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Attention, Learning, and Memory in Posttraumatic Stress Disorder

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Abstract

This study compared attention and declarative memory in a sample of combat veterans with post-traumatic stress disorder (PTSD, *n* = 24) previously reported to have reduced concentrations of the hippocampal neuronal marker *N*-acetyl aspartate (NAA), but similar hippocampal volume compared to veteran normal comparison participants (*n* = 23). Healthy, well-educated males with combat-related PTSD without current depression or recent alcohol/drug abuse did not perform differently on tests of attention, learning, and memory compared to normal comparison participants. Further, hippocampal volume, NAA, or NAA/Creatine ratios did not significantly correlate with any of the cognitive measures when adjustments for multiple comparisons were made. In this study, reduced hippocampal NAA did not appear to be associated with impaired declarative memory.

Keywords

posttraumatic stress disorder; memory; hippocampus; alcoholism; *N*-acetyl aspartate

Patients with posttraumatic stress disorder (PTSD) often describe difficulties with concentration, attention, and memory. Poorer performance on tests of attention, declarative memory, and other cognitive domains attributable to PTSD status have been found in many but not all studies (e.g. Crowell, Kieffer, Siders, & Vanderploeg, 2002). Decreased performance on neurocognitive tasks may be of particular relevance to PTSD because multiple studies have documented decreases in hippocampal volume (Bremner et al., 1997; Bremner, Randall, Scott, Bronen, et al., 1995; Gilbertson et al., 2002; Gurvits et al., 1996; Stein, Koverola, Hanna, Torchia, & McClarty, 1997; Villarreal et al., 2002) and decreased reduced concentrations of the neuronal marker *N*-acetyl aspartate (NAA; Freeman, Cardwell, Karson, & Komoroski, 1998; Schuff et al., 2001). Hippocampal atrophy in PTSD was associated with decreased function in explicit memory in a sample of combat veterans (Bremner, Randall, Scott, Bronen, et al., 1995), though not in women with history of childhood sexual assault (Stein et al., 1997).

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We reported that NAA is reduced in the hippocampus of PTSD patients in the absence of hippocampal volume loss (Schuff et al., 2001). NAA occurs at high concentrations in neurons and is virtually nondetectable in other tissues (Birken & Oldendorf, 1989). Hippocampal NAA levels are presumed to reflect neuronal number or metabolism, in contrast to hippocampal volume, which reflects all cell types including neurons and glia. Reduced hippocampal NAA in our study was consistent with findings implicating hippocampal damage in PTSD. Further, reduced NAA in the setting of normal hippocampal volume suggests that NAA may be a more sensitive measure of the integrity of the hippocampus.

Comorbid alcohol and substance abuse affect performance on neurocognitive testing (Goldman, Brown, Christiansen, & Smith, 1991), are associated with lower hippocampal volume (Laakso et al., 2000), and may therefore confound studies of these functional and structural domains in PTSD. Our previous study utilized the most stringent criteria for excluding comorbid conditions of any study examining hippocampal volume in PTSD. Specifically, we excluded participants with a history of alcohol or substance abuse/dependence for the past 5 years and participants with history of major depression within the past 3 months.

This study compares attention, learning, and memory performance in the sample of Vietnam combat veterans with and without posttraumatic stress disorder reported in the Schuff et al. (2001) study. Given our finding of reduced hippocampal NAA in PTSD, our original hypothesis was that PTSD participants, even after controlling for comorbid alcoholism, substance abuse, and depression, would perform worse on tests of attention and declarative memory when contrasted with normal comparison participants. Further, given that NAA is a sensitive measure of hippocampal neurons that are crucial for performance on tasks of memory, we hypothesized that performance on tests of declarative memory would be directly related to hippocampal NAA concentration.

Method

Participants

Medically healthy male Vietnam combat veterans (PTSD participants $N = 24$, mean age = 51.0 years, $SD = 2.5$ years; normal comparison participants $N = 23$, mean age = 52.4 years, $SD = 3.1$ years) were recruited from the San Francisco Veterans Affairs Medical Center and from the community. After a complete description of the study to the participants, written consent was obtained. The Structured Clinical Interview for DSM-IV Diagnosis (SCID; First, Spitzer, Williams, & Gibbon, 1996) was used to rule out both current psychiatric disorders other than PTSD and a history of current major depression in the past 3 months or alcohol or substance abuse during the previous 5 years. PTSD diagnosis and severity was assessed with the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995). Normal comparison participants with a lifetime history of PTSD were excluded. All participants with history of head trauma, neurologic disorder, or systemic illness affecting brain function were excluded. All participants taking benzodiazepines or antipsychotic medication in the past 6 weeks were also excluded. A total of 96 participants consented and were evaluated. Forty-nine were excluded because of the above inclusion and exclusion criteria.

The mean education level was 15.1 years ($SD = 2.2$ years) for the PTSD participants and 16.4 years ($SD = 2.4$ years) for normal comparison participants. Among the PTSD participants, 14 had past history of major depression and 14 had past history of substance or alcohol abuse/dependence. Six of the PTSD participants were on antidepressant medications. Among the comparison participants, 4 had past history of major depression and 9 had past history of substance or alcohol abuse/dependence. None of the normal comparison subjects were on antidepressant medication. The mean CAPS score of the participants with PTSD was 64.7 ($SD = 11.7$) and the mean CAPS of the normal comparison group was 6.4 ($SD = 7.2$). Eighteen

of our PTSD participants were Caucasian, 3 were African American, 2 were Hispanic, and 1 was Native American. Eighteen of our normal comparison participants were Caucasian, 2 were African American, 2 were Asian American, and 1 was Hispanic. Eleven of our PTSD participants were married, 8 were separated or divorced, and 5 were single. Thirteen of our normal comparison participants were married, 5 were separated or divorced, and 5 were single. The mean score on the Global Assessment of Functioning Scale (Endicott, Spitzer, Fleiss, & Cohen, 1976) was 57 (SD = 4.5) for the PTSD participants and 78 (SD = 6.5) for normal comparison participants, $t(2, 45) = -12.8$, $p < .001$.

Measures

The California Verbal Learning Test (CVLT; Delis, Freeland, Kramer, & Kaplan, 1988) was used to assess a variety of verbal learning and recall functions including short-term and delayed recall, recognition performance, and vulnerability to interference effects. The primary measures analyzed for this study included immediate recall over five trials, short delay (3 min) and long delay (20 min) recall, intrusions, and recognition discriminability.

Subtests from the Wechsler Memory Scale-III (Wechsler, 1997) were used to measure auditory and visual attention and working memory (Digit Span and Spatial Span), and visual memory (Faces I and II—Family Pictures I and II subtests). The Benton Visual Form Discrimination (Benton, 1992) was used to assess immediate visual memory and the capacity for complex visual form discrimination. The BVFD is a widely used measure of learning and memory involving designs displayed briefly and then identified from memory by the participant. Administration Form F assesses the capacity for complex visual form discrimination, and Form G assesses immediate visual memory. The numbers of correct choices using the standard criteria outlined in the manual determined the performance score.

Data from the subscales of measures listed above are presented according to the domain they quantify: (1) Attention and working memory: WMS-III—Digit span and Spatial span; BVFD—Form F (matching); (2) Learning/immediate recall: CVLT—List A, Trial 1, Trial 5, total recall, List B; WMS-III—Faces I, Family pictures-I; BVFD-Form G (memory); and (3) Delayed recall and recognition: CVLT—short delay free recall, long delay free recall, intrusions, discriminability; WMS-III—Faces II, Family pictures II.

Magnetic Resonance Imaging (MRI) and 1H Magnetic Resonance Spectroscopic Imaging (MRSI) acquisition and processing were described in detail elsewhere (Schuff et al., 2001). In brief, the participants were scanned on a 1.5-T VISIONTM Magnetic Resonance (MR) system using a double spin echo sequence (DSE) with Time Repetition (TR)/Time Echo (TE) 1/TE 2 = 2500/20/80-ms timing, 3-mm slice resolution, and a volumetric magnetization-prepared rapid gradient echo sequence with TR/TE/Time of Inversion (TI) = 10/4/300-ms timing, 15°-flip angle, 10×1.0 mm^2 inplane resolution, and 1.4-mm thick coronal partitions for structural MRI. A Point Resolved Spectroscopy (PRESS) 1H MRSI (Bottomley, 1987) sequence with TR/TE = 1800/135-ms timing and 1.1-ml sized MRSI voxels was used to acquire water suppressed 1H MR spectra simultaneously from both hippocampi.

Procedure

Neuropsychological testing was administered the morning before neuroimaging or on a different day. A masters level psychology research associate (ML) obtained the neuropsychological measures under the supervision of a clinical neuropsychologist (JR). The tests were administered in the same order for all subjects. Testing took approximately 2 hr including a mid-session 20-min break. Participants were instructed to abstain from using alcoholic beverages and were breathalyzed before testing. Participants also had urinalysis for

drug toxicology on the day of neuropsychological assessment. Data from participants with detectable alcohol levels or positive toxicology screens were excluded from the analysis.

Statistical Analysis

Descriptive data provide means, standard deviations, and effect sizes of the measures of explicit memory and attention. Group differences in cognitive performance were tested using two-tailed *t* tests. With our sample sizes we had 0.80 power to detect an effect size of 0.70. The relationship between explicit memory and hippocampal NAA was examined with Pearson correlation coefficients (two-tailed). We had 0.80 power to detect a correlation of $r = .40$.

Results

Performance on measures of attention, learning, and memory are presented in Table 1. There were no sig-nificant differences in performance between PTSD and comparison participants on any of the cognitive measures. Most effect sizes had an absolute value of less than 0.3. The only exceptions showed that PTSD participants had less recall on Trial 1 for the CVLT in PTSD (effect size = 0.34) and better visual recall on Faces II (effect size = 0.44).

There were no significant correlations between measures of cognitive performance and right or left hippocampal NAA. To exclude the possibility that systematic errors in estimating NAA concentration, such as correction for tissue atrophy or T1 and T2 relaxation, obscured a correlation with the neurospychological data, we also determined NAA/Cr ratios, which reduce dependencies of atrophy and relaxations. After adjusting for multiple comparisons there were no significant correlations between the NAA/Cr ratios and the neurospychological measures. Before adjustment complex visual form discrimination (Benton F) was positively correlated with left hippocampal volume, $r = .34$, $p < .05$, and there was a trend for a positive association of immediate visual recall (Benton G) with right hippocampal volume, $r = .30$, $p = .065$. However, these two correlations did not remain significant after adjusting for multiple comparisons.

Discussion

The major results of the study show that in this sample of well-educated combat veterans with chronic PTSD and non-PTSD participants, carefully chosen to minimize the impact of comorbid depression, substance, and alcohol abuse, there were no significant group differences in standard measures of attention or memory for word-lists, realistic scenes, abstract designs, or unfamiliar faces. In addition there was no correlation of attention or memory with hippocampal volume or NAA.

Our sample differs from previously published studies of PTSD and cognition on four variables. First, mean years of education level in our sample (15.1 years for PTSD) was the highest of any of the above cited studies in which the mean years of education ranged from 12.7 to 14.2. Second, the mean age of our participants (51–52 years) was older than the participants in the studies referenced above; however, this is not likely to bias our negative finding. Third, our participants, as previously reported did not differ from comparison participants with respect to hippocampal volume (Schuff et al., 2001). Finally, our sample met the most stringent entry criteria, relative to the studies listed above, for excluding participants who had a major depressive episode in the past 3 months, or a diagnosis of alcohol or substance abuse in the past 5 years.

One implication of these results is that prior reports of impaired attention and declarative memory in PTSD, utilizing neurocognitive measures similar to those used in this study, may have been confounded by comorbid diagnoses of substance abuse and/or depression. An

examination of the positive studies demonstrating significant differences in attention, learning, and memory show a range of effect sizes for PTSD status on neurocognitive functioning from 0.7 to 2.0 (Bremner et al., 1993; Bremner, Randall, Scott, Capelli, et al., 1995; Gil, Calev, Greenberg, Kugelmass, & Lerer, 1990; Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001; Jenkins, Langlais, Delis, & Cohen, 1998; Moradi, Doost, Taghavi, Yule, & Dalgleish, 1999; Roca & Freeman, 2001; Sachinvala et al., 2000; Uddo, Vasterling, Brailey, & Sutker, 1993; Vasterling et al., 2002; Vasterling, Brailey, Constans, & Sutker, 1998; Yehuda et al., 1995). Our study was not sufficiently powered to detect small effect sizes for cognitive performance; however, we did have adequate power to find effects similar in magnitude to most of those reported in the literature. The exclusion criteria regarding alcohol abuse in the prior literature with both positive and negative findings ranges from having no exclusions (but often with matching PTSD and comparison participants on alcohol abuse) to 1 year without alcohol abuse or dependence. Hence, recent alcohol abuse does not appear to explain the variability in effect sizes across these studies. Although the effects of alcohol abuse on the brain are in part reversible within the first 3 months of abstinence (Pfefferbaum et al., 1995), the study by Gilbertson et al. (2001) that excluded participants with a history of alcohol abuse in the past year, demonstrated that the effects of alcohol on cognition are still measurable for at least a year after abstinence.

The presence of current comorbid major depression is a problem in most studies of PTSD and neurocognitive functioning. The exclusion of participants with current major depression in this sample potentially could have biased the sample to having milder PTSD symptoms. However, the mean CAPS score of our PTSD participants (64.7) is above 60, which has been defined as the threshold for severe PTSD symptomatology by Weathers, Keane, and Davidson (2001). Although some investigators have suggested that depression is inextricably linked to PTSD, this study as well as recent neurobiological data (Yehuda, Halligan, Grossman, Golier, & Wong, 2002), suggest that major depression must still be carefully considered as a possible confound.

A careful review of the literature demonstrated that the three studies of cognition conducted to date with the largest sample sizes have yielded negative findings (Barrett, Green, Morris, Giles, & Croft, 1996; Crowell et al., 2002; Zalewski, Thompson, & Gottesman, 1994). The study by Barrett is particularly noteworthy because they found that veterans with PTSD alone ($N = 236$) did not show lower scores on measures of cognitive functioning compared to normal comparison participants ($N = 1,835$), whereas veterans with both PTSD and a concurrent diagnosis of depression, another anxiety disorder, or substance abuse ($N = 128$) performed significantly less well. These results suggest that a diagnosis of PTSD alone is not strongly associated with cognitive impairment as assessed by these methods.

Similar to a study of women victimized by childhood sexual abuse (Stein et al., 1997), we found no relationship between hippocampal volume and measures of declarative memory function. Stein and colleagues suggested that the relatively young age of their women with childhood sexual abuse (mean age = 32) may have accounted for the lack of relationship between hippocampal volume and declarative memory. We found similar results in a sample that is approximately 20 years older with normal hippocampal volume. It remains possible that the relationship between hippocampal measures and cognitive performance are not discernible until advanced ages, or in subjects with dementia. For example, the study that showed the largest reduction in hippocampal volume, 26% (Gurvits et al., 1996), did not show an association of hippocampal size with verbal declarative memory. Finally, it is possible that measures of gross brain structure and neurochemistry (e.g., NAA) are too insensitive to relate to subtle differences in neurocognition that may exist in samples of participants who perform within the normal range of cognitive function.

Although the association between hippocampal volume and performance on the Benton Visual Form Discrimination did not remain significant after correcting for multiple comparisons, the correlation is similar to a finding by Gurvits et al. (1996) for a different test format using BVFD stimuli. In their study of combat PTSD ($N = 7$) and combat normal comparison participants ($N = 7$) a relationship was observed between Benton 15-s delayed recall performance and total hippocampal volume. Similarly, in our study, performance on the Benton visual form discrimination was correlated with hippocampal volume. These findings are consistent with preclinical data that demonstrate an important role of the hippocampus in regulating visuospatial processing (for review see Sewards & Sewards, 2002).

Finally, it is important to note that performance measures in both of our groups are within the normal range. The similar scores on measures of attention in the two groups are noteworthy, because decreased attention may account for differences in performance on many neuropsychological measures (Gilbertson et al., 2001). It is also possible that our measures (e.g., subtests of the WMS-III and CVLT) are not sufficiently challenging for higher-functioning, ambulatory PTSD patients, and thus may fail to detect subtle memory deficits in this subgroup. For example, Bremner and colleagues detected strong group differences in PTSD versus comparison participants in logical memory performance, which involves a relatively challenging test of paragraph recall (Bremner, Randall, Scott, Bronen, et al., 1995). Future studies might assess a broader range of cognitive functions, such as narrative recall, other measures of concentration and working memory, planning, and organizational skills that may be more sensitive to differences in neurocognitive performance in higher-functioning individuals.

Summary and Conclusions

In a sample of PTSD participants who do not differ from normal comparison participants with respect to hippocampal volume, we were unable to discern any differences in performance on tasks of attention, learning, and memory. Further, to our knowledge, this is the first study examining the relationship of hippocampal NAA with cognition in PTSD. Despite significantly reduced hippocampal NAA in PTSD, as previously reported by our group (Schuff et al., 2001), we were unable to find neurocognitive correlates of reduced NAA. Future studies will be needed to further examine neurocognitive correlates to brain volume and chemistry using both more sensitive neuropsychological measures as well as spectroscopy measures in other regions of the brain besides the hippocampus that are involved in attention and declarative memory.

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Table 1

Neuropsychological Performance Measures

Variable	*M* (*SD*)		Effect size (*d*)	*t*(2, 45)
	PTSD (*n* = 24)	Controls (*n* = 23)		
Attention and working memory				
WMS-III: Digit span	17.7 (3.9)	17.3 (4.5)	0.10	<1
WMS-III: Spatial span	15.9 (2.6)	15.5 (2.4)	0.16	<1
BVFD:Form F (matching)	30.6 (1.9)	30.9 (1.6)	−0.17	<1
Learning/immediate recall				
CVLT List A Trial 1 # correct	6.6 (1.6)	7.2 (1.9)	−0.34	−1.2
CVLT List A Trial 5 # correct	12.1 (2.3)	12.1 (2.2)	0.00	<1
CVLT total recall	50.7 (9.2)	51.0 (9.5)	−0.03	<1
CVLT List B interference list	6.1 (1.8)	6.3 (2.1)	−0.10	<1
WMS-III: Faces I	37.6 (4.3)	37.9 (3.6)	−0.08	<1
WMS-III: Family pictures I	43.0 (8.5)	42.7 (10.3)	0.03	<1
BVFD: Form G—memory	28.3 (3.0)	27.9 (2.5)	0.15	<1
Delayed recall and recognition				
CVLT short delay free recall	10.9 (2.5)	10.3 (3.2)	0.21	<1
CVLT long delay free recall	11.2 (2.7)	11.3 (2.9)	−0.04	<1
CVLT intrusions	3.7 (3.1)	4.3 (4.3)	−0.16	<1
CVLT discriminability	95.1 (4.3)	93.9 (6.1)	0.23	<1
WMS-III: Faces II	38.3 (3.1)	36.9 (3.2)	0.44	1.4
WMS-III: Family pictures II	42.7 (8.3)	43.1 (9.0)	−0.05	<1

Note. WMS-III = Wechsler Memory Scale-III; BVFD = Benton Visual Form Discrimination; CVLT = California Verbal Learning Test.