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Neuropsychological function in college students with and without posttraumatic stress disorder

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Abstract

Previous research on the neuropsychology of posttraumatic stress disorder (PTSD) has identified several neurocognitive deficits that co-occur with the disorder. However, it remains unclear whether these deficits are due to trauma exposure, PTSD symptomatology or psychiatric/substance abuse comorbidity. We examined trauma exposure, PTSD symptoms and neuropsychological performance in 235 undergraduate students, i.e. a non-clinical sample. The sample comprised 146 subjects with trauma exposure (38 with current PTSD and 108 without lifetime PTSD) and 89 no-trauma comparison (NC) subjects who were administered tests of attention, working memory, psychomotor speed, word generation and executive functioning. Relationships of neuropsychological functioning to measures of psychiatric symptoms and substance abuse were examined. Current PTSD (PTSD+), trauma-exposed without PTSD (PTSD−) and NC subjects did not differ significantly on the vast majority of neuropsychological tests. There were very few significant associations between neuropsychological performance and clinical variables, and those that were statistically significant were small in magnitude. The striking lack of differences in neuropsychological performance between the three groups suggests that college students with trauma exposure, regardless of the presence of PTSD symptoms, may be cognitively resilient. Neuropsychological impairment may not be an invariant feature of PTSD, but when it is present, it may be associated with poorer functional outcomes.

Keywords: Trauma; Cognition; Neuropsychology; Executive functioning; Undergraduates

1. Introduction

The neuropsychological functioning of persons with posttraumatic stress disorder (PTSD) has become an important area of inquiry. Previous research on neuropsychological functioning in patients with PTSD has identified impairments in attention, learning, memory and executive functioning in combat veterans (Gil et al., 1990; Sutker et al., 1991; Uddo et al., 1993; Bremner et al., 1993, 1995; Beckham et al., 1998; Vasterling et al., 1998, 2002) and women with post-rape PTSD (Yehuda et al., 1995; Jenkins et al., 2000). However, some studies have found few or no impairments in these domains (Gurvits et al., 1993; Stein et al., 1999; Crowell et al., 2002), leading researchers to question whether the cognitive profile of

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PTSD is actually reflective of comorbid major depressive disorder (Barrett et al., 1996) or, perhaps, substance abuse (Sullivan et al., 2000; Stein et al., 2002). Some studies have controlled for one or both of these factors and found impairments in attention and attentional shifting (Beckham et al., 1998; Jenkins et al., 2000; Vasterling et al., 2002) and learning (Bremner et al., 1995).

Another question is whether neuropsychological impairment is related to psychiatric symptomatology or more directly to trauma exposure. One large study, for example, found no significant differences in cognitive performance among Vietnam veterans with PTSD, generalized anxiety disorder and no psychiatric diagnosis (Zalewski et al., 1994). Although the literature on neuroendocrine functioning in PTSD has produced some conflicting findings, stress-related glucocorticoid alterations could underlie changes in cognitive functioning, either as a cause or as an effect of hippocampal functioning (Sapolsky, 2002). Additional stress-related findings, such as increased corticotropin-releasing factor levels, decreased catecholamine levels in the CNS, and increased variability in cortisol release, are further evidence for the possibility that the cognitive changes of PTSD may be due to the effects of trauma itself (Yehuda, 2001).

Yet another potential explanation for impaired cognition in PTSD is lower premorbid cognitive functioning. Retrospective case-control studies suggest that lower IQ is a risk factor for development of PTSD (McNally and Shin, 1995; Macklin et al., 1998; Gurvits et al., 2000). It may be that individuals with lower IQs cope less effectively with trauma on a cognitive level, or that those with higher IQs cope more effectively. PTSD also is associated with smaller hippocampal size (Hull, 2002), although recent research suggests that smaller hippocampi may be a premorbid risk factor for PTSD rather than a result of trauma exposure (Gilbertson et al., 2002). The smaller hippocampi that may confer vulnerability to PTSD also are associated with lower IQ (Andreasen et al., 1993). Thus, it is unclear whether lower IQ and smaller hippocampi are independent or related risk factors for PTSD.

The majority of the existing research on cognition and the neurobiology of PTSD focuses on learning, memory and hippocampal functioning. Abnormalities in executive functioning, too, would be predicted on the basis of the frontal-subcortical pathways associated with anxiety and stress (Arnsten, 1998; Kaufman et al., 2000). In a recent study of non-substance-abusing women who had experienced intimate partner violence (IPV), we found very few differences between IPV subjects and normal controls on a comprehensive neuropsychological battery (Stein et al., 2002). IPV subjects performed worse than controls on selected measures of attention, working memory and executive functioning, possibly reflecting frontal-subcortical impairment, but IPV subjects with PTSD did not perform uniformly worse than IPV subjects without PTSD, suggesting that trauma exposure may be more explanatory of cognitive deficits than is PTSD symptomatology. Due to the dearth of learning and memory deficits seen in our previous work (Stein et al., 2002), we elected to focus on measures of attention, working memory and executive functioning in the present study.

In summary, if cognition is impaired in PTSD, the source of impairment is unclear. Deficits could be due to the trauma exposure itself, premorbid neurocognitive dysfunction, current PTSD symptoms, depressive symptoms, substance abuse or a combination of these factors. Ideally, prospective longitudinal studies would address these questions, but as a more immediately feasible alternative, we chose to cross-sectionally study neuropsychological function in three subject groups: those with a history of experiencing serious psychological trauma who have not developed PTSD, those with a history of experiencing serious psychological trauma who have developed PTSD, and those who have not experienced serious psychological trauma. We undertook the current investigation with these questions in mind and chose to study a non-treatment-seeking sample, i.e. undergraduate students, to reduce potential confounds. We hypothesized that on a brief neuropsychological battery emphasizing executive functioning, non-traumatized comparison students (NCs) would perform better than students with PTSD (PTSD+), and that the performances of traumatized students

Table 1
Sociodemographic and clinical characteristics of NC, PTSD− and PTSD+ students

Measure	NC		PTSD−		PTSD+		*F*	*P*	Tukey/Dunnett C*
	N	*M* (S.D.)	*N*	*M* (S.D.)	*N*	M (S.D.)			
Age, years	87	18.8 (1.5)	105	19.2 (2.2)	38	19.0 (2.7)	0.9	0.396	
Education, years[a]	87	13.3 (0.7)	103	13.5 (0.9)	38	13.3 (0.7)	1.8	0.169	
Number of lifetime traumata[a]	87	0.0 (0.0)	105	3.2 (1.7)	38	4.2 (2.0)	168.2	<0.001	1<2<3
Childhood sexual abuse[a]	85	0.0 (0.0)	103	0.3 (0.5)	38	0.4 (0.5)	22.7	<0.001	1<2,3
Childhood Trauma Questionnaire									
Emotional abuse[a]	87	7.5 (3.1)	105	8.9 (3.5)	38	14.1 (5.5)	42.0	<0.001	1<2<3
Physical abuse[a]	87	6.2 (1.9)	105	7.0 (2.7)	38	9.3 (5.6)	13.1	<0.001	1,2<3
Sexual abuse[a]	87	5.5 (1.5)	105	7.4 (4.4)	38	8.3 (5.0)	10.1	<0.001	1<2,3
Emotional neglect	87	8.9 (4.5)	105	9.4 (4.4)	38	12.8 (4.7)	10.4	<0.001	1,2<3
Physical neglect[a]	87	6.1 (2.1)	105	7.2 (3.2)	38	8.1 (2.9)	7.2	0.001	1<2,3
Total score[a]	87	34.2 (9.2)	105	39.9 (12.8)	38	52.6 (16.0)	30.1	<0.001	1<2<3
Posttraumatic Stress Diagnostic Scale									
Re-experiencing[a]	87	0.0 (0.0)	105	1.7 (2.5)	38	6.0 (3.5)	97.3	<0.001	1<2<3
Avoidance[a]	87	0.0 (0.6)	105	1.3 (2.0)	38	3.9 (2.5)	70.8	<0.001	1<2<3
Numbing[a]	87	0.1 (0.5)	105	1.1 (1.9)	38	4.2 (2.8)	74.9	<0.001	1<2<3
Hyperarousal[a]	87	0.1 (0.8)	105	1.7 (2.4)	38	6.1 (3.8)	91.2	<0.001	1<2<3
Total score[a]	87	0.2 (1.3)	105	5.9 (7.6)	38	20.2 (10.6)	116.0	<0.001	1<2<3
State-Trait Anxiety (Trait)	87	39.8 (10.9)	105	39.1 (10.8)	38	48.8 (11.0)	11.8	<0.001	1,2<3
Beck Depression Inventory	87	6.8 (6.6)	103	7.0 (6.8)	38	12.9 (8.4)	11.6	<0.001	1,2<3
Alcohol Use Disorders Inventory	87	5.0 (4.6)	105	5.8 (5.6)	38	5.7 (6.0)	0.5	0.581	
Drug Abuse Screening Test[a]	87	0.9 (1.2)	105	1.2 (1.6)	38	1.8 (1.8)	5.0	0.007	1<3

*In Tukey/Dunnett C column, 1=NC, 2=PTSD− and 3=PTSD+; groups separated by < differ significantly ($P<0.05$). The Tukey test was used when the groups demonstrated homogeneity of variance; the Dunnett C test was used when the groups did not demonstrate homogeneity of variance.

[a]Groups did not demonstrate homogeneity of variance.

without PTSD (PTSD−) would be intermediate between the other two groups.

2. Methods

2.1. Participants

The sample comprised 230 undergraduate students (62 men and 168 women) at San Diego State University who participated in a screening questionnaire at the beginning of the semester and were subsequently recruited to participate in neuropsychological testing. All participants had either learned English as their first language or had learned English by age 10, ensuring their ability to participate in English-language neuropsychological testing.

Individuals were excluded from the study if any of the following applied: history of head injury associated with loss of consciousness of more than 15-min duration or overnight hospitalization; current use of psychotropic medication.

The demographic and baseline clinical characteristics of the three groups are presented in Table 1.

2.2. Procedure

Undergraduate students ($n=1690$) completed the screening questionnaires, along with several other questionnaires included by other researchers, as part of their introduction to a psychology course. A subset of subjects who indicated that they were willing to participate in additional studies were contacted and participated in the current study ($n=245$). Subjects were administered the neuropsychological measures by research assistants who went through extensive training provided by the first author, and all neuropsychological scores were double-checked by the first author. All subjects

gave written informed consent to participate in this study and earned class credit for participating.

Data from 15 subjects were not included in the current analyses: nine subjects had experienced head injuries resulting in >15-min losses of consciousness; five subjects had a symptom course consistent with Acute Stress Disorder (i.e. they had posttraumatic stress symptoms lasting <4 weeks), but not PTSD; one subject's diagnostic data were missing. Thus, the final sample size was 230.

Trauma exposure and PTSD status were ascertained using the Posttraumatic Stress Diagnostic Scale (PDS; Foa, 1995), described below. Participants were classified into the following three groups: 87 NCs, who had never experienced a DSM-IV PTSD Criterion A event; 105 PTSD− students, who had experienced one or more qualifying traumata but had no lifetime history of PTSD; and 38 PTSD+ students, who met full or partial DSM-IV criteria for PTSD. Partial PTSD was defined as meeting DSM-IV criteria with the exception that they have only two (rather than three) Criterion C (Avoidance/Numbing) symptoms and/or one (rather than two) Criterion D (Hyperarousal) symptoms (Stein et al., 1997).

2.3. Measures

2.3.1. Screening questionnaire measures

The Posttraumatic Stress Diagnostic Scale (PDS) is a 49-item self-report questionnaire designed to permit the diagnosis of PTSD and the quantification of symptom severity (Foa, 1995). The first part of the scale asks about exposure to 11 different types of very stressful or traumatic events. Subsequent parts of the scale ask about PTSD symptoms and resultant functioning.

Subjects also completed a standardized, retrospective measure of childhood maltreatment, the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994, 1997); a widely used measure of trait anxiety, the State-Trait Anxiety Inventory, Trait version (STAIT; Spielberger, 1983); and a widely used measure of depressive symptoms, the Beck Depression Inventory (BDI; Beck et al., 1988). The Alcohol Use Disorders Identification Test (AUDIT; Bohn et al., 1995) and the Drug Abuse Screening Test (DAST; Skinner, 1982) were used to screen for alcohol and drug abuse, respectively.

2.3.2. Neuropsychological tests

Neuropsychological tests were administered to all subjects by trained raters. Scoring followed standardized procedures. Word generation was assessed with the Controlled Oral Word Association Test, in which examinees have 1 min to name as many words as they can that begin with a certain letter (F, A and S) and 1 min to name as many animals as they can (Spreen and Strauss, 1998). Attention and working memory were assessed with the WAIS-III Digit Span and Letter–Number Sequencing subtests (both auditory-verbal attention and working memory tests; Wechsler, 1997) and the Digit Vigilance Test, which measures sustained visual attention (Lewis and Rennick, 1979). Psychomotor speed was assessed with Part A of the Trail-Making Test, in which examinees draw a line connecting numbered circles as quickly as possible (Reitan and Wolfson, 1993). Executive functioning was assessed with Part B of the Trail-Making Test, which requires shifting cognitive sets between numbers and letters, and the computerized version of the Wisconsin Card Sorting Test, 64-item version (WCST-64; Kongs et al., 2000). The WCST-64 measures rule learning/abstraction in a context that requires maintaining and shifting cognitive set in response to verbal feedback from the examiner. Premorbid verbal intellectual functioning was measured with the American National Adult Reading Test, which assesses the ability to pronounce irregularly spelled English words (ANART; Grober and Sliwinski, 1991). Tests specifically assessing test-taking effort were not administered.

2.4. Statistical analyses

Analysis of variance (ANOVA) with post hoc testing using the Tukey test compared means of symptom and demographic variables between groups. In order to limit the number of statistical tests performed on the neuropsychological tests, we conducted multivariate analysis of variance (MANOVA) for all pertinent subtests of a single test. Only when the overall MANOVA was statis-

tically significant did we proceed with ANOVA and post-hoc Tukey testing to localize the effects. The Dunnett C post hoc test was used when groups did not demonstrate homogeneity of variance. Associations of neuropsychological performance and symptom or trauma severity were examined using Pearson's correlation coefficient or, for non-normally distributed variables, Spearman's rho correlation coefficient. All statistical tests were two-tailed, and P values <0.05 were considered statistically significant.

3. Results

3.1. Characteristics of study participants

The demographic and baseline clinical characteristics of the study participants are presented in Table 1. The NC, PTSD− and PTSD+ groups did not differ in age, years of education (see Table 1) or ethnic minority status ($\chi^2=0.1$, d.f.$=2$, $P=0.947$). Although 27% of the participants were men, 30% of the NC and PTSD− groups were men, whereas only 13% of the PTSD+ subjects were men (this difference was not significant; $\chi^2=4.4$, d.f.$=2$, $P=0.111$). As would be expected, compared with NCs, trauma-exposed subjects had higher rates of childhood abuse and higher scores on the measures of PTSD, anxiety and depressive symptoms. PTSD severity scores were lower than would be expected in a treatment-seeking sample. The three groups did not differ on self-reported alcohol use. PTSD+ participants reported significantly higher substance abuse than did the NC subjects, however.

3.2. Group differences in neuropsychological performance

The NC, PTSD− and PTSD+ groups did not exhibit significant differences in premorbid verbal intellectual functioning or in performance on most of the neuropsychological tests (see Table 2). For the entire sample, the mean IQ estimate was 108 (within the average range) and normed scores for all the neuropsychological tests were within the average range (i.e. Digit Span and Letter Number Sequencing scaled scores were both 10; *t*-scores for Digit Vigilance time, Digit Vigilance errors, Trail Making Test, Part A, Letter Fluency, Category Fluency, Trail Making Test, Part B and Wisconsin Card Sorting Test total errors were 52, 45, 51, 45, 49, 50 and 55, respectively).

There were two statistically significant differences between the groups. On the WCST, PTSD− subjects performed significantly worse than NCs on the number of trials needed to complete the first category, which is a general indicator of problem-solving ability. However, PTSD− subjects performed significantly better than NCs on the 'learning to learn' index, which measures the ability to increase efficiency of learning over successive categories.

3.3. Relationships between symptom and abuse severity and neuropsychological functioning

We examined bivariate correlations in the traumatized subjects (PTSD+ and PTSD−) between all 21 neuropsychological test scores and the seven scores on the CTQ, STAIT, BDI, AUDIT, DAST, number of lifetime traumata and PDS. Pearson correlations were used when the data were normally distributed and Spearman Rho correlations were used when skewness and/or kurtosis were >3.0. Of the 147 correlations, only eight were significant—the same number that would be significant at the 0.05 level by chance alone. With that caveat, the significant correlations are presented in Table 3. Six of the eight correlations were in the unexpected direction (i.e. better neuropsychological performance was associated with more trauma and worse anxiety, alcohol use and drug abuse), and two correlations were in the expected direction (number of trials to complete the first category on the WCST was associated with more alcohol use; failure to maintain set on the WCST was associated with more severe depressive symptoms). The few differences between groups and the overall lack of correlation between neuropsychological performance and measures of PTSD symptomatology and substance abuse were contrary to our expectations.

4. Discussion

Most previous studies of neuropsychological functioning in subjects with PTSD have identified

Table 2
Neuropsychological performances of NC, PTSD− and PTSD+ students

Test	NC		PTSD−		PTSD+		F^a	P	Tukey/Dunnett C[b]
	N	M (S.D.)	N	M (S.D.)	N	M (S.D.)			
Premorbid intelligence									
ANART	86	26.8 (7.5)	104	26.8 (5.7)	37	28.7 (6.2)	1.3	0.282	
Attention and working memory									
WAIS-III Digit Span	86		104		37		1.3	0.266	
Forward		10.6 (2.0)		10.8 (1.8)		11.1 (1.8)			
Backward		6.5 (2.0)		6.5 (1.8)		7.3 (2.1)			
Total		17.1 (3.4)		17.3 (3.0)		18.3 (3.4)			
WAIS-III Letter-Number Seq.	86	10.6 (1.9)	104	10.8 (2.1)	37	11.5 (2.5)	2.4	0.093	
Digit Vigilance Test	86		104		37		1.1	0.340	
Total time		328.8 (68.3)		346.6 (68.6)		328.3 (57.4)			
Total errors		6.7 (5.9)		6.0 (5.1)		7.3 (8.7)			
Psychomotor Speed									
Trail Making Test, Part A[c]	83		104		37		0.6	0.665	
Time (s)		21.7 (6.9)		22.9 (6.8)		22.8 (6.8)			
Errors		0.2 (0.5)		0.2 (0.5)		0.1 (0.4)			
Word Generation							0.2	0.955	
FAS	85		104		37				
Total score		38.0 (7.9)		37.4 (9.3)		36.9 (8.0)			
Animals	85		104		37				
Total score		20.8 (4.4)		20.9 (3.9)		20.6 (4.1)			
Executive functioning									
Trail Making Test, Part B[c]	83		104		37		0.4	0.799	
Time (s)		53.2 (14.0)		53.5 (17.5)		53.4 (23.3)			
Errors		0.4 (0.7)		0.5 (1.2)		0.4 (0.8)			
Wisconsin Card Sorting Test	86		101		37		1.9	0.012	
Total correct		51.0 (6.5)		50.3 (6.5)		50.0 (7.5)	0.4	0.661	
Total errors (SS)		106.7 (16.6)		105.3 (12.9)		106.0 (14.3)	0.2	0.822	
Perseverative resp. (SS)		108.0 (14.4)		106.0 (13.8)		103.6 (12.8)	1.4	0.260	
Perseverative errors (SS)		107.4 (14.5)		106.1 (13.8)		104.4 (12.1)	0.7	0.524	
Nonpersev. errors (SS)		105.0 (13.0)		102.4 (12.7)		104.7 (14.9)	1.0	0.367	
Concept. level resp. (SS)		107.5 (13.4)		105.3 (13.0)		106.0 (14.3)	0.7	0.506	
Categories completed		4.0 (1.1)		3.9 (1.1)		3.8 (1.1)	0.7	0.476	
Trials to complete 1st category[d]		13.2 (5.7)		16.6 (10.4)		14.2 (6.0)	4.2	0.017	1<2
Failure to maintain set[d]		0.2 (0.5)		0.3 (0.6)		0.4 (0.7)	1.7	0.191	
Learning to learn		−2.8 (7.8)		0.6 (7.4)		−1.7 (9.3)	4.1	0.019	1<2

[a]Measures for each test included in a single MANOVA (or ANOVA if only one measure for that test); P value shown is for main effect of diagnostic group. ANOVAs and post-hoc testing (Tukey) for individual measures conducted only if $P<0.05$ for MANOVA (Wilks' lambda).

[b]In Tukey column, 1=NC, 2=PTSD− and 3=PTSD+; groups separated by < differ significantly ($P<0.05$). The Tukey test was used when the groups demonstrated homogeneity of variance; the Dunnett C test was used when the groups did not demonstrate homogeneity of variance.

[c]Trail Making Test, Parts A and B were tested in a single MANOVA.

[d]Groups did not demonstrate homogeneity of variance.

All scores are raw scores unless otherwise indicated.

Table 3
Correlations between neuropsychological performance and questionnaire measures in traumatized (PTSD− and PTSD+) students ($N=141$ unless otherwise noted)

	# Lifetime traumata	CTQ total	PDS	STAIT	BDI	AUDIT	DAST
Attention and working memory							
WAIS-III Digit Span							
Forward							0.19*
Backward	0.23**			0.17*		0.23*	
WAIS-III Letter-Number Seq.				0.21**			0.17*
Digit Vigilance Test							
Total time							
Total errors							
Psychomotor Speed							
Trail Making Test, Part A							
Time (s)							
Errors$^{\rho}$							
Word generation							
FAS total score							
Animals total score							
Executive functioning							
Trail Making Test, Part B							
Time (s)							
Errors							
Wisconsin Card Sorting Test							
Total correct							
Total errors (SS)							
Perseverative resp. (SS)							
Perseverative errors (SS)							
Nonpersev. errors (SS)							
Concept. level resp. (SS)							
Categories completed							
Trials to complete 1st Cat. $^{\rho}$						0.18* (138)	
Failure to maintain set$^{\rho}$						0.18* (136)	
Learning to learn							

*$P<0.05$; **$P<0.01$; $^{\rho}$Spearman's Rho correlation used; all others are Pearson correlations; PDS = Posttraumatic Stress Diagnostic Scale; CTQ = Childhood Trauma Questionnaire; STAIT = State-Trait Anxiety Inventory (Trait version); BDI = Beck Depression Inventory; AUDIT = Alcohol Use Disorder Inventory Total score; DAST = Drug Abuse Screening Test.

specific cognitive deficits, but ours did not. In fact, there were very few differences between groups on neuropsychological performance on a neuropsychological battery emphasizing executive functioning. One significant difference, between the NC and PTSD− groups, favored the NCs, whereas the other significant difference favored the PTSD− group. Moreover, these few group differences were small in magnitude and, in our opinion, were not likely to result in clinical significance.

These results suggest that college students with trauma exposure, regardless of PTSD status, do not exhibit marked neuropsychological dysfunction. Furthermore, we found very few significant associations between the clinical variables and neuropsychological scores within the trauma-exposed subjects, suggesting that the clinical variables are not responsible for the lack of group differences in neuropsychological performance.

Our investigation addressed many of the confounding factors in earlier studies by including a traumatized, non-PTSD comparison group and by measuring premorbid IQ, as well as comorbid depression, alcohol use and substance abuse. The PTSD+, PTSD− and NC groups did not differ

significantly on premorbid IQ or current alcohol use. The PTSD+ group had significantly more severe depressive symptoms and higher rates of substance abuse, but because they did not perform worse than the other two groups on the neuropsychological tests, we did not control for these differences statistically.

The neuropsychological battery used in the current study emphasized executive functioning, based on the results of our previous work (Stein et al., 2002) finding rather isolated deficits in executive functioning. Importantly, we did not measure memory performance, which has been found to be impaired in PTSD subjects in some previous studies (Sutker et al., 1991; Bremner et al., 1993, 1995; Yehuda et al., 1995). Other recent investigations have found no memory impairments in PTSD subjects, however (Stein et al., 2002; Vasterling et al., 2002). Additionally, executive functioning is a broad construct including several aspects of frontal-subcortical functioning that were not assessed in our battery. It is possible that the PTSD+ group had impairments in other frontal/executive domains (see Koenen et al., 2001). To increase our sample size, we used questionnaires rather than clinical interviews to establish the diagnostic groups in this study, which may have resulted in a higher than expected rate of endorsement of PTSD symptomatology. However, the groups differed in PTSD and associated symptoms in the expected directions. Another limitation of this study is that the results probably do not generalize to non-college populations. It is also possible—even likely—that our sample was, on average, less severe than treatment-seeking individuals in terms of current PTSD symptoms. Thus, it could be that cognitive deficits relate to 'dose' of trauma exposure, response to the trauma (e.g. PTSD severity) or to some combination thereof. As such, we may have sampled a group with lower average exposure to trauma (either in terms of duration or intensity), leading to a lower likelihood of cognitive dysfunction. This hypothesis could be tested in future studies by obtaining more detailed information about trauma exposure, in an attempt to quantify the 'dose'.

A broader implication of these results is that neuropsychological dysfunction is not an invariant feature of PTSD. When present, however, cognitive deficits may be associated with increased suffering and treatment-seeking, as well as poorer psychosocial functioning. Premorbid cognitive resources may influence coping style, which may then influence the development of PTSD symptoms, including possible cognitive impairments. Those with significant neuropsychological deficits may have poorer functional outcomes, similar to other psychiatric populations (e.g. patients with schizophrenia; Green et al., 2000). Thus, premorbid cognitive functioning may be highly predictive of post traumatic symptomatology, similar to the 'cognitive reserve' hypothesis accounting for the better functional outcomes of highly educated individuals who develop Alzheimer disease (Stern, 2002).

Further research on the mechanisms that yield cognitive differences among individuals with PTSD, trauma-exposed individuals who do not develop PTSD, and nontraumatized individuals will improve our understanding of the cognitive sequelae of traumatic stress. Based on the current findings, however, we must conclude that college students are not an appropriate population to address these initial questions. Although future longitudinal investigations may show that cognitive deficits emerge over time, it appears that young people with trauma exposure who are able to attend college may represent a 'cognitively resilient' group. The factors underlying their cognitive resiliency remain unclear, but do not appear to include higher IQ, specific neurocognitive strengths or lack of substance use. The relationship between cognitive functioning and everyday functioning in PTSD is not as well understood as it is in Alzheimer disease and schizophrenia, and further research should attempt to clarify the specific cognitive domains that are associated with poorer functional outcomes in PTSD.

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