, McNally, & Zeitlin, 1992; Foa, Feske, Murdock, Kozak & McCarthy, 1991); motor vehicle accident survivors (Bryant, & Harvey, 1995) and ferry disaster survivors (Thrasher, Dagleish & Yule, 1994).

Kaspi, et al. compared a PTSD group of 30 Vietnam veterans with a control group, and found more interference for combat words than for other words in the PTSD group. Similar results in rape victims with PTSD have been found with a longer response latency for colour naming of rape-related words than neutral, negative or positive word types (Foa, et al. 1991). Interestingly, the rape victims with PTSD had notably higher means on the Beck Depression Inventory than victims without PTSD and the control group. The diagnosis of depression with PTSD did not have the same effect as combined generalised anxiety and depression on the interference task.

The study using motor vehicle accident survivors with PTSD also included a group with simple phobia of driving as well as a control group with low anxiety (Bryant, & Harvey, 1995). This was the first study to compare interference on the modified Stroop test with individuals with PTSD and simple phobia. This was based on the proposed model for a stronger attentional bias in the PTSD condition by Litz and Keane (1989). As hypothesised, participants with PTSD demonstrated greater interference on strong threat words than did the simple phobia of driving and the low anxiety groups. In addition, *trait* and *state* anxiety mean measures showed no significant difference with PTSD and simple phobia. Contrary to what was expected, the simple phobia group did not display an interference effect. This study suggested that attentional processes might

be different for PTSD patients compared to simple phobia. Of particular importance is the fact that comorbid conditions such as depression were not assessed with the participants in this study.

Another study using the modified Stroop task with PTSD patients was conducted to extend trauma events from rape or war veterans (ie. intentional threat by a person) to disaster survivors of the *Herald of Free Enterprise* ferry that capsized at Zeebrugge in 1987 (Thrasher, Dalgleish & Yule, 1994). The results of this study exhibited similar patterns of selective processing bias for disaster-specific material in survivors with high PTSD vs low PTSD symptomatology as in previous studies with veterans, rape victims and motor vehicle accident survivors. Depression was measured in this study using the Beck Depression Inventory and the high PTSD group showed significantly higher levels of depression than the low PTSD and the control group. This suggests that depression and PTSD may not have the same counteractive processes found with depression and anxiety (Bradley, Mogg, Millar & White, 1995).

Consistent results with selective processing in anxiety disorders implies that individual specific threat related material is represented differently than in those without the disorder (Foa et al., 1991). Furthermore, it appears from these studies that: a) individuals with high PTSD symptomatology process threat material differently than individuals with other anxiety disorders and b) that comorbidity of PTSD and depression does not diminish the selective processing bias as with anxiety and depression.

Cognitive Distortions and PTSD

Foa , Steketee and Rothbaum (1989) proposed a model of the development and maintenance of PTSD, which extended classical conditioning of learned "alarm" reactions. The fear structures mentioned above are networks in memory that contain

information about the stimuli or responses that are dangerous, as well as information about the physiological responses to the threat or perceived threat. Foa et al. proposed that PTSD symptoms are produced when the meaning of threat becomes part of the network as a result of a traumatic event becoming an associated stimulus. A cognitive distortion such as an exaggerated expectation for the trauma to re-occur because of violated beliefs of safety during the traumatic experience then results. Other cognitive distortions include a belief that the anxiety will persist forever because of little control of the intrusive thoughts. Predictability and attributions of threat as well as a perception of incontrollability are two main concepts that are also distorted in PTSD sufferers and which are central to the maintenance of the symptoms.

To summarise, it is evident that cognition plays a major role in maintaining the trauma symptoms. Both areas of research in selective information processing biases for the threat-related material and also with cognitive distortions have shown a role of cognitions. By examining each of the trauma symptoms in relation to the literature on memory deficit and more specifically to verbal learning and memory, a deeper understanding of this complex area may be clarified. The model of memory used is Baddeley's framework of Working Memory.

Examination of Specific Trauma Symptoms in Relation to Learning and Memory Deficits.

Specific trauma symptoms in relation to PTSD have included intrusive experiences, defence avoidance and anxious arousal, while this study also includes symptoms of depression, dissociation and anger to examine the effects on verbal learning and memory.

Intrusive experiences reflect the "B" group symptoms of the DSM-IV diagnostic criteria for PTSD. They include nightmares, flashbacks and memories that are easily triggered by current events and repetitive thoughts of previous traumatic experience that intrude into conscious awareness. The individual exposed to trauma perceives these symptoms as being under "no control and come from nowhere" (Briere, 1995). Such experiences would predict negative effects on learning and memory performance tasks. Research into this specific symptom in relation to cognitive deficits has not been conducted.

Defensive Avoidance symptoms consist of avoiding internal and external stimuli that bring back painful experiences of the trauma. Individuals have reported trying to eliminate or stop thoughts or memories from conscious awareness as well as avoiding events or places that may reactivate the upsetting thoughts. Defensive avoidance is a coping mechanism by which the individual sustains psychological function and reduces anxiety to levels less overwhelming for daily living. Clearly defensive avoidance is a conscious and intentional process, unlike dissociation or repression (Briere, 1995).

Anxious Arousal

According to the process efficiency theory (Eysenck and Calvo, 1992), worries and other anxious thoughts tend to use resources of working memory such as processing and storage (Baddeley, 1986). The central executive and the phonological loop are assumed to be active with worry or other anxious thoughts (Derakshan & Eysenck, 1998). It may be that anxious arousal will have negative effects on cognitive performance, specifically verbal learning and memory. The results of research in the area of anxiety and performance are inconclusive. Theoretical and methodological factors may explain some of the inconsistencies. Eysenck suggested that anxiety has two

main effects on individuals a) the worry which acts as a distracter on the task, and b) an increase in arousal which may activate motivation to put more effort on the task and sometimes may counterbalance the worry (Baddeley, 1990). By studying neural mechanisms of the limbic cortex and the frontal lobes, individual differences in these two processes may provide unique ways of understanding that anxiety is not simply a distraction to cognitive mechanisms but may in fact play a vital role in cognitive adaption (Luu, Tucker, & Derryberry, 1998).

Baddeley (1990) reviews the literature on anxiety and performance. In the last two decades evidence has shown that anxiety impairs a number of cognitive tasks specifically, anagram task performance, mental arithmetic, digit span and free recall. An area that has had extensive research in relation to anxiety and performance is scholastic performance.

Test anxiety was originally thought to comprise two factors. The first is the emotional response with the physiological changes of sweating and increased heart rate, and secondly, the self-talk of negative future consequences eg. " I'll fail the test". The latter, referred to as worry, was significantly correlated with test results. Furthermore, reports of worrying have since been associated with less use of deeper processing strategies while studying (Baddeley, 1990).

Examining the literature more specifically on verbal memory and anxiety, Wiens, McMinn and Drossen (1988) used the RAVLT scores and the Spielberger State-Trait Anxiety Inventory and found no significant correlations. Yet another study by Unkenstein and Bowden (1991) found a correlation of -.57 in an alcohol rehabilitation group, but these findings were not replicated in a second study. It appears that worry and

arousal may be competing factors that can explain the inconsistencies when researching cognitive function and anxiety, particularly new verbal learning.

There is evidence that mood and emotional factors may influence learning and memory. Stress may distort normal attention, thus affecting the way material is encoded. Depression is also suggested to impair learning, probably due to inadequate encoding rather than deficits in retrieval (Baddeley, 1990).

Depression is another response to a traumatic event with perceptions of loss of control over the trauma experience. It can also be a secondary symptom as a result of perceptions of loss of control over overwhelming feelings of anxiety or anger (Carlson 1997).

Depression may manifest cognitively, emotionally, behaviourally and physiologically. Only the cognitive factors will be discussed here. Cognitively, depression has been shown to lead to biased recall of negative stimuli and recall deficits for positive and neutral stimuli (Burt, et al. 1995). Numerous clinical studies have been performed with mixed findings, some indicating poor memory on some tasks but not on others, and some have reported significant improvement after treatment with antidepressants or other therapies (See review by Burt, Zembar and Niederehe, 1995). These inconsistencies may have been due to a number of variables including: subtype of depression, severity, inpatients or outpatients, age, assessment tasks eg. visual or verbal, effort required for acquisition and recall to name a few. Burt et al. (1995) conducted a meta-analysis using data from 99 studies on recall and 48 studies on recognition in clinically depressed and non-depressed samples and found a significant association between depression and memory impairment. More specifically, there were greater depression effects for verbal than for visual stimuli. In addition, secondary analyses were

conducted to determine if memory impairments were specific to depression or other psychiatric disorders such as schizophrenia, substance abuse, anxiety and personality disorder. The findings indicated no memory impairments with subjects with only substance abuse or only anxiety disorder when compared to a control group, but significant impairment in the comorbid mixed group.

Burt et al. (1995) gave some theoretical explanations for the association of depression and memory deficits. They include the mood congruency effects, conservative response biases or impoverished output due to low energy or motivation, impaired use of effortful memory strategies to sustain attention, or lack of motivation to use them, and the length of retention interval.

Hertel (1997) expands on these possible explanations of memory deficits for neutral events associated with depression. Interestingly, depressed individuals have good memory for negative events (explained by the self-focusing process) and recall related to depressed mood. On the other hand, self-focusing or intrusive thoughts for neutral material impaired memory can be explained by two stages of the information processing model. In the initial stage of encoding, intrusive thoughts may redirect attention, since with a depressed person, self-concerns are more demanding of attention. The second stage at which memory can be affected is at the retrieval time, when intrusive thoughts may redirect attention. Hence, with severe depression and greater frequency of intrusive thoughts, the greater the inability to redirect attention to the task. Williams, Watts, MacLeod and Matthews (1988) concluded that deficits of memory in depression could be predicted by the degree of involvement along the continuum of controlled to automatic processing.

Dissociation according to Briere (1995) is a coping avoidance mechanism that is unconscious, and which results from a traumatic experience and severe psychological distress. Behavioural and cognitive processes include disengagement, depersonalisation, derealisation, out of body experiences and emotional numbing. High levels of dissociation have been reported to be associated with distractibility, "spacing out" and feeling out of touch with the body.

Dissociation symptoms have been shown to be strongly associated with traumatic experiences, particularly the pathological forms of fugue and dissociative disorders (Putman & Carlson, 1997; Sanders & Giolas, 1991). Assessing dissociation with trauma victims is strongly recommended by Carlson (1997) to detect the cognitive avoidance strategies used and disturbances in memory and identity. Reports of dissociation during the trauma event may involve cognitive, affective, behavioural and physiological avoidance, eg. unable to feel the pain of physical leg injury during battle. Dissociative activity during the trauma is a strong predictor of developing PTSD (van der Kolk, 1996). Dissociation has been associated with other disorders and to occur in non-clinical contexts. However, the pathological symptoms of dissociation will cause distress and dysfunction in daily activities for individuals having experienced a traumatic event.

The integration of dissociation symptoms into a framework of trauma response by Carlson (1997) provides a very useful association with verbal learning and memory deficits. Van der Kolk, Van der Hart and Marar (1996) consider the two trauma symptoms described above, intrusive experiences and defensive avoidance, as dissociative because the experience is out of context. However, as defined by Briere (1995), dissociation is unconscious, yet the intrusive experience can be conscious. It needs to be made clear also that avoidance can be conscious or unconscious. When

depersonalisation or distortions of the self and distortions in perceptions of the world are experienced, it is a form of cognitive avoidance of which the individual is unaware. The above discussion of the two symptoms (of the conscious form) gave some explanations as to possible processes in the working memory system that can compromise the verbal learning process. The question then arises as to the interaction of such unconscious processes and the working memory system.

If we adopt the definition by Carlson of pathological dissociation as:

> "*cognitive, affective, behavioural, and physiological re-experiencing and avoidance that results in lack of integration of thoughts, feelings, behaviours and sensations into the stream of consciousness*",

then we must conclude that dissociation involves some degree of both conscious and unconscious awareness. According to Horowitz (1986), this conscious awareness has some motivational basis to process the trauma and possibly alleviate the cognitive dissonance so as to interpret the event in ways consistent with the schemas of self and the world. In an effort to understand dissociation symptoms in relation to verbal memory deficits and Baddeley's working memory system, this adoption of dissociation may be helpful to integrate theory, clinical observations and research of trauma symptoms.

Carlson's definition of dissociation involving re-experiencing and avoidance is supported by a number of researchers who have reported these two symptoms to go through a cycle of phases (Herman, 1992; Horowitz, 1986; Spurrell & Mcfarlane, 1995). Although Herman supports the cycle, she applies the model to childhood abuse trauma using a different approach. The avoidance phase is the dissociative state that traumatised children adopt as a defense mechanism. Herman (1992) hypothesises that this automatic coping mechanism can be used to extreme levels and lead to feelings of disconnectedness

and detachment. Other disturbing behaviours may result, such as self–mutilation, in the struggle for the traumatised individual to feel, and be connected with the self and leave the dissociative state. Self-injury has been a major concern with therapists working with individuals experiencing dissociation (Halpern & Henry, 1994). So far, research investigating PTSD in relation to verbal learning and memory deficits has not assessed levels of dissociation. Furthermore, much of the research on dissociation has focused on memory impairment in terms of fragmentation for recall of the trauma. Van der Kolk and Fisler (1995) review the literature on the comparison of memories for stressful and trauma events and this may shed some new insight into verbal memory and trauma. The reports of intrusive recollection of the traumatic events in patients suffering PTSD was shown not to change over time. This was different for reports of "flushbulb memories" in which subjects had changed their recollection after a number of years. The reason for these differences according to van der Kolk and Fisler is the way the event is initially stored as sensory fragments without coherent sematic components. In addition, the memories are said to be highly state-dependent and cannot be elicited at will. Eventually, subjects reported that a narrative of their traumatic event developed over time. This is different for non-traumatic sensory information processing in which the input is synthesised into symbolic form without conscious awareness. The process involves the translation of the sensory impressions into a personal story. The lack of this process with trauma events may have implication on new information processing.

Memory problems are also associated with dissociative symptoms about the trauma and with the false memory syndrome, but this study will not expand on these issues. An additional important element is that the memory impairment may lead to

under reporting of the traumatisation or the event (van den Hout, Merckelbach & Pool, 1996; van der Kolk, 1996; Carlson, Armstrong, Loewenstein & Roth, 1997).

The processing of information of trauma experiences from a neurological perspective suggests reasons for learning and memory impairment associated with dissociation. Neuroimaging during an intrusive experience (considered being dissociative) found an increase of activity in the right hemisphere that is associated with the activity of emotions as well as in the right visual association cortex. However, there was a decrease in activity (ie blood flow and oxygen utilisation) in Broca's area in the left inferior frontal cortex associated with verbal representation (Rauch, van der Kolk, Fisler, et al in press). This area is also involved in the phonological loop of the verbal working memory.

However a recent study of adult women traumatised by childhood sexual abuse measured significant differences on measures of the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986) and found no impairment on explicit memory function when compared to the comparison group (Stein, Hanna, Vaerum & Koverola, 1999)

Anger The symptom of anger has been already discussed in relation to trauma, but no study has explored the emotion of anger and verbal learning and memory. Elevated levels of anger have been positively associated with the development of PTSD (Riggs, Dancu, Gershuny, Greenberg & Foa, 1992). Anger may contribute to verbal learning and memory deficits through similar processes of interference with attention as depression.

Other important factors that have been reported to affect measures of verbal memory have been the effects of demographic variables such as age, education,

intelligence, and gender on the Rey Auditory Verbal Learning Test (RAVLT); Schmidt 1996).

Demographic Variables that influence Verbal Learning, Memory and Trauma

Age and Memory

Children's performance on the RAVLT improves with age, while experimental research confirms that memory performance does decline as a function of normal ageing (Schmidt, 1996). The RAVLT normative data found a significant effect of age on performance over the ages of 5-79 years.

Early studies report that working memory declines with increasing age, eg. a drop in backward digit span from age 70-79 (Berg, 1980; Johansson & Berg, 1989). A number of experimental studies conducted by Salthouse, Babcock and Shaw (1991) that compare younger and older adults imply that there are no age-related differences on structural and operational capacities of working memory. It is important to understand neurological assessment measures for active verses passive measures of working memory. The active aspect of working memory requires tasks that constantly update and manipulate information processing. Dobbs and Rule (1989) also found an age associated decline in working memory between ages of 60-69 and 70 + years, suggesting an overall decline of the processing capacity or the speed at which the processing changes can occur.

Fisk and Warr (1996) examined specific sub-systems of working memory and compared older with younger groups of adults. Deficits were observed in working memory, perceptual speed and central executive functioning but not in functioning of the phonological loop. The findings suggested that when perceptual speed is controlled for, all of the age variance in working memory and central executive functioning differences

were almost eliminated. Once again a slowing down in the rate at which information is activated is evident.

Longitudinal studies on aging and memory change have the added advantage of greater sensitivity to age change since individual differences no longer need to be taken into account statistically. Hultsch, Hertzog, Small, McDonald-Mizczak and Dixon (1992) measured change on memory, intellectual ability and information processing tasks over a 3 year interval on 328 community dwelling men and women aged 55-86 years. They found declines in working memory, world knowledge, and verbal fluency, even when processing time variables were controlled statistically. However, word recall showed no significant change. Baddeley, Wilson and Watts (1996) also review evidence that recall of lists of items drop very little with age.

Age is also known to moderate emotions. Hence, assessment of the perceived trauma symptoms may be effected with increases in age. Older adults report few negative emotional experiences, have greater emotional control and lesser expressivity when compared to a younger cohort. This indicates that with age, there is increased competence for emotional regulation. In particular, there is a decrease in subjective experience of anger, sadness and fear and an increase in happiness (Gross, Carstensen, Tsai, Skorpen, & Hsu, 1997).

Gender and Memory

Gender has been related to performance on verbal memory tests in some studies. Others reported negative findings, while still others have reported that females out-performed males on the RAVLT (Schmidt, 1996).

Vakil and Blachstein (1997) used the RAVLT on 528 people aged 21-91. The results showed a significant and consistent advantage for females over males on most of

the verbal memory measures. Developmental sex differences in verbal learning have also been found. A total of 811 boys and girls ages between 5 and 16 were administered the California Verbal Learning Test- Children's Version and sex differences were found at all age levels. Girls performed better than boys on all of the immediate and delayed recall trails and the delayed recognition trail (Kramer, Delis, Kaplan, O'Dennell & Prifitera, 1997).

Education and Intelligence and Memory

Research on the RAVLT has generally found a small moderate positive correlation between education and memory indices. Significant correlations have varied from study to study. Similarly, studies with intelligence and verbal memory have been inconsistent, and Schmidt (1996) suggests that these factors do not contribute substantially in interpreting test performance.

However, intelligence has been reported to have significant negative correlations in relation to PTSD symptoms (approximately $r = -.33$ to $-.37$) and predict an additional 10% of the variance after combat exposure had accounted for 17% of the regression on scores of PTSD (McNally & Shin, 1995; Vasterling, Brailey, Constans, Borges and Sutker, 1997). A recent study examining pre-combat intelligence in a PTSD and non-PTSD group found that people with lower intelligence were more likely to develop PTSD symptoms, even when controlling for the extent of combat exposure (Macklin, Metzger, Litz, McNally, Lasko and Orr, 1998).

Alcohol, Trauma and Memory

Although memory impairments have been associated with the severity of trauma in sexual and physically abused adult survivors and war veterans with PTSD as already discussed, research also indicates that a high proportion of trauma victims have alcohol

related problems. Keane, Gerardi Lyons, and Wolfe (1988) have demonstrated PTSD and substance abuse disorders. American studies have indicated between 40-70 % of women in treatment for alcohol or drug dependency have experienced incest in childhood (Bammer, 1993). Childhood physical and sexual abuse victims have reported alcohol dependency (Bammer, 1993; Fleming, Mullen, Sibthorpe et al. 1998; Ladwig & Anderson, 1989; Moncrieff & Farmer, 1998). The prevalence of alcoholism in veterans in an American study of the Vietnam and Korean wars attending a clinic not devoted to the treatment of alcoholism was 60%, compared to 25% of others attending the clinic at the same time (Branchey, Davis & Lieber, 1984). In Perth, the Vietnam Veteran Counselling Service reported between 80-90% of the veterans that use their services experience alcohol abuse (D. Unbovick, personal communication, May 26,1998). Alcohol consumption in the studies by Bremner, et al. (1993,1995) had been controlled for by matching for the total number of years of the alcohol abuse in the two groups. However, this may not adequately control for the effects of alcohol on memory function *per se*.

Alcohol and other substance are known to have negative effects on memory (Parsons, 1987; Uddo & Gouvier, 1990). In a study by Waugh, Jackson, Fox and Hawke et al. (1989), the effects of alcohol consumption of 131 healthy male social drinkers was examined. The results showed a decline on the Rey Auditory Verbal Learning Test, Austin Maze, and the Little Man and Spatial Memory Tests when comparing the high consuming group to group that consumed lesser amounts.

In another study relating to alcohol and verbal memory, 491 patients treated on an alcohol ward and who were free from psychosis and brain damage were administered the MMPI and the Rey Auditory Verbal Learning Test to evaluate the effects of alcohol and

depression on memory function. The RAVLT scores of the alcohol group were lower on initial repetition, recall and recognition trials and the depressed group only had lowered initial repetition scores (Query & Megran, 1984).

Rationale to Link Trauma Symptoms and *Verbal* Learning and Memory Deficits

Since World War II, studies of war veterans have shown the negative behavioural and emotional outcomes that constitute PTSD (Yehuda & McFarlane, 1995). Early clinical reports also indicated that 67-100% of veterans with PTSD suffered problems in memory and concentration (Archibald & Tuddenham, 1965; Burstein, 1985).

In the last decade, research has focused on the long-term cognitive impairment of veterans. Wolfe and Charney (1991) advocated neurological assessment to be incorporated in the PTSD diagnosis as a result of many veterans' complaints of disturbances in concentration and memory. Memory problems in relation to extreme trauma have now been recognised and have certainly gone beyond the early perception of compensation neurosis as reported by Archibald and Tuddenham (1965). A comparison of peacetime neuroses with war neuroses found a higher percentage of cases with war experiences to have resistant symptoms. The cause was considered to be because of secondary gains of pensions and disabilities rather than the traumatic stress of war.

In the last decade, problems in memory, and in particular verbal deficits have been reported, with traumatised individuals who have experienced sexual/ physical abuse, war veterans or other traumatic events two to three decades ago are of concern. The role of verbal learning and memory in most therapeutic interventions relies on verbal recall and learning of linguistically based materials. Verbal deficits would clearly affect progress in these therapies. Difficulties in learning and concentration also have implications for impaired academic performance (See review by Frayne, 1999).

From a psychological perspective, very little is known of the processes underlying the long-term consequences of trauma, PTSD and how these may involve learning and memory function. The neurobiological research on PTSD so far suggests that it is possible for "states" of hyperarousal to become "traits" with definite anatomical changes, such as in hippocampus volume, as well as other permanent physiological changes (Perry, et al. 1995). Recent studies using neuropsychological tests of attention and learning have reported both global and more specific deficits. For example, global cognitive deficits are reported with accompanying broad psychopathology in former prisoners of war and combat veterans (Sutker, Winstead, & Allain, 1991). Bremner, Scott, Delaney, et al. (1993) narrow the focus of cognitive deficits to short term memory with combat veterans. Another study of PTSD in adult survivors of physical and sexual abuse provided information on deficits in short-term *verbal* memory (Bremner, Randall, Scott, Capelli, et al. 1995). Uddo, Vasterling, Brailey and Sutker (1993) used the RAVLT to assess verbal memory and found poorer performance in the PTSD veterans than the comparison sample. They showed lower scores on both the repeated trials and greater sensitivity to proactive interference and perseverative errors. Yehuda, Keefe, Harvey, et al. (1995) also used a specific verbal measure, the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan & Ober, 1987). Unlike the previous findings of Uddo, et al., their results showed no differences between veterans with PTSD and a community control group on measures of initial attention, immediate memory cumulative learning and active interference from previous learning. However, differences were found on both short and longer delayed recall trials. In a study of Persian Gulf War veterans (Vasterling, Brailey, Constans & Sutker, 1998), deficiencies were found on immediate and delayed trials of RVLT, with more intrusions and false positives on the

recognition trial. The symptoms of re-experiencing and avoidance accounted for the variance on intrusions. A recent study by Jenkins, Langlais, Delis and Cohen (1998) of rape victims with PTSD found a significant difference on the delayed free recall using the CVLT when compared to two control groups. Yet, Stein, Hanna, Vaerum and Koverola (1999) report no evidence of explicit memory impairment between child sexual abuse survivors and a comparison group using the California Verbal Learning Test.

Studies to date in relation to PTSD and memory deficits have focused on a global measure of PTSD. Comparison of some of the studies need to take into account the changes made in the DSM, but also that such comparisons of PTSD and non-PTSD groups are not homogeneous and may give inconsistent results. By exploring the specific symptoms as a result of perceived trauma this may alleviate such confound in relation to verbal learning and memory deficits.

Methodological Issues Related to the Studies of PTSD and Learning and Memory Deficits.

A closer examination of the literature to assess the reasons for inconsistency in the studies relating to PTSD and verbal and learning deficits appears to be related to a number of methodological issues. These include: small sample sizes, insufficient control groups, ethnic differences across samples, unreliable measures of severity, comorbid disorders, different settings, the use of a number of different neuropsychological tests, and different time intervals to measure delayed recall and alcohol consumption.

Sample Size

Very small sample sizes have been a general criticism of such studies with sample ranges from 12 to 26 participants in size across control and test groups. Results are very difficult to generalise from these studies.

Insufficient Control Groups

The majority of the studies have used one control group from either the community or enlisted recruits to compare a PTSD sample of in-patients or outpatients matched for age, sex, years of education, handedness, socioeconomic status and alcohol abuse. Significant differences in memory deficits between the two groups do not imply that these are due to PTSD. No control groups with trauma exposure and no PTSD symptoms have been included in the designs, with the exception of the more recent rape victims study by Jenkins, et al. (1998), making the results of other studies in this area inconclusive. In addition, Stein, et al. (1999) names the two groups in the study child sexual abuse and healthy comparison participants, since only 77% of the sexual abuse group have a diagnosis that met the DSM-IV, adding to problems of comparing results.

Ethnic Differences Across Samples

In addition, the composition of the groups varied. For example, the majority was white in Bremner, Randall, Scott, et al. (1995), while in Vasterling, et al. (1998), the majority was made up of African Americans. The different cultural context makes comparisons of the studies difficult and may have some implications for the cognitive assessments. In addition, the majority of studies use males only (Bremner, Scott, Delaney, et al. 1993; Uddo, et al. 1993; Yehuda, Keefe, et al. 1995), while two studies used a small number of females (Bremner, Randall, Scott, et al. 1995; Vasterling, et al.

1998). Significant differences between gender are difficult to assess with such small samples.

Unreliable Measures of Severity

The diagnosis of PTSD in the veteran studies (Bremner, Scott, Delaney, et al. 1993; Bremner, Randall, Scott, Capelli, et al. 1995; Uddo et al., 1993; Yehuda, Keefe, et al., 1995) was assessed with the Structured Interview as well as with the Mississippi Scale-Combat Related (Keane, Caddell, & Taylor, 1988) to measure severity of the veterans PTSD symptoms. A closer examination of the Mississippi Scale-Combat Related indicates that it does not assess all of the symptoms of PTSD as per the DSM-1V. In addition, only a total score is obtained and subscale scores of the particular symptoms that make up total PTSD severity symptom are not assessed. The study by Vasterling, et al. (1998) of Persian Gulf War veterans utilised the Mississippi Scale for Desert Storm War Zone Personal (adapted from Keane,Caddell & Taylor, 1988) and included a rating of symptom severity for each PTSD symptom using criteria B, C and D according to the DSM-1V. The study by Bremner, Randall Scott Capelli, et al. (1995) with adult survivors of childhood abuse used the Schedule for Affective Disorders and Schizophrenia - Lifetime Version (SADS-L; Endicott & Spitzer, 1978) to exclude participants from the study and also select those with an Axis 1 disorder. Participants were included if they had a history of severe childhood physical, sexual and emotional abuse using the Early Trauma Inventory (ETI; Kriegler, Blake, Schnurr, Bremner, Zaidi & Krinsley, 1992). This scale was developed as part of a parallel project and an index of severity of abuse exposure was incorporated so that the relationship between severity of childhood abuse and memory function could be examined. The severity indexes were formed by counting the number of times the physical, emotional and sexual abuse

occurred multiplied by the total number of years the abuse was maintained. Such a measure of severity of abuse has limitations because of the retrospective nature and often inaccurate report of abuse by patients, especially if they were very young when the abuse occurred. Van der Hart and Nijenhuis (1999) recommend corroborating evidence when childhood abuse is reported and a detailed index measure may be of questionable accuracy.

"Severity" is complex and some studies above have measured severity in terms of symptoms of a total PTSD score, while others have attempted to measure severity in terms of the specific symptoms of the criteria B, C and D of the DSM-IV for PTSD. "Severity" in these studies focuses on what has happened after the event, while the study by Bremner, Randall Scott Capelli, et al. (1995) with adult survivors of childhood measured severity in terms of the traumatic event(s). Measuring the severity of a traumatic exposure is problematic for a number of reasons. These include that the individual perception of an event differs in the degree of distress from one person to another. In addition, the possible low test-re test reliability of recall for a number of distressing events maybe additive. There are biases in a retrospective assessment to find the stressors for PTSD diagnosis, since the forgetting process is a normal adaption to the trauma event (McFarlane & de Girolamo, 1996).

In addition, the above studies had the experience of a particular trauma as the inclusion criterion for the study eg, war experience, sexual and/or physical abuse, but other traumatic events in the individual lives since the original event have not been reported. So far no scales have been developed to assess severity of the trauma and while measures have been developed to assess symptom severity, concerns remain as to their accuracy. Another concern is the time that has lapsed between the trauma and symptom

measurement. What other factors play a part in coping with the symptoms to minimise or maximise them?

Comorbid Disorders

Another issue regarding the studies of PTSD and memory deficits is the presentation of common comorbidities with PTSD. Research investigating cognitive impairment with concurrent affective comorbidities may have additional impacts on memory. So far methodologies have been inconsistent, with some studies having included comorbidities in the PTSD group (Bremner, Randall, Scott, et al. 1995; Vasterling, et al. 1998), while others excluded subjects in the PTSD group with comorbidities (Bremner, et al. 1993; Uddo, et al. 1993; Yehuda, Keefe, et al. 1995). The recent study by Stein, et al. (1999) measured depression and dissociation symptoms. The comparisons of findings in relation to trauma and PTSD in relation to memory deficits can thus be confounded by comorbid disorders. However, Jenkins, et al. matched control groups for anxiety. In addition, since depressive symptoms were observed in 53% of the PTSD group and 6% of no PTSD rape group and 0% of the non-traumatised no PTSD group, depression scores were covaried in the statistical analysis.

Different Neuropsychological Tests and Different Time Intervals for Delayed Recall

In addition, the above studies have used a variety of neuropsychological tests to assess verbal memory. Studies of Bremner, et al., employed two subtests of the Wechler Memory Scale-Revised (WMS-Revised; Wechsler, 1987): logical memory, that is the free recall of two story narratives, which is felt to represent verbal memory, and Figural Memory, felt to represent visual memory. The Verbal and Visual Selective Reminding tests were used as well. Yehuda, Keefe, et al. (1995) and Stein et al. (1999) used the CVLT, while Uddo et al. (1993) and Vasterling, et al. (1998) used the RAVLT.

Conclusive outcomes from these assessments are difficult in the absence of a common measure and time interval for the delayed recall also differed.

Alcohol and Drug Use

A measure of alcohol abuse in the studies above helped match controls, but drug dependency and abuse was not accounted as matched (Bremner, Randall, Scott Capelli et al. 1995). Bremner, Scott, Delaney et al. (1993) included a wash out period of one month, or an abstinence of six months (Uddo et al. 1993) while other studies excluded participants using alcohol and drugs (Stein, et al. 1999) or drugs alone (Jenskins, et al. 1998). Marijuana consumption elicits diverse physiological and psychological effects on humans, including memory loss. In particular, animal studies examining delta-sup-9-tetrahydrocannabinal (THC), the major psychoactive component of marijuana caused shrinkage of neuronal cell bodies and nuclei as well as genomic DNA strand breaks in the hippocampus. Neuronal death was inhibited by nonsteroidal antioxidants such as aspirin, vitamin E, and indomethacin (Chan, Hinds, Impey, & Storm, 1998).

In an experimental study, administrating 3 doses of alcohol and 3 doses of marijuana to volunteers males 18-26 yr old with histories of moderate alcohol and marijuana consumption was examined. There was impairment on digit symbol and word recall tests, but no effect in time perception and reaction time tests (Heishman, Arasteh & Stitzer, 1997). However, in a more recent study examining polysubstance abuse in women only in relation to verbal attention and memory impairment, no significant differences were found when compared to controls on measures of attention or memory. The polydrug group performed lower on the WAIS-R vocabulary subtest and more on depressive symptomology of the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, Erbaugh, 1961).

Such variations of methods used for alcohol and drug use in the above studies make comparison very difficult.

The Present Study

The present study was designed to extend the studies of Bremner, Randall, Scott, et al. (1995), Bremner, Scott, Delaney, et al. (1993), Jenkins, Langlais, Delis & Cohen, (1998), Stein, Hanna, Vaerum and Koverola, (1999), Uddo, Vasterling, Brailey and Sutker, (1993), Vasterling, et al. (1998) and Yehuda, Keefe, et al. (1995) by examining the relationship of specific trauma symptoms (intrusive experience, defense avoidance anxious arousal, including depression, dissociation and anger) on verbal learning and memory deficits. Demographic variables such age, IQ, education, gender, alcohol and drug use additionally served as predictor variables in relation to verbal learning and memory deficits. A set of trauma severity variables was established to take into account the different perceived traumas experienced by participants to predict verbal memory deficits.

This present study administered the Trauma Symptom Inventory (TSI), which assesses specific symptoms in the form of subscales of Intrusive Experiences, Defensive Avoidance, Anxious Arousal, Depression, Dissociation, and Anger/Irritability (Briere, 1995). The Trauma Symptom Inventory (TSI; Briere, 1995) was employed since it is a reliable and valid inventory, with normative data for age *and* gender. Of particular importance, the TSI includes 3 validity scales, which distinguishes it from all other measures of PTSD assessments and diagnostic interviews (Edens, Otto, Dwyer, 1998). It

shows the heterogeneous nature of symptom levels in response to trauma rather than a total PTSD severity score.

This study is the first to examine specific trauma symptoms resulting from a response to perceived trauma of combat-related veterans, adults with childhood sexual physical abuse, as well as a variety of other traumatic events, in a Western Australian sample. By examining demographic variables on which previous studies have matched comparison groups (such as age, IQ, & alcohol), this study attempted to clarify which independent variables from sets of 1) demographic variables, 2) trauma severity and 3) trauma symptoms, are more likely to add to prediction of deficits in verbal learning and verbal memory. Furthermore, it also explored which of the trauma symptom/s played a significant prediction on verbal learning and memory deficits.

Research Questions:

1. To what extent do demographic factors predict deficits in verbal learning and memory?
2. When the set of demographic variable is included into the predictive equation, does trauma severity predict a significantly increased amount of variance in learning and memory measures?
3. By partially out the set of demographic variables and the set of trauma severity variables, does the set of trauma symptoms of intrusive experience, defensive avoidance, anxious arousal in addition, depression, dissociation and anger significantly predict verbal learning and memory deficits?

Method

Research Design

The study employed a cross-sectional, correlational design. It examined the relationship between sets of demographic variables, trauma severity and trauma symptoms on verbal learning and memory. The set of demographic predictor variables included gender, age, IQ, years of education, and the level of alcohol abuse and drug use. The set of trauma severity measures was made up of the number of traumatic events experienced, distress levels for each of the events and duration of the trauma. The third set of predictor variables was made up of the subscales of the Trauma Symptom Inventory (TSI; Briere, 1995): Intrusive Experiences, Defensive Avoidance, Anxious Arousal, Depression, Dissociation and Anger. Verbal learning and memory was measured using the Rey Auditory-Verbal Learning Test (RAVLT; Spreen & Strauss, 1991). The verbal learning measures included Trial 1 and the sum of Trials 1-5, while verbal memory measures included Trial 7, immediate recall, and Trial 8, delayed recall.

Participants

One hundred and forty eight participants were recruited from service providers and the general community in the Perth metropolitan area of Western Australia. To qualify for the study, the inclusion criteria included participants over the age of 18 years old who reported experiencing a traumatic event. These events included childhood sexual or physical abuse, war experiences, sudden death of a family member or close friend, near death experience of self or another, and victims of crime, such as bank hold ups and burglary. Additional traumatic events that were included were diagnosis of a life threatening disorder and high risk surgery. Some participants reported to have

experienced more than one traumatic event. Twenty five reported to have been in the war, 39 had experienced sexual abuse, 27 physical abuse, 69 experienced sudden death of a family member or friend, 6 reported events with crime such as bank-hold up. Forty eight reported having had a traumatic experience such as an abortion, adoption, relationship break-up, discovering husband to be a paedophile, and severe medical condition. Participants were screened but not included because of the exclusion criteria These included participants who reported a history of schizophrenia, traumatic brain injury, brain tumours, epilepsy, meningitis, HIV infection, childhood learning disabilities, currently using benzodiazepine medication, or a loss of consciousness if more than 10 mins.

Materials

The ***Rey Auditory Verbal Learning Test*** (RAVLT; Spreen & Strauss, 1991) provided measures of verbal learning and memory. A copy of the inventory is included in Appendix B. This test requires the participant to remember a list of 15 unrelated words (List A) repeated verbally one per second, over five different trials. A second list of 15 unrelated words, List B, intended to interfere with previous learning is presented, next followed by a request to recall as many words from the original List A as possible (immediate recall). A second part to the test was a recognition test. The participant was asked to *recognise* as many words as possible from a list of 50 words. This List includes words from the original List A and B. This was followed by a delay of 30 minutes when the participant was then requested to *recall* as many words as possible from the original List A.

This test provides several measures of short-term auditory verbal memory, rates of learning, learning strategies, retroactive and proactive interference, presence of

confabulation or confusion in memory processes, retention of information, and differences between learning and retrieval. This study focuses on selected measures of acquisition and retrieval, and does not analyse the interference measures.

There are a number of alternative lists available should follow-up evaluations be needed in order to avoid practice effects. Reliability and validity studies have been performed and norms for gender and age are available. Normative studies have shown a gradual decline with age using the RAVLT, with more pronounced declines after the age of 70. Females have been found to score better than males on many of the RAVLT scoring categories, however, norms are stratified for age only.

Test retest reliability over a one- year interval was .55. The total score reliability for words recalled over the five trials of List A was .70 and .38 for recall of list B (Snow, Tierney, Zorzitto, Fisher & Reid, 1988). Validity studies have consistently shown lower levels of performance on the RAVLT with left hemisphere damage than with right hemisphere damage (Ivnik, Sharbrough, & Laws, 1988). Korsakoff's patients have consistently shown poorer recall over the five trials, but their recognition performance was closer to normal (Janowsky, Shimamura & Squire, 1989). Studies with Alzheimer's patients indicate a slower learning curve with lower recall and more intrusions. With the progression of the disease, lowered performance on this test is more pronounced (Mitrushina, Satz & Van Gorp, 1989).

The ***Wechsler Adult Intelligence Scale-Revised (WAIS-R) Vocabulary Subtest*** (Wechser, 1981). (See Appendix B) The Vocabulary subtest is the best single indicator of general intelligence (Groth-Marnet, 1997). It has been found to have a high degree of stability and is used as an indicator of a person's intellectual potential and to make an estimate of individuals premorbid level of functioning (Lezak, 1995). The maximum

score is 70 and high scores suggest high general intelligence, and that past ideas and concepts can be recalled adequately. On the other hand, low scores suggest a number of possible limitations such as low general intelligence, poor language development, English as a second language, limited education and or poor motivation. Split-half reliability for the Vocabulary subtest was .96. A number of validity studies of the WAIS-R have been conducted, including a wide number of criterion measures (Groth-Marnat, 1997).

The ***Trauma Symptom Inventory*** (TSI; Briere, 1995). This scale was chosen to measure the participants' perceived symptoms of trauma as the predictor variables. It is a 100-item self-administered questionnaire of psychological sequelae of traumatic events. The TSI comprises three validity scales: Atypical Response, Response Level and Inconsistent Response. The 10 clinical scales of interest here include: Intrusive Experiences, Defensive Avoidance, Anxious Arousal, Depression, Dissociation and Anger/Irritability. Five other scales, which are not used in this study, include Sexual Concern, Dysfunctional Sexual Behaviour, Impaired Self-Reference, and Tension Reduction Behaviour. Respondents complete a separate answer sheet and each symptom item is rated according to its frequency of occurrence over the prior 6 months using a 4-point scale ranging from 0 (*never*) to 3 (*often*). The inventory is psychometrically valid and norms have been established on four samples: general population, (N = 836); clinical, (N = 370); university, (N = 279) and military, (N = 3,659) with mean alpha coefficients = .86, .87, .84 and .85, respectively. Separate norms are available for different combinations of sex and age under 55 and 55 and over. The range of T Scores on the 5 scales is from approximately 40 to 95. The validity research exhibited construct, convergent, predictive and incremental validity (Briere, 1995). Construct validity was

shown by subjects who reported past trauma who scored higher on all 10 clinical scales than those who reported no trauma (Briere, 1995). Convergent validity was shown between TSI Defensive Avoidance and the Avoidance subscale of the Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979) with a correlation of .69. In addition, the Intrusive Experience score of the TSI and the IES had a high correlation $r = .67$. Criterion validity was established using two studies. The first study using known group methodology resulted in a positive prediction for PTSD of 92% and a negative prediction of PTSD of 91% (n = 449). The PTSD diagnosis was checked against scores of the IES and the Symptom Checklist (SCL; Foy, Sipprelle, Rueger, & Carroll, 1984). In another sample of psychiatric in-patients ($n = 105$), the TSI scales identified 89% of those independently diagnosed with Borderline Personality Disorder and 82% of non-borderlines (Briere, 1995).

The Adapted Trauma Symptom Inventory

For ethical considerations, the TSI was adapted to avoid direct questions on sexuality. Hence an adapted version was formed with permission from the copyright holder. This version is composed of the 72 items of the subscales required for this study. The items also include the validity scales and the critical items. In addition, the items and answer sheet are included in one form to avoid errors in marking, particularly for the older adults and war veterans. (See Appendix C).

The Alcohol Use Disorders Identification Test (AUDIT; Bador, de la Fuente, Saunders, & Grant, 1992). This scale was chosen as a measure of alcohol use. It is a brief multicultural screening tool for the early identification of problem drinkers. The AUDIT comprises two parts: a 10 item scale that include 3 questions on alcohol consumption, 4 questions on dependence symptoms, and 3 questions about alcohol- related problems

over the previous year. The second part is a Clinical Screening Procedure that includes a trauma history and a clinical examination. The AUDIT Part One may be used in an interview or in a paper and pencil format. (See Appendix D). The highest possible score for all 10 items is 41. Test studies have shown adequate reliability of .80 using a cross sectional sample of 989 undergraduate students (Fleming, Barry & MacDonald, 1991). Validity studies have been conducted using different cut off scores between 8 and 15 to meet the criteria for alcohol misuse of the Diagnostic Interview Schedule (DIS). Sensitivity and specificity vary greatly. However, the WHO recommends a total score of 11 or greater and the study by Fleming et al. (1991) produced a sensitivity of .84 with this cutting score. Only Part One of the AUDIT was used for the study.

Demographic Information included, age, gender, level of education completed, trauma type and distress. After assessing the first few participants it became evident that more than one traumatic event was experience. Participants ticked more than one box. A subjective distress score on a scale from 1 to 10 for each of the traumas with 1 = little distressing and 10 = extremely distressing was asked to be placed next to each trauma type in freehand rather than circle the number. The duration of trauma from one day to more than one year for each trauma was asked to be included in freehand. Illicit and prescribed drug use was also included. (See Appendix E)

A Trauma Severity Measure was obtained from the demographic questions. The number of traumas reported, plus the distress experienced for each of the traumas and the duration time were considered appropriate for this measure. These three indices; number of traumas, distress and duration were added, similar to the procedure using the Early Trauma Inventory (ETI) in the study by Bremner, Randall, Scott et al. (1995).

Procedure

Advertisements were placed at agencies in the Perth metropolitan area after ethical approval was granted by the various organisations involved. Initial ethical approval was obtained from the Edith Cowan University School of Psychology. These organisations included: Incest Survivor's Association, William Street Family Therapy Centre, Osborne Park Hospital, Returned Services League Anzac House and associated community branches, RSL War Veterans Homes, and the Harry Hunter Rehabilitation Centre. The author also gave a short presentation about the study for those agencies that requested it. Adults who had experienced a traumatic event and were either seeking counselling or part of the organisation were invited to volunteer in the study. Other advertisements were placed in community newspapers and volunteers responded to a contact number. (See Copies of the Advertisements in Appendix F). Screening for any of the exclusion criteria was made, and a brief outline of the procedure was given. Suitable appointment times were arranged at the centres or other suitable counselling rooms in the metropolitan area. On arrival, an outline of the procedure was detailed and all participants gave informed consent. (See Copy of Consent Form in Appendix G.) They were made aware that they may withdraw at any time. The first part of the assessment was the RAVLT, which was individually administered, followed by the WAIS-R Vocabulary subscale. The second part was the adapted TSI, followed by the AUDIT and the demographic questionnaire. The delayed recall trial of the RAVLT was then administered. The total time to complete the assessment varied from 30 to 45 minutes, depending on age. A total of 1 hour was allocated for each participant and any remaining time was given to discussion of any concerns about the study and a list of referrals for follow-up counselling regarding their trauma if that was appropriate.

The Statisical Package of the Social Sciences (SPSS) for Windows was used for all data screening and data analysis procedures.

RESULTS

Data Preparation

One participant refused to respond to part of the Rey Auditory Learning Test and two were short of time to complete the WAIS-R Vocabulary test. Means for the appropriate age group of the tests were used to replace these missing values for the two vocabulary standard scores from the WAIS-R and one mean score on the Trial 1-5 (Total Score) (Cumulative Learning), Trial 7 (Immediate Recall) and Trial 8 (Delayed Recall) of the RAVLT (Tabachnick & Fidell, 1989).

After examining the three validity scales of the Trauma Symptom Inventory (TSI), eight participants were found to have T-scores > 90 on the Atypical Response Scale and four participants had T- scores of > 75 on the Inconsistent Response Scale. The manual of the TSI (Briere, 1995) suggests that such scores reflect invalid responses. Therefore, these 12 participants were deleted from the study, leaving 136.

Reliability of the Scales

The present study adapted the TSI by including items from only 6 of the clinical scales and the 3 validity scales. Hence it was considered appropriate to analyse these scales to obtain estimates of the internal consistency reliability and check the generalisability of the original coefficients.

Cronbach's alpha was calculated for the 3 validity scales Atypical Response, Response Level, Inconsistent Response and the 6 clinical scales of Intrusive Experiences, Anxious Arousal, Defensive Avoidance, Depression, Dissociation and Anger /Irritability and are reported in Table 3. This table reports comparisons of Cronbach's alpha for both

the original scale using four different samples (Briere, 1995) and the present study using the reduced number of scales of the TSI.

Table 3

Comparison of Cronbach's Alpha for Original and Adapted Scale of the Trauma Symptom Inventory (TSI).

TSI Scale	Standardization sample (N = 828) Full Scale α	University sample (N =279) Full Scale α	Clinical sample (N = 370) Full Scale α	Navy recruit sample (N = 3,659) Full Scale α	Present sample (N = 134) Adapted Scale α
Atypical Response	.75	N/A	N/A	.75	.57
Response Level	.80	N/A	N/A	.78	.82
Inconsistent Response	.51	N/A	N/A	.55	.95
Intrusive Experience	.89	.87	.90	.87	.90
Defensive Avoidance	.90	.89	.88	.87	.86
Anxious Arousal	.86	.84	.87	.82	.85
Depression	.91	.87	.90	.88	.90
Dissociation	.82	.86	.88	.84	.82
Anger/Irritability	.90	.90	.89	.88	.91

The original internal consistency reliability for the Alcohol Use Disorder Identification Test (AUDIT) using 10 items was a Cronbach's Alpha of .80 (Fleming, Barry & MacDonald, 1991). The internal reliability in the present sample was α = .95.

Data Screening

Three univariate outliers were shown on the stem and leaf plots for two of the predictors, Depression and Dissociation. In addition, three extreme low scores were found in the criterion verbal learning Trial 1- 5 Total scores (Cumulative Learning) and two extreme

low scores on verbal memory Trial 7 (Immediate Recall). The scores were modified by recoding them to one unit smaller or larger than the next most extreme score (Tabachnick & Fidell, 1987). Normality and linearity were found to be satisfactory.

Two multivariate outliers were revealed using a Mahalanobis distance values at α = .001. These were deleted from the data, remaining with N = 134, 59 males and 75 females. Scatterplots were performed and showed acceptable linearity. Descriptive data of the variables are summarised in Table 4.

Examination of the Bivariate Correlations.

The intercorrelation matrix for all the RAVLT scores, and all the predictor variables are shown in Table 5. As can be observed from the table, there was no evidence of multicollinearity among the predictor variables. However, predictor variables of the trauma symptoms of the adapted TSI showed significant relationships with one another.

Hierarchical Multiple Regression Analyses

Four hierarchical regression analyses were employed to determine the predictive power of three sets of variables; the demographic set, the trauma severity set and the set of trauma symptoms from the adapted TSI on verbal measures learning and memory. These were; a) the RAVLT Trial (1-5) Total Score (Cumulative Learning), b) the RAVLT Trial 1 (Initial Learning), c) the RAVLT Trial 7 (Immediate Recall) and d) the RAVLT Trial 8 (Delayed Recall).

Demographic variables were entered first to provide a baseline level of prediction. The trauma severity variables were added in the second step because these factors have priority in the DSM-IV diagnostic system. The DSM-IV criterion A, primarily focuses on the trauma event before response to the trauma symptoms are

considered. Finally, the set of trauma symptoms from the adapted TSI was added in the third step. These steps were used in each of the four hierarchical regressions, one for each criterion variable of learning and memory.

Table 4.

Means and Standard Deviation Scores of the Variables (N = 134)

VARIABLES	M	SD
DEMOGRAPHICS		
AGE	48.47	18.19
WAIS-R Vocabulary [a]	12.20	2.85
AUDIT [b]	10.75	11.73
TRAUMA SEVERITY		
Number of Traumas	2.15	1.41
Distress	17.37	10.54
Duration (Years)	5.14	1.48
ADAPTED TSI [c]		
Intrusive Experience	62.10	12.56
Defensive Avoidance	59.50	10.80
Anxious Arousal	59.36	10.50
Depression	58.49	10.31
Dissociation	62.80	12.43
Anger/Irritability	57.02	10.64
LEARNING & MEMORY TRIALS		
RAVLT [d] Trial (1-5) Total Score	55.62	9.86
RAVLT [d] Trial 1. (List A)	54.60	11.18
RAVLT [d] Trial 7. (List A)[1]	52.81	9.82
RAVLT [d] Trial 8. [2]	52.78	10.80

WAIS-R[a] = Wechsler Adult Intelligence Scale-Revised. Possible score range from 0 – 20.
AUDIT[b] = Alcohol Use Disorder Identification Test. Possible score range from 0 – 41.
Adapted TSI[c] = Trauma Symptom Inventory. Possible score range from 40 – 95.
RAVLT[d] = Rey Auditory Verbal Learning Test. Possible score range from 24 – 76.

[1] Retroactive Interference
[2] Delayed Recall

Table 5.
Correlations between Sets of Demographic Variables, Trauma Severity, Trauma Symptoms and Verbal Learning and Memory Trials. (N = 134)

		1.	*2.*	*3.*	*4.*	*5.*	*6.*	*7.*	*8.*	*9.*	*10.*	*11.*
1.	Age	1.00	-.11	.11	-.30**	-.26**	.11	-.01	.05	-.03	.11	-.26**
2.	Gender	-.11	1.00	.13	.19*	-.37**	-.13	.14	.15	.13	.08	.02
3.	WAIS-R [a] (Vocabulary)	.11	.13	1.00	.46**	-.20*	-.02	.05	.03	.06	-.05	-.07
4.	Education	-.30**	.19*	.46**	1.00	-.04	.04	.11	.07	.12	-.03	.02
5.	AUDIT [b] (Alcohol Use)	-.26**	-.37**	-.20*	-.04	1.00	-.07	-.03	-.13	-.03	.04	.25**
6.	Drugs	.11	-.13	-.02	.04	-.07	1.00	.11	.16	.10	.03	.16
7.	Trauma Severity	-.01	.14	.05	.11	-.03	.11	1.00	.74**	.99**	.43**	.29**
8.	Number of Traumas	.05	.15	.03	.07	-.13	.16	.74**	1.00	.68	.20*	.21*
9.	Distress	-.03	.13	.06	.12	-.03	.10	.99**	.68**	1.00	.32**	.27**
10.	Duration	.11	.08	-.05	-.03	.04	.03	.43**	.20*	.32**	1.00	.25**
11.	Total TSI[c] Score (Adapted)	-.26**	.02	-.07	.02	.25**	.16	.29**	.21*	.27**	.25**	1.00
12.	Intrusive Experience	-.14	-.07	-.07	-.00	.22*	.11	.28**	.18*	.26**	.24**	.83**
13.	Defensive Avoidance	-.12	.05	-.05	-.01	.22*	.09	.28**	.22*	.25**	.29**	.85**
14.	Anxious Arousal	-.20*	.07	-.04	.07	.12	.26**	.23**	.16	.22**	.14	.86**
15.	Depression	-.29**	.00	-.02	.02	.20*	.25**	.29**	.23**	.27**	.23**	.84**
16	Dissociation	-.27**	.05	-.05	.03	.20*	.10	.25**	.21*	.22*	.27**	.88**
17	Anger/Irritability	-.33**	.01	-.11	.00	.30**	.03	.14	.07	.13	.10	.79**
18.	RAVLT [d] Trial I (List A)	-.08	.29**	.40**	.32**	-.21*	.01	-.07	.04	-.05	-.23**	-.16
19.	RAVLT Trial 6 (List B) [1]	.05	.15	.23**	.09	-.17*	-.10	.04	.08	.04	-.02	-.15
20.	RAVLT Trial 7 [2]	.05	.39**	.32**	.25**	-.10	-.18*	-.10	-.06	-.09	-.13	-.16
21.	RAVLT Trail 8 [3]	.17	.28**	.21*	.11	-.09	-.15	-.04	-.00	-.03	-.11	-.16
22.	RAVLT Trial (1-5) Total	.31**	.31**	.39**	.13	-.29**	-.06	-.07	-.01	-.05	-.16	-.24**

* $p < .05$ (2-tailed) ** $p < .01$ (2 tailed)

Continued

WAIS-R [a] = Wechsler Adult Intelligence Scale-Revised, AUDIT [b] = Alcohol Use Disorder Identification Test, TSI[c] = Trauma Symptom Inventory.
RAVLT [d] = Rey Auditory Verbal Learning Test.
[1] Proactive Interference, [2] Retroactive Interference, [3] Delayed Recall

		12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.
1.	Age	-.14	-.12	-.20*	-.29**	-.27**	-.33**	-.08	.05	.05	. 17	.31**
2.	Gender	-.07	.05	.07	.00	.05	.01	.29**	.15	.39**	.28**	.31**
3.	WAIS-R[a] (Vocabulary)	-.07	-.05	-.04	-.02	-.05	-.11	.40**	.23**	.32**	.21*	.39**
4.	Education	-.00	-.01	.06	.02	.03	.00	.32**	.09	.25**	.11	.13
5.	AUDIT[b] (Alcohol Use)	.22*	.22*	.12	.20*	.20*	.30**	-.21*	-.17*	-.10	-.09	-.29**
6.	Drugs	.11	.09	.26**	.25**	.10	.03	.01	-.10	-.18*	-.15	-.06
7.	Trauma Severity	.28**	.28**	.23**	.29**	.25**	.14	-.07	.04	-.10	-.04	-.07
8.	Number of Traumas	.18*	.22*	.16	.23**	.21*	.07	.04	.08	-.06	-.00	-.01
9.	Distress	.26**	.25**	.22**	.27**	.22*	.13	-.05	.04	-.09	-.03	-.05
10.	Duration	.24**	.29**	.14	.23**	.27**	.10	-.23**	-.02	-.13	-.11	-.16
11.	Total TSI[c] Score (omitted)	.83**	.85**	.86**	.84**	.88**	.79**	-.15	.15	-.16	-.16	-.24**
12.	Intrusive Experience	1.00	.74**	.65**	.61**	.62**	.55**	-.18*	-.20*	-.14	-.13	-.15
13.	Defensive Avoidance	.74**	1.00	.68**	.64**	.70**	.55**	-.18*	-.14	-.13	-.13	-.17
14.	Anxious Arousal	.65**	.68**	1.00	.73**	.70**	.63**	-.08	-.07	-.04	-.04	-.16
15.	Depression	.61**	.64**	.73**	1.00	.72**	.59**	-.12	-.20*	-.17	-.16	-.23**
16	Dissociation	.62**	.70**	.70**	.72**	1.00	.69**	-.11	-.10	-.22*	-.20*	-.28**
17	Anger/Irritability	.55**	.55**	.63**	.59**	.69**	1.00	-.12	-.04	-.11	-.16	-.23**
18.	RAVLT[d] Trial 1 (List A)	-.18*	-.18*	-.08	-.12	-.11	-.12	1.00	.28**	.46**	.44**	.69**
19.	RAVLT Trial 6 (List B)[1]	-.20*	-.14	-.07	-.20*	-.10	-.04	.28**	1.00	.09	.21*	.26**
20.	RAVLT Trial 7[2]	-.14	-.13	.04	-.17	-.22*	-.11	.46**	.09	1.00	.78**	.65**
21.	RAVLT Trail 8[3]	-.13	-.13	-.04	-.16	-.20*	-.16	.44**	.22*	.78**	1.00	.68**
22.	RAVLT Trial (1-5) Total	-.15	-.17	-.16	-.23**	-.28**	-.23**	.69**	.26**	.65**	.68**	1.00

* $p < .05$ (2-tailed) ** $p < .01$ (2 tailed)

WAIS-R[a] = Wechsler Adult Intelligence Scale-Revised; AUDIT[b] = Alcohol Use identification Test; TSI[c] = Trauma Symptom Inventory; RAVLT[d] = Rey Auditory Verbal Learning Test.
[1] Proactive Interference, [2] Retroactive Interference, [3] Delayed Recall

Prediction of Verbal Learning

The first hierarchical multiple regression using the set of demographic variables in step 1 and RAVLT Trial 1-5 (Total Score) (Cumulative Learning) as the criterion was significant ($R^2 = .32$, $F(6,127) = 9.83$, $p < .001$). This was also the case with the addition of the trauma severity set of variables, ($R^2 = .36$, $F(9,124) = 7.74$, $p < .001$). This was followed by the set of trauma symptoms in step 3 with $R^2 = .39$, $F(15,118) = 4.98$, $p < .001$. The only significant predictive demographic variables were age, gender and IQ. Duration of the trauma, part of the set of trauma severity variables, approached significance at $p = .06$ and from the set of trauma symptoms, dissociation was the only symptom approaching significance, $p = .09$. (See Table 6).

A hierarchical multiple regression using the same sets of predictor variables and RAVLT Trial (1 Initial Learning) as the criterion resulted in the set of demographics accounting for 24% of the variance, ($F(6,127) = 6.74$, $p < .001$), the set of trauma severity variables added 6% ($F(9,124) = 5.81$, $p < .001$), and the trauma symptoms accounting for an additional 2% of the predictive power ($F(15,118) = 3.60$, $p < .001$). Specific significant variables responsible for the variance included gender and IQ, while duration of the trauma approached significance at $p = .06$. No trauma symptom approached significance for predicting initial learning. See Table 7 for details of the regression results.

Table 6.

Results of the Hierarchical Multiple Regression Analysis of the Demographics, Trauma Severity and the Perceived Trauma Symptoms to Performance on the RAVLT (Trial 1-5) (Cumulative Learning) N = 134.

Predictor	Variables	*B*	Beta	Step	R^2 Changes
Demographics	Gender	6.60	.33***	1	
	Age	.16	.30**	1	
	WAIS-R (Vocabulary)	1.02	.29**	1	
	Education	.24	.03	1	
	Audit (Alcohol Use)	-.00	-.01	1	
	Drugs	-.26	-.03	1	.32***
Trauma Severity	Number of Traumas	.03	.00	2	
	Distress	-.04	-.04	2	
	Duration	-1.04	-.16 (.06)	2	.04***
Trauma Symptoms	Intrusive Experience	.11	.14	3	
	Defensive Avoidance	-.04	-.04	3	
	Anxious Arousal	-.01	-.01	3	
	Depression	-.01	-.01	3	
	Dissociation	-.18	-.22 (.09)	3	
	Anger/Irritability	.02	.02	3	.03***

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

Prediction of Verbal Memory

Trial 7 (Immediate Recall) of the RAVLT was used as a criterion for the same three sets of variables as above to predict verbal immediate memory. In the first step, the demographic variables predicted a similar amount of variance as

Table 7.

Results of the Hierarchical Multiple Regression Analysis of the Demographics, Trauma Severity and the Perceived Trauma Symptoms to Performance on the RAVLT Trial 1. (Initial Learning) N = 134.

Predictor	Variables	*B*	Beta	Step	R^2 Changes
Demographics	Gender	5.39	.24**	1	
	Age	-.05	-.09	1	
	WAIS-R (Vocabulary)	1.29	.32***	1	
	Education	.90	.08	1	
	Audit (Alcohol Use)	-.00	-.00	1	
	Drugs	.78	.07	1	.24***
Trauma Severity	Number of Traumas	1.02	.13	2	
	Distress	-.13	-.12	2	
	Duration	-1.24	-.16 (.06)	2	.06***
Trauma Symptoms	Intrusive Experience	.01	.01	3	
	Defensive Avoidance	-.15	-.14	3	
	Anxious Arousal	.03	.02	3	
	Depression	-.07	-.07	3	
	Dissociation	.05	.05	3	
	Anger/Irritability	-.02	-.02	3	.02***

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

before ($R^2 = .27$, $F(6,127) = 7.73$, $p < .001$), with gender, IQ and Alcohol use as significant variables and drug use approaching significance at $p = .08$.

The second step, ($R^2 = .30$, $F(9,124) = 6.01$, $p < .001$) had no significant weights for any specific variables of the set of trauma severity measures. The third step, with the inclusion of the trauma symptoms, accounted for a further 6% of variance, ($F(15,118) = 4.52$, $p < .001$). See Table 8 for details. Significant

trauma symptoms predicting deficits of verbal memory included dissociation, $p < .001$, and anxious arousal, $p < .01$.

Another analysis to predict memory using Trial 8 of the RAVLT (Delayed Recall) was followed in the same sequence as above. Using delayed recall as the criterion, the set of demographic variables reached an $R^2 = .17$, $F(6,127) = 4.38$, $p < .001$, with gender and age significant predictor variables, with alcohol use approaching significance at $p = .06$ as well as drug use at $p = .07$. The trauma severity set was significant as well ($R^2 = .19$, $F(9,124) = 3.34$, $p < .001$), but no individual variables were significant.

Trauma symptoms accounted for an additional 5% of the variance, ($F(15,118) = 2.60$, $p < .01$), with Anxious Arousal significantly predicting verbal memory deficits for the full set of regression weights. See Table 9.

Table 8.

Results of the Hierarchical Multiple Regression Analysis of the Demographics, Trauma Severity and the Perceived Trauma Symptoms to Performance on the RAVLT Trial 7. (Immediate Recall) N = 134.

Predictor	Variables	*B*	Beta	Step	R^2 Changes
Demographics	Gender	8.17	.41***	1	
	Age	.06	.11	1	
	WAIS-R (Vocabulary)	.81	.24**	1	
	Education	1.01	.11	1	
	Audit (Alcohol Use)	.15	.18*	1	
	Drugs	-1.46	-.15 (.08)	1	.27***
Trauma Severity	Number of Traumas	.37	.05	2	
	Distress	-.12	-.13	2	
	Duration	-.39	.06	2	.04***
Trauma Symptoms	Intrusive Experience	.04	.05	3	
	Defensive Avoidance	-.08	-.08	3	
	Anxious Arousal	.26	.29*	3	
	Depression	-.04	-.04	3	
	Dissociation	-.27	-.34**	3	
	Anger/Irritability	.02	.02	3	.06***

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

Table 9.

Results of the Hierarchical Multiple Regression Analysis of the Demographics, Trauma Severity and the Perceived Trauma Symptoms to Performance on the RAVLT Trial 8 (Delayed Recall) N = 134.

Predictor	Variables	*B*	Beta	Step	R^2 Changes
Demographics	Gender	7.08	.32***	1	
	Age	.13	.22*	1	
	WAIS-R (Vocabulary)	.54	.14	1	
	Education	.42	.04	1	
	Audit (Alcohol Use)	.17	.18 (0.6)	1	
	Drugs	-1.8	-.16 (0.7)	1	.17***
Trauma Severity	Number of Traumas	.48	.06	2	
	Distress	-.05	-.05	2	
	Duration	-.66	-.09	2	.02***
Trauma Symptoms	Intrusive Experience	.01	.02	3	
	Defensive Avoidance	-.12	-.02	3	
	Anxious Arousal	.33	.33*	3	
	Depression	-.02	-.02	3	
	Dissociation	-.20	-.23	3	
	Anger/Irritability	-.09	-.09	3	.05***

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

Table 10 provides a summary of the outcomes of the four hierarchical multiple regressions predicting verbal learning and memory.

Table 10

Summary of Prediction of Verbal Learning and Memory on Performance to Trials of the RAVLT.

	Prediction of Verbal Learning		Prediction of Verbal Memory	
	Trial 1-5.	***Trial 1.***	***Immediate Recall***	***Delayed Recall***
Demographics	32% Gender*** IQ** Age**	24% Gender** IQ***	27% Gender *** IQ** Alcohol Use* Drug Use (p = .08)	17% Gender*** Age* Alcohol Use (p = .06) Drug Use (p = .07)
Trauma Severity	4% Duration (p =.06)	6% Duration (p=.06)	4%	4%
Trauma Symptoms	3% Dissociation (p = .09)	2%	6% Anxious Arousal* Dissociation**	5% Anxious Arousal*

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

Discussion

The general aim of this study was to investigate the specific symptoms of trauma rather than a global PTSD diagnosis to predict deficits in verbal learning and memory. A first stage in a series of hierarchical regression equations examined the predictive ability of several demographic factors. After the demographic factors were included into the predictive equation, a second set of variables for trauma severity was added to determine if there was a significant increase in variance. Thirdly, after partially out the sets of demographic and trauma severity variables, the trauma symptoms of intrusive experience, defensive avoidance, anxious arousal, depression, dissociation and anger were added to the prediction of verbal learning and memory deficits.

Demographic Factors

Overall, the four hierarchical multiple regressions resulted in the set of demographic variables predicting the most variance in the measures of verbal learning and memory ranging from 17% to 32% (See Table 10 for a Summary of Results). Previous studies in this area have matched index and control groups, suppressing the variation of demographic variables. This study explored the contribution of the specific variables making up the set of demographic factors. From this set, gender was significant in predicting verbal learning and memory for all measures. An examination of gender found that females scored higher means than males on all trials of the RAVLT. (See summary table Appendix H). This is consistent with findings by both Vakil and Blachstein (1997) using the RAVLT and Kramer, et al. (1997) using the California Verbal Learning Test that females score better than males in verbal learning and memory tests. However,

two previous studies examining PTSD and memory combined males and females in the same group (Bremner, Randall, Scott, et al. 1995 and Vasterling, et al. 1998). Future studies examining verbal measures need to consider gender issues explicitly.

Trauma symptoms and gender have not been examined in previous studies in this area and must be noted that the instruments used do not give norms for PTSD global scores for males and females or age. Appendix H shows no significant differences between trauma symptoms and gender because the TSI takes this into account hence giving more valid results.

IQ significantly predicted verbal learning and immediate recall but not delayed recall. However, the present study did not replicate the negative correlations of IQ in relation to trauma symptoms that McNally and Shin (1995) reported with PTSD symptoms.

Age was another significant predictor from the set of demographic variables of one measure each of verbal learning and memory, Trial 1-5 and delayed recall. These findings are consistent with the literature, particularly that related to the function of the phonological loop that is not affected with age (Fisk & Warr, 1996). In addition, it must be noted that the present study was based on voluntary participation in response to advertisements and older participants motivated to help in the study were possibly functioning better than those who did not want to participate. This may partially explain the significant positive relationship with age in relation to verbal learning and memory.

Alcohol use significantly predicted the immediate recall trial of the RAVLT and approached significance for delayed recall ($p = .06$). Alcohol use did not significantly predict verbal learning. A similar pattern appeared for drug

use, which however only approached significance. Although not significant, these results are consistent with the known effects of alcohol use on memory (Parsons, 1987; Uddo & Gouvier, 1990; Waugh, et al. 1989).

Trauma Severity Factors

The set of trauma severity factors significantly predicted an increased amount of variance of verbal learning and memory once the demographic variables were partialled out in step 1 of the equation. As can be seen from Table 10, trauma severity added to the prediction of verbal learning 4% to 6% and 2% to 4% for verbal memory.

The type of trauma was not significant in predicting either verbal learning or memory. Duration of the trauma approached significance ($p = .06$) only for the prediction of verbal learning, and not for memory. This is the first study to consider a set of trauma severity variables in relation to the trauma events as well as a response of specific trauma symptoms versus a diagnosis of PTSD.

Trauma Symptoms

The third objective of this study was to examine the trauma symptoms of intrusive experience, defensive avoidance, anxious arousal and also depression, dissociation and anger after partialling out the sets of demographic and trauma severity variables for predictions of verbal learning and memory. Trauma symptoms overall significantly contributed to the prediction of verbal learning and memory by an additional 2% to 3% of the variance in verbal learning and 5% in 6% for verbal memory. Although the six trauma symptoms accounted for a small portion of the variance in predicting verbal learning and memory, these findings give perspective and add to the body of knowledge in this area.

Of particular interest, the results show the contribution of the symptom of dissociation as a significant predictor of Immediate Recall. The symptom of dissociation has not been measured in previous studies using PTSD and non-PTSD groups with the exception of Stein et al. (1999). However, this study grouped participants into child sexual abuse survivors and healthy comparison, with only 77% of the sexual abuse survivors diagnosed with PTSD. Participants in the previous studies who were categorised into the PTSD group may well have had symptoms of dissociation. The present study supports both Carlson (1997) and Perry et al. (1995) who recommend that the symptom of dissociation to be measured in relation to trauma and PTSD.

The symptom of anxious arousal reached significance only for prediction of verbal memory and not verbal learning. Both Trial 7 Immediate Recall and Trial 8 Delayed Recall suggest that anxious arousal affects the mechanism of retrieval of verbal information.

Interestingly, from the predictive equation the symptoms of Intrusive Experience, Defensive Avoidance, Depression and Anger/Irritability were found not to contribute to verbal learning and memory significantly.

An examination of the means of the trauma symptoms (See Table 4) indicated that the scores are below clinical significance. According to Briere (1995) *T* scores at or above 65 are considered clinically significant. Although a wide range of trauma symptoms were obtained in this sample, perhaps a clinical sample may have had higher trauma symptoms scores and led to more predictable variance in verbal learning and memory.

Integrating Physiological and Psychological Findings

This study is the first attempt to explore a psychological perspective that may be congruent with the biological findings thus far. There is a suggestion that the symptom of dissociation may be in some way related to impaired immediate recall that may in turn be linked to the physiological mechanism of an under supply of blood in the Broca's area found by Rauch, van Kolk, Fisher et al. (in press). Further research needs to investigate the process that links the rehearsal process in the phonological loop to physiological changes. Such a speculative model could provide a means to explain the findings on verbal learning and memory deficits.

Research into the function of the frontal lobe and limbic systems suggest that there are feedback mechanisms that support new learning and consolidation of information into long-term memory (Tulving, Markowitsch, Craik, Habib & Houle, 1996). It appears that trauma symptoms particularly anxious arousal may interupt this process.

A factor that was not considered in this study and may have contributed to the prediction of verbal learning and memory is the function of the Central Executive (CE) from Baddeley's (1986) Working Memory Model. The Central Executive helps to maintain and control information through active attention as the executive function of the frontal lobe. In addition, if damage to neurones through the trauma response occurs in the frontal lobe, further problems in initiating, inhibiting, sequencing, planning and organising of the material to be encoded and retrieved would be hampered.

The model proposed by Perry, et al. (1995) examining the two physiological response systems, the parasympathetic associated with the release

of acetylcholine and the dissociation trauma symptom, and the sympathetic system associated with the release of norepinephrine and the trauma symptom of hyperarousal, needs further investigation in relation to verbal learning and memory.

Methodological Issues

Methodological Limitations of the Present Study

The participants in this study were not randomly selected, hence a biased sampling of volunteers may have occurred. Research in the area of trauma is very difficult and requires sensitivity on the part of the researcher and ethical considerations so as not to further traumatise participants. Hence, only those clients that freely responded to an advertisement and who were highly motivated participated. This is a limitation that has no resolution, but needs to be considered in light of the generalisability of the results.

In addition, while the present study employed a correlational design to examine predictors of verbal learning and memory, relationships between the significant predictors on the series of criterion must not be assumed to be causal.

The gender of the researcher may need to be considered when doing research on trauma. A female researcher may have influenced the way males responded to some of these questions in a way that a male researcher would not do. Ideally, both male and female interviewers would be used and counterbalanced across participants. Some participants may respond to questions in a socially desirable manner, while others may be very open and honest. By selecting the TSI as the assessment tool to measure trauma symptoms, participants could be excluded if they scored above acceptable limits on the three

validity scales. However, questions on alcohol and drug use and other demographic variables were not confirmed by another source.

The question on drug use could have been expanded with a list of different illicit drugs. Exploration of the effects of different patterns of use (ie single vs polydrug use) on verbal deficits might then may have been possible. Previous studies reported drug use but did not explicitly consider the influence of drug use on learning and memory.

Ethnic and cultural factors were not addressed in the demographic questions and may contribute to a degree on the dependent measures. However, participants were not of clearly indigenous origins and all had good knowledge of the English language to read the questionnaire and understand conversation.

Trauma severity measures no doubt have their limitations, given that they are retrospective in nature. In addition, any memory impairment that was present may lead to under reporting of the severity and the number of traumatic events (van der Kolk, 1996). Measures of verbal learning and memory as they might have been prior to the traumas were not addressed in the present study or in previous studies. The question thus remains as to whether these deficits were present before the traumatic events occurred.

Another factor not considered in the present study was that some participants had received treatment for their trauma symptoms at some point, while others had never spoken to anyone about their trauma and presented to assist with the present research. Previous studies in this area have not been explicit as to the inclusion of participants with previous treatment or no treatment. Rather they have been described as new admissions either in an out-patient or in-patient setting (Bremner, Scott, Delaney, et al. 1993;

Bremner,Randall, Scott, et al. 1995; Uddo et al. 1993;Yehuda, Keefe, et al. 1995). Ideally the new admissions had not received treatment for their symptoms prior to admission or clinic, however this is not clear.

The age of onset of the trauma has not been a consideration in any of the studies in this area. Recently, childhood traumas and a diagnosis of PTSD have been associated with adverse brain development (Boring, et al. 1999) and significant alteration of the biological stress systems (De Bellis, et al. 1999). While the trauma of adult survivors of childhood sexual and physical abuse occurred at an earlier developmental stage, adult combat veterans appeared to have similar anatomical changes related to the traumatic events ie smaller hippocampal volumes. The age of onset of the trauma and symptoms of PTSD may need to be explored further in future research using a longitudinal design and be another factor in the set of trauma severity variables.

Methodological Strengths of the Present Study

Despite the limitations discussed above, the study has a number of methodological strengths. These were: a) the sample size, b) the research design using demographics as predictors, c) the use of trauma severity measures, d) the use of the Trauma Symptoms Inventory including its validity checks, e) the addressing of other comorbid symptoms, and the f) sequencing of the assessment. Each of these will be addressed in turn.

The sample size in previous studies comprised groups of 12 to 26 participants. The present study in total interviewed 148 participants from the community who perceived they had experienced a traumatic event. This is the first study to undertake such a large sample size of participants who had experienced trauma in relation to verbal learning and memory.

The present study used a cross-sectional, correlational design, and examined the predictive relationship of sets of demographic, trauma severity and trauma symptom variables with verbal learning and memory. Demographic variables contributed significantly to the predicted variance, with the suggestion that gender plays an important role in the predictability. Previous studies compared groups with PTSD and without PTSD by matching participants on demographic factors. The significant findings add to the body of knowledge and may guide future research in this area.

Although the trauma severity measure has its limitations, previous studies have confounded trauma severity with the trauma symptoms. The present study has made the distinction and investigated the predictive power of each in relation to both verbal learning and memory, which have not been examined in other studies. In addition, although previous studies have focused on a particular type of trauma, there is an implicit assumption that the trauma severity of the event at the time was perceived to be equally distressing for all participants. The fact that this study included various types of perceived traumatic events means that trauma severity was an important variable to include as a predictor, even given its limitations.

The TSI is a multidimensional measure of trauma symptoms, unlike a global measure of PTSD. Unlike other measures, the TSI includes three validity scales, which allow the elimination of invalid test results that would otherwise have been used in other studies. Of a total of 148 volunteer participants, 12 test results or 8% were invalid. That is, responses that may have been indiscriminately omitted or answered in inconsistent, atypical ways. By excluding them in the present study, the results maybe considered more valid.

Previous studies found a relationship between a PTSD diagnosis and verbal memory deficits, although some studies included comorbid disorders with a PTSD diagnosis (Bremner, Randall, Scott et al. 1995; Vasterling, et al. 1998). Other studies excluded participants with comorbidities (Uddo et al. 1993; Bremner, Scott, Delaney, et al. 1993; Yehuda et al. 1995). Since the TSI includes scales for intrusive experiences, defensive avoidance, anxious arousal, as well as depression, dissociation and anger/irritability these symptoms could be included as predictors. Participants with symptoms of depression in previous studies were described as a variant of PTSD, making direct comparisons difficult. As seen from the present study the symptoms of depression and anger did not make a significant contribution to the equations for predicting verbal learning and memory.

Given that previous studies have focused on comparing groups with PTSD and without PTSD, the interview to confirm PTSD diagnosis was one of the first assessments. Therefore, participants were exposed to memories of the trauma and symptoms related to the trauma. The verbal learning and memory tests followed. Such a sequence may exacerbate the interference with the processes of verbal learning and memory. Hence, in the present study participants were informed that the interview would not include discussion of the trauma event and the initial assessment was the verbal learning and memory tests. The TSI was administered last. However, the delayed recall trial followed the TSI and there thus may have had a greater impact on delayed retrieval.

Future Issues

So far considerable evidence supports a relationship between stress and memory in animal studies (Drugan, Ryan, et al. 1984; Seligman & Maier, 1967). High cortisol levels in human studies in relation to stress and memory deficits have also been reported (de Quervain, et al. 2000; Kirschbaum, Wolf, May et al. 1996; Seeman et at. 1997). Other neurobiological findings such as levels of cortisol, norepinephrine, epinephrine and dopamine as a result of PTSD are inconclusive (Golier & Yehuda, 1998; Lemieux & Coe, 1995; Liberzon et al, 1999). Morphological changes using MRI with PTSD groups (both war veterans and childhood sexual and physical abuse) have been associated with decreased in hippocampus volume and verbal memory deficits (Bremner, Randall, Scott, et al.1995; Bremner, Randall Vermetten, et al. 1998). A PTSD diagnosis has been associated with memory, learning and attentional problems with inconsistencies in the specific details of the findings (Vasterling, et al.1998). The present findings suggest that the symptom of dissociation is a significant predictor of deficits in immediate recall and possibly in verbal learning, but further studies need to examine a clinical population confirmed PTSD cases. Dissociation needs to be assessed as a separate symptom rather than as part of the avoidance or re-experiencing criteria of the DSM-IV for PTSD diagnosis (Carlson, 1997; Perry, et al. 1995). Furthermore, it seems useful that the symptom of dissociation be integrated into a conceptual framework of trauma (Carlson, 1997), especially in relation to changes in verbal memory. Perry et al. (1995) described the adaption style to trauma as a continuum from arousal to dissociation and given that anxious arousal made a significant contribution to predicting changes in verbal

memory, this trauma symptom may make a valuable contribution if integrated into a framework.

"Trauma severity" has been conceptualised either as the trauma per se (Bremner, Scott, Delaney, et al. 1993) or as the response symptoms to trauma (Bremner, Randall, Scott, et al. 1995; Jenkins, et al.1998; Uddo, et al. 1993; Vasterling et al, 1998; and Yehuda, Keefe, et al. 1995). There is a need for clarification and theoretical development to guide future research. McFarlane and de Girolamo (1996) discuss the difficulties associated with measurement of traumatic exposure, and this work would be a good starting point.

Implications for Future Research

The symptoms of trauma in the form of PTSD often last a lifetime. The sequelae and implications of deficits in verbal learning and memory need to be explored. Difficulty learning new verbal material would have serious implication in a number of settings, in particular children's early academic performance (see review by Frayne, 1999), individuals in "talking" therapy, and in court-room settings (de Quervain, et al. 2000) to mention a few. Hence early diagnosis would be important. Future research is recommended in such settings in light of this exploratory study. Developing treatment programs for individuals suffering with trauma symptoms may focus on specific symptoms such as dissociation and anxious arousal. Such programs may therefore aid in developing an early intervention program such as functional and positive adaptive coping skills which may alleviate the negative trauma symptoms and consequently ameliorate long term cognitive impairment of verbal learning and memory deficits. In addition, more focus on non-verbal techniques to enhance the processing of the trauma in

therapy maybe helpful in alleviating the life long symptoms these people experience.

Future studies need to focus on specific symptoms of trauma because of the complex heterogeneous nature of the classification of PTSD. Symptoms of dissociation and anxious arousal in relation to verbal deficits need further investigation using a collaborative perspective, such as a combination found in a neuropsychophysiological model. Methodological designs of future studies into verbal deficits need careful consideration of demographic factors, particularly gender. A model may be developed which would offer a theoretical foundation from which to begin a more systematic examination of trauma symptoms and verbal memory function. Furthermore, deficits in verbal learning and memory in relation to specific trauma symptoms may suggest that neuropsychobiological research may help to establish better models of the process by which trauma and stress influence memory processes. Finally, longitudinal rather than cross sectional methods may have an important role in future research.

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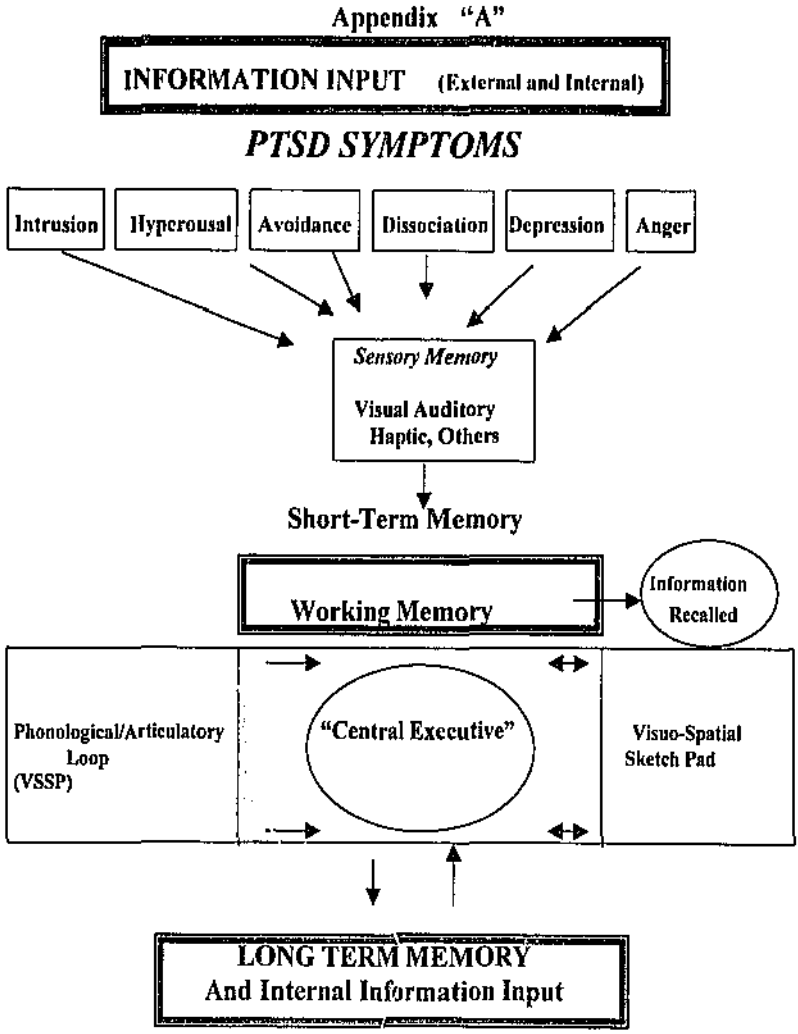

Figure 1. Diagrammatic representation of the flow of information and PTSD symptoms through the memory system. Adapted and Revised from Atkinson & Schiffrin, 1971; Baddelely, 1986.

Appendix "B"

Rey Auditory Verbal Learning Test

&

Wechsler Adult Intelligence Scale – R. Vocabulary

List A	LIST B
DRUM	DESK
CURTAIN	RANGER
BELL	BIRD
COFFEE	SHOE
SCHOOL	STOVE
PARENT	MOUNTAIN
MOON	GLASSES
GARDEN	TOWEL
HAT	CLOUD
FARMER	BOAT
NOSE	LAMB
TURKEY	GUN
COLOUR	PENCIL
HOUSE	CHURCH
RIVER	FISH

Vocabulary Score

1.	Bed	
2.	Ship	
3.	Penny	
4.	Winter	
5.	Breakfast	
6.	Repair	
7.	Fabric	
8.	Assemble	
9.	Enormous	
10.	Conceal	
11	Sentence	
12.	Consume	
13.	Regulate	
14.	Terminate	
15.	Commence	
16.	Domestic	
17.	Tranquil	
18.	Ponder	
19.	Designate	
20.	Reluctant	
21.	Obstruct	
22.	Sanctuary	
23.	Compassion	
24.	Evasive	
25.	Remorse	
26.	Perimeter	
27.	Generate	
28.	Matchless	
29.	Fortitude	
30.	Tangible	
31.	Plagiarise	
32.	Ominous	
33.	Encumber	
34.	Audacious	
35.	Tirade	
	Total Score	**Max = 70**

Appendix "C"

Adapted Trauma Symptom Inventory

TRAUMA SYMPTOM INVENTORY

Adapted from John Briere[3]

0 1 2 3
Never Often

In the last 6 months, how often have you experienced:

1.	Nightmares or bad dreams	0	1	2	3
2.	Trying to forget about a bad time in your life	0	1	2	3
3.	Irritability	0	1	2	3
4.	Stopping yourself from thinking about the past	0	1	2	3
5.	Getting angry about something that wasn't very important	0	1	2	3
6.	Sadness	0	1	2	3
7.	Flashbacks .(sudden memories or images of upsetting things)	0	1	2	3
8.	Feeling you were outside of your body	0	1	2	3
9.	Lower back pain	0	1	2	3
10.	Sudden disturbing memories when you were not expecting them	0	1	2	3
11.	Wanting to cry	0	1	2	3
12.	Not feeling happy	0	1	2	3
13.	Becoming angry for little or no reason	0	1	2	3
14.	Feeling depressed	0	1	2	3
15.	Thoughts or fantasies about hurting someone	0	1	2	3
16.	Your mind going blank	0	1	2	3
17.	Fainting	0	1	2	3
18.	Periods of trembling or shaking	0	1	2	3
19.	Pushing painful memories out of your mind	0	1	2	3
20.	Threatening or attempting suicide	0	1	2	3
21.	Feeling like you were watching yourself from far away	0	1	2	3
22.	Feeling tense or "on edge"	0	1	2	3
23.	Getting into trouble because of sex	0	1	2	3
24.	Not feeling like your real self	0	1	2	3

Please Turn Over

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In the last 6 months, how often have you experienced:

		0 Never	1	2	3 Often
	Continue				
25.	Wishing you were dead	0	1	2	3
26.	Worrying about things	0	1	2	3
27.	Being easily annoyed by other people	0	1	2	3
28.	Starting arguments or picking fights to get your anger out	0	1	2	3
29.	Getting angry when you didn't want to	0	1	2	3
30.	Not being able to feel your emotions	0	1	2	3
31.	Using drugs other than marijuana	0	1	2	3
32.	Feeling Jumpy	0	1	2	3
33.	Absent-mindedness	0	1	2	3
34.	Feeling paralysed for minutes at a time	0	1	2	3
35.	Yelling or telling people off when you felt you shouldn't have	0	1	2	3
36	Intentionally hurting yourself (for example, by scratching, cutting, or burning) even though you weren't trying to commit suicide	0	1	2	3
37	Aches and pains	0	1	2	3
38.	High anxiety	0	1	2	3
39.	Wishing you had more money	0	1	2	3
40.	Nervousness	0	1	2	3
41.	Feeling tired	0	1	2	3
42.	Feeling mad or angry inside	0	1	2	3
43.	Getting into trouble because of your drinking	0	1	2	3
44.	Staying away from certain people or places because they reminded you of something	0	1	2	3
45.	One side of your body going numb	0	1	2	3
46.	Suddenly remembering something upsetting from your past	0	1	2	3
47.	Wanting to hit someone or something	0	1	2	3
48.	Feeling hopeless	0	1	2	3
49.	Hearing someone talk to you who wasn't really there	0	1	2	3
50.	Suddenly being reminded of something bad	0	1	2	3
51.	Trying to block out certain memories	0	1	2	3
52.	Violent dreams	0	1	2	3

Please Turn Over

	In the last 6 months, how often have you experienced:	0 Never	1	2	3 Often
	Continue				
53.	Just for a moment, seeing or hearing something upsetting that happened earlier in your life	0	1	2	3
54.	Frightening or upsetting thoughts popping into your mind	0	1	2	3
55.	Losing your sense of taste	0	1	2	3
56.	Worrying that someone is trying to steal your ideas	0	1	2	3
57.	Not letting yourself feel bad about the past	0	1	2	3
58.	Feeling like things weren't real	0	1	2	3
59.	Feeling like you were in a dream	0	1	2	3
60.	Not eating or sleeping for 2 or more days	0	1	2	3
61.	Trying not to have any feelings about something that once hurt you	0	1	2	3
62.	Daydreaming	0	1	2	3
63.	Trying not to think or talk about things in your life that were painful	0	1	2	3
64.	Feeling like life wasn't living	0	1	2	3
65.	Being startled or frightened by sudden noises	0	1	2	3
66.	Seeing people from the spirit world	0	1	2	3
67.	Trouble controlling your temper	0	1	2	3
68.	Wanting to set fire to a public building	0	1	2	3
69.	Feeling afraid you might die or be injured	0	1	2	3
70	Feeling so depressed that you avoid people	0	1	2	3
71	Thinking that someone was reading your mind	0	1	2	3
72.	Feeling worthless	0	1	2	3

Appendix "D"

Alcohol Use Disorders Identification Test

Alcohol Use Disorders Identification Test
(AUDIT)

The following questions are about the past year
Please tick or circle the box.

1. How often do you have a drink containing alcohol?
   - ☐ Never 0
   - ☐ Monthly or less 1
   - ☐ 2 to 4 times a month 2
   - ☐ 2 to 3 times a week 3
   - ☐ 4 or more times a week 4

2. How many drinks containing alcohol do you have on a typical day when you are drinking?
   - ☐ None 0
   - ☐ 1 or 2 1
   - ☐ 3 or 4 2
   - ☐ 5 or 6 3
   - ☐ 7 or 8 4
   - ☐ 10 or more 5

3. How often do you have six or more drinks on one occasion?
   - ☐ None 0
   - ☐ Less than monthly 1
   - ☐ Monthly 2
   - ☐ Weekly 3
   - ☐ Daily or almost daily 4

4. How often during the last year have you found that you were unable to stop drinking once you had started?
   - ☐ Never 0
   - ☐ Less than monthly 1
   - ☐ Monthly 2
   - ☐ Weekly 3
   - ☐ Daily or almost daily 4

5. How often during the last year have you failed to do what was normally expected from you because of drinking?
   - ☐ Never 0
   - ☐ Less than monthly 1
   - ☐ Monthly 2
   - ☐ Weekly 3
   - ☐ Daily or almost daily 4

Please Turn Over

6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?
   - ☐ Never 0
   - ☐ Less than monthly 1
   - ☐ Monthly 2
   - ☐ Weekly 3
   - ☐ Daily or almost daily 4

7. How often during the last year have you had a feeling of guilt or remorse after drinking?
   - ☐ Never 0
   - ☐ Less than monthly 1
   - ☐ Monthly 2
   - ☐ Weekly 3
   - ☐ Daily or almost daily 4

8. How often during the last year have you been unable to remember what happened the night before because you had been drinking?
   - ☐ Never 0
   - ☐ Less than monthly 1
   - ☐ Monthly 2
   - ☐ Weekly 3
   - ☐ Daily or almost daily 4

9. Have you or someone else been injured as the result of your drinking?
   - ☐ Never 0
   - ☐ Less than monthly 1
   - ☐ Monthly 2
   - ☐ Weekly 3
   - ☐ Daily or almost daily 4

10. Has a relative, friend, or a doctor or other health worker been concerned about your drinking or suggested you cut down?
    - ☐ Never 0
    - ☐ Less than monthly 1
    - ☐ Monthly 2
    - ☐ Weekly 3
    - ☐ Daily or almost daily 4

Appendix "E"

Demographics

1. Present age:.....

2. Gender: Male Female

3. Tick the level of Education Completed:

Primary	☐
Less than or Equal to 11 Years	☐
Completed 12 Years	☐
Tertiary	☐

4. Tick the type of trauma did you experience?

Sexual Abuse	☐
War Experience	☐
Physical Abuse	☐
Sudden Death of a close family member	☐
Bank Hold-up	☐
Near drowning	☐
Other (Describe in Brief)	☐.....

5. On a scale 1-10, 1 = *little distressing* 10 = *extremely distressing*

Circle how you perceive the trauma that you experienced?

1 2 3 4 5 6 7 8 9 10

6. Tick the duration of the trauma event/s.

Day/Once	☐	
Week	☐	
Months	☐	
One Year	☐	
More than one Year	☐	Please write number of years.....

7. List any prescribed and illicit drugs you are presently taking.

.....
.....

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.....

.....

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Appendix F.

RESEARCH

VOLUNTEERS URGENTLY

NEEDED

AGE: OVER 18 years old

I am a Master of Psychology student at Edith Cowan University doing a study on how experiencing a traumatic event can impact on people's memory. I am looking for individuals to participate in my study who have experienced some form of trauma in the past.

The information obtained from you will be treated in confidence. The results will be pooled for the written report in such a way that identification of individuals is not possible in any way.

Please speak to your counsellor/psychologist to make arrangements with the researcher Santina Tonizzo or Telephone: 9275 8113

LOCAL COMMUNITY NEWSPAPER ADVERTISEMENT

I am a Master of Psychology student at Edith Cowan University, doing a study on how experiencing a traumatic event can impact on people's memory. I am looking for individuals to participate in my study who are over the age of 18 years old and have experienced trauma. The information obtained from you will be treated in confidence. The results will be pooled for the written report in such a way that identification of individuals is not possible in any way.

If you feel you would like to participate please phone 9227 8745.

LOCAL COMMUNITY NEWSPAPER ADVERTISEMENT

I am a Master of Psychology student at Edith Cowan University, doing a study on how experiencing a traumatic event can impact on people's memory. I am looking for individual veterans to participate in my study who have either experience being in World War 1, World War 11, Korean War or Vietnam War.

The information obtained from you will be treated in confidence. The results will be pooled for the written report in such a way that identification of individuals is not possible in any way.

Please contact me at the Returned Services League Anzac House (RSL) if you feel you would like to participate.

Phone 9325 9799.

Appendix "G"

CONSENT FORM

Survey on Reported Symptoms of Trauma and the Effects on Memory

Dear Sir/Madam

This study is being conducted as part of my Master of Psychology at Edith Cowan University. The purpose of the study is to examine the relationship between symptoms of individuals who have experienced trauma and their current thinking. If you agree to take part in the study there are two parts. In the first part you will be asked to participate in an individually administered verbal test which may take between 5-10 minutes. In Part 2, you will be asked to answer a questionnaire using a rating scale that may take a further 10-15 minutes.

Your participation is entirely voluntary. Some of the questions may be sensitive to some individuals and if you wish not to answer any question or to withdraw from the study at any time, you are free to do so. If you are in an ongoing counselling program, this study is separate and if you decide not to take part, there will be no effects on your counselling.

The information obtained from you will be treated in confidence. The results will be pooled for the written report in such a way that identification of individuals is not possible in any way.

If you have any questions or concerns you may contact me at the university on 9400 5551, or my University supervisor Associate Professor Ed Helmes on 9400 5543 regarding this research.

Thank-you for your participation, it is greatly appreciated.

Yours sincerely,

S. Tonizzo

- I, (Participant).....have read the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this activity, realising I may withdraw at any time.

Signature of Participant Date

.....

APPENDIX "H"

A Summary Table of the Means, Standard Deviation Scores of the Variables for Males and Females and the Univariate Results.

	Males (*N* = 59)		Females (*N* = 75)		Univariate *F*(1,132)
	M	*SD*	*M*	*SD*	
DEMOGRAPHICS					
AGE	50.61	20.00	46.79	16.56	1.46
WAIS-R Vocabulary [a]	11.80	2.94	12.52	2.76	0.15
Education					4.88
AUDIT [b]	15.66	12.98	6.89	9.00	21.26*
Drugs					2.38
TRAUMA SEVERITY					
Number of Traumas	1.92	1.09	2.33	1.60	2.96
Distress	15.88	9.26	18.53	11.37	2.10
Duration	5.02	1.61	5.24	1.37	.74
ADAPTED TSI [c]					
Intrusive Experience	63.10	13.25	61.26	12.02	0.75
Defensive Avoidance	58.91	10.76	60.02	10.78	0.35
Anxious Arousal	58.54	11.05	60.01	10.07	0.65
Depression	58.44	10.08	58.53	10.55	0.00
Dissociation	62.06	12.89	63.37	12.10	0.37
Anger/Irritability	56.88	11.67	57.13	9.83	0.02
LEARNING & MEMORY TRIALS					
RAVLT [d] Trial 1-5 Total Score	52.19	9.05	58.32	9.69	14.02*
RAVLT [d] Trial 1 (List A)	50.99	9.57	57.44	11.59	11.92*
RAVLT [d] Trial 7 (List A)[4]	48.54	9.32	56.16	8.91	23.18*
RAVLT [d] Trial 8 [5]	49.37	11.18	55.47	9.76	11.32*

a. Possible score range from 0 - 20.
b. Possible score range from 0 - 41.
c. Possible score range from 40 - 95
d. Possible score range from 24 - 76

Significant level * p < .002 Bonferroni Adjustment

[4] Retroactive Interference
[5] Delayed Recall