

Attention, Learning, and Memory Performances and Intellectual Resources in Vietnam Veterans: PTSD and No Disorder Comparisons

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Attention, learning, memory, and estimated intellectual potential were examined in 26 Vietnam veterans diagnosed with posttraumatic stress disorder (PTSD) and in 21 Vietnam veterans without mental disorders. Results revealed PTSD-associated cognitive deficits on tasks of sustained attention, working memory, initial learning, and estimated premorbid intelligence but not on measures of focus of attention, shift of attention, or memory savings. Cognitive task performances adjusted for estimated native intelligence remained negatively correlated with PTSD severity. An intellectual measure adjusted for cognitive task performances was negatively correlated with PTSD severity, even after the authors statistically controlled the level of combat exposure. Results suggested that although intellectual resources may constitute a vulnerability–protective factor for PTSD development, PTSD was associated with cognitive impairment independent of intellectual functioning.

Physiological, neuroimaging, and neuropsychological research have led to significant advances in knowledge of the neurobiological correlates of posttraumatic stress disorder (PTSD) that collectively point to dysfunction of frontal-limbic neural circuits (see Bremner, Southwick, & Charney, 1999; Friedman, Charney, & Deutch, 1995; Pitman, Shalev, & Orr, 2000, for reviews). However, it remains unclear if such neurobiological abnormalities are a result of exposure

to stress or instead reflect premorbid vulnerabilities. Although animal models have suggested that exposure to prolonged laboratory-induced stress results in neurobiological alterations to the organism (see Bremner et al., 1999; Rasmusson & Charney, 1997, for reviews), research examining the neurobiological correlates of trauma-induced stress disorders in humans is necessarily correlational.

Within the behavioral literature addressing the psychobiology of PTSD, there have been two distinct areas of exploration: (a) the examination of the neurocognitive correlates of PTSD and (b) the examination of intellectual functioning as a premorbid vulnerability, or buffer, to psychopathology development following trauma exposure. Neurocognitive indices of frontal-limbic system dysfunction have been well documented in persons suffering PTSD. Although specific cognitive deficit patterns have varied slightly from cohort to cohort, and some studies have failed to uncover evidence of cognitive impairment specifically related to a PTSD diagnosis (cf. Golier et al., 1997; Stein, Hanna, Vaerum, & Koverola, 1999; Zalewski, Thompson, & Gottesman, 1994), research examining neuropsychological functioning in PTSD has generally revealed findings suggestive of mild attention and anterograde memory impairment (Beckham, Crawford, & Feldman, 1998; Bremner et al., 1993; Gilbertson, Gurvits, Lasko, & Pitman, 1997; Jenkins, Langlais, Delis, & Cohen, 1998; Sachinvala et al., 2000; Sutker, Vasterling, Brailey, & Allain, 1995; Uddo, Vasterling, Brailey, & Sutker, 1993; Vasterling, Brailey, Constans, & Sutker, 1998; Yehuda et al., 1995). For exam-

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ple, Vasterling et al. (1998) found that Gulf War (GW) veterans diagnosed with PTSD showed relative impairments in sustained attention, working memory, initial acquisition of information, and sensitivity to retroactive interference compared with GW veterans without mental disorders diagnoses. Other researchers focusing more exclusively on learning and memory have noted PTSD-related deficits in initial learning (e.g., Bremner et al., 1993), delayed recall (e.g., Bremner et al., 1993; Jenkins et al., 1998; Yehuda et al., 1995), and retroactive interference (e.g., Yehuda et al., 1995).

Implicit in the assumptions underlying studies assessing neurocognition in PTSD is that observed cognitive impairments are sequelae of psychological traumatization rather than either precursors of the disorder or artifacts of lower intellectual sophistication. However, associations have been found between PTSD diagnosis and fewer intellectual resources as measured by indirect, preexposure measures of intellectual functioning, such as arithmetic and verbal reasoning tasks (Centers for Disease Control Vietnam Experiences Study, 1988; Pitman, Orr, Lowenhagen, Macklin, & Altman, 1991), the Armed Forces Qualification Test (Maier, 1993), general technical performance (Macklin et al., 1998), educational achievement (Green, Grace, Lindy, Gleser, & Leonard, 1990; Harel, Kahana, & Kahana, 1988; Kulka et al., 1990), and military rank (Sutker, Bugg, & Allain, 1990). Similarly, PTSD has been found to be associated with lower scores on postmilitary estimates of native intellectual functioning, such as the Shipley Institute of Living Scale—Revised (Zachary, 1986) and the Wechsler Adult Intelligence Scale—Revised (WAIS-R; Wechsler, 1981) indices (Gurvits et al., 2000; McNally & Shin, 1995; Vasterling, Brailey, Constans, Borges, & Sutker, 1997).

These findings challenge the assumption that PTSD-related cognitive deficits are simply outcomes of the disorder and raise the question that lower intellectual functioning may be associated with both PTSD development and presence of mild information processing deficits. Although individual strengths and weaknesses are integral to the concept of intelligence, intelligence theory in general purports that, in an intact brain, different abilities, including attention and anterograde memory, tend to be intercorrelated (see Lezak, 1995; Spearman, 1927, for reviews), reflecting proportionate brain development of underlying brain regions (Piercy, 1964). By this view, less proficient attention, learning, and memory performances would be unsurprising in a group with documented detriments in intellectual functioning.

Although much of the research examining neurocognitive functioning in PTSD has attempted to control for the influence of premorbid intellectual factors on cognitive task performance by equating PTSD and comparison samples on intellectual performance scores, the relationship between intellectual resources and neurocognitive dysfunction in PTSD has not been examined directly. Likewise, it is often presumed that retrospectively administered intellectual tasks are robust to acquired attention, learning, and memory deficits, but few attempts have been made in the PTSD literature to parcel out the potential variance in intellectual

performances associated with cognitive dysfunction. The primary goal of this study was to examine neurocognitive performances in attention, learning, and memory domains and performance on a retrospectively administered estimate of premorbid intellectual resources in Vietnam combat-exposed veterans with and without PTSD diagnoses. Study goals were to (a) replicate previous model-driven research examining attention, learning, and memory in GW veterans with relatively recent onset PTSD (Vasterling, Brailey, Constans, & Sutker, 1998) in a sample characterized by chronic presentation of PTSD (i.e., Vietnam veterans); (b) examine intellectual vulnerabilities associated with PTSD; (c) examine the degree to which PTSD is related to attention, learning, and memory performances independently of intellectual functioning; and (d) examine the degree to which PTSD is related to intellectual performance independently of neurocognitive status, as measured by attention, learning, and memory performances.

On the basis of previous research documenting PTSD-related deficits on tasks assessing specific attention, learning, and memory processes (Vasterling et al., 1998) and the body of literature indicating poorer performances associated with PTSD severity on intellectual tasks (Centers for Disease Control Vietnam Experiences Study, 1988; Macklin et al., 1998; McNally & Shin, 1995; Pitman et al., 1991; Vasterling et al., 1997), we hypothesized that PTSD-diagnosed Vietnam veterans would perform more poorly than a sample of Vietnam veterans without mental disorders on tasks reflecting sustained attention, working memory, initial registration of new verbal and nonverbal information, and an estimate of native intellectual functioning derived from the Information and Vocabulary subtests of the WAIS-R. On the basis of animal models of stress exposure that suggest clear neurobiological alterations associated with stress exposure (cf. Bremner et al., 1999; Rasmusson & Charney, 1997) and findings from the psychopathology literature that indicate that preexposure indices of intellectual resources are associated with PTSD diagnosis (Centers for Disease Control Vietnam Experiences Study, 1988; Green et al., 1990; Harel et al., 1988; Kulka et al., 1990; Macklin et al., 1998; Pitman et al., 1991; Sutker et al., 1990), we predicted that neurocognitive and intellectual performances would be independently associated with PTSD severity after controlling statistically for shared variances between the two variable sets.

Method

Participants

Participants were 26 PTSD-diagnosed and 21 psychopathology-free male Vietnam veterans, selected from 181 volunteers who were recruited from newspaper advertisements, veterans' organization newsletters, flyers posted at a Veterans Affairs Medical Center and Veterans Resource Center, and mailings to participants in a PTSD outpatient clinic. The study was described as a research project targeting "human adjustment to various situations" encountered by Vietnam veterans.

Volunteers were not included in the protocol (a) if they reported a history of head trauma other than mild injury (i.e., loss of

consciousness greater than 15 min or requiring medical care), central nervous system disease, systemic medical illness, or developmental problems suggestive of learning disorder or attention deficit hyperactivity disorder or (b) if they were currently taking neuroleptic or anticonvulsant medications. As assessed by the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1996), veterans with current (i.e., previous 3 months) diagnoses of alcohol or substance disorders, lifetime history of bipolar or psychotic disorders, or sub-threshold manifestations of PTSD were also excluded from study participation. In addition, veterans in the no mental disorders comparison sample were excluded if they met criteria for any current Axis I disorder or lifetime PTSD, even if currently asymptomatic.

Deployment status was verified by review of military discharge records (DD-214s), and psychopathology categories were derived from SCID (First et al., 1996) diagnoses. All veterans assigned PTSD diagnoses reported the onset of PTSD symptoms to have been during or shortly after their return from Vietnam. Of the 26 PTSD-diagnosed participants, 14 met criteria for a current comorbid mental disorder, including major depression ($n = 10$), dysthymia ($n = 3$), generalized anxiety disorder ($n = 2$), and specific phobia ($n = 1$). Given that mood and anxiety disorders are sufficiently common among PTSD-diagnosed individuals that they may be conceptualized as subcomponents of PTSD psychopathology (Sutker, Uddo-Crane, & Allain, 1991), study volunteers were not excluded on the basis of these comorbidities. At the time of testing, no veteran in the no mental disorders group was taking psychotropic medications; 16 veterans in the PTSD group were taking psychotropic medications, including antidepressants ($n = 12$), nonantihistamine anxiolytics ($n = 10$), and sleep medication ($n = 7$).

The majority of participants were right-handed (93.6%) and White (63.8%); mean age for all participants was 50.81 years ($SD = 4.11$ years). Veterans served an average of 13.28 months ($SD = 8.35$ months) in the Vietnam War zone. As shown in Table 1, PTSD-diagnosed and psychopathology-free veterans did not differ on these variables. As expected from the high lifetime comorbidity rates of PTSD and alcohol-use disorders observed in Vietnam veterans (Kulka et al., 1990), veterans with current PTSD diagnoses were more likely to meet criteria for a past alcohol-use disorder than veterans without current mental disorder diagnoses (see Table 1). However, PTSD-diagnosed veterans did not differ from the no mental disorders sample in their estimates of average daily alcohol consumption during the year prior to study enrollment, as measured by the Khavari Alcohol Test (Khavari &

Farber, 1978). Neither the overall sample ($M = 0.32$ oz [9.46 ml], $SD = 0.53$ oz [15.67 ml]) nor the two subsamples exceeded the national daily average consumption of 0.85 oz (25.13 ml) of absolute alcohol as reported by the National Institute on Alcohol Abuse and Alcoholism (NIAAA, 1993). The two subsamples did not differ in lifetime nonalcohol substance-use disorder diagnoses.

Measures

Psychopathology and group assignment. Following provision of written informed consent, the SCID and self-report instruments were administered by a master's level technician who underwent over 20 hr of didactic and supervised experiential training in SCID administration. The SCID sessions from this and from a related study were videotaped for subsequent reliability determinations made by one of four licensed clinical psychologists. Using a random sample of 10.0% of the 90 tapes available for review, diagnostic agreement was high for both PTSD diagnosis (100.0%) and non-PTSD Axis I disorders (97.7%). Diagnosis of mental disorders was accomplished using the SCID diagnoses (First et al., 1996). Severity of PTSD symptoms was measured by the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (Keane, Caddell, & Taylor, 1988). Self-reported level of combat exposure was assessed using the Combat Exposure Scale (CES; Keane et al., 1989). Drug and alcohol use was assessed with the Khavari Alcohol Test and with the Alcohol and Nonalcohol Substance Use Disorders module of the SCID.

Attention measures. Participants were next administered the battery of attention and memory measures described by Vasterling et al. (1998). Following Mirsky, Anthony, Duncan, Ahearn, and Kellam's (1991) model, attention was conceptualized as entailing four components: (a) *focus-execute*, the ability to focus on and respond appropriately to environmental cues selected from an array; (b) *sustain*, the maintenance over time of optimal levels of focused attention or vigilance; (c) *shift*, the capacity to change the focus of attention in an adaptive fashion; and (d) *encode*, the ability to register, recall, and manipulate information mentally. To minimize the potential for inflated Type I error, we selected two variables of interest for each of the attention factors: (a) letter cancellation omissions (Talland, 1965) and Stroop Test (Stroop, 1935) interference T scores (focus-execute); (b) Continuous Performance Test (CPT; Conners, 1992) AX paradigm number of hits (i.e., correct responses to targets) and commission errors (sustain); (c) Wisconsin Card Sorting Test (WCST; Berg, 1948) percentage of correct responses and of perseverative responses (shift); and (d) WAIS-R Digit Span and Arithmetic age-scaled scores (encode).

Table 1
Sample Characteristics

Variable	PTSD ($n = 26$)	No mental disorders ($n = 21$)	$F(1, 45)$ or $\chi^2(1, N = 47)$	$p <$
Right-handed	92.31%	95.24%	0.84	.67
Age (years)	50.19 (3.28)	51.57 (4.91)	1.32	.26
Minority	42.30%	28.57%	0.95	.34
Premilitary education (years)	10.64 (1.60)	13.09 (1.70)	25.32	.01
Total education (years)	12.60 (2.20)	14.91 (2.12)	12.96	.01
Time in Vietnam (months)	13.62 (9.59)	12.85 (6.72)	0.94	.77
Average daily alcohol intake (oz)	0.36 (0.60)	0.27 (0.43)	0.38	.54
Diagnosis				
Past alcohol use disorder	73.08%	33.33%	7.42	.01
Past nonalcohol substance use disorder	38.46%	19.04%	2.09	.15

Note. Average daily alcohol intake was in the past year. Values shown with parentheses indicate means and standard deviations. PTSD = posttraumatic stress disorder.

The Stroop Test was administered and scored according to Golden's (1978) method; WCST administration and scoring were accomplished using methods described by Heaton (1981).

Learning and memory measures. Learning and memory of auditory-verbal and visuospatial information were assessed by administration of the Rey Auditory Verbal Learning Test (AVLT; Rey, 1964) and the Continuous Visual Memory Test (CVMT; Trahan & Larrabee, 1988), respectively. Both instruments allow multiple exposures of the material to be learned and allow measurement of initial registration and retention over delayed intervals. Variable selection reflected learning and memory constructs most commonly examined in the PTSD literature. Variables of interest for the AVLT pertained to proficiency of List A recall and included the sum of correctly recalled words from List A, Trials 1–5 (learning); retroactive interference ratio, calculated as List A recall/Trial 5 (memory); and a memory savings ratio, expressed as long-delay recall/short-delay recall (memory). Variables of interest for the CVMT were total correct in acquisition phase (learning) and total correct in delayed recall (memory).

Estimated native intellectual potential. Participants were administered the Information and Vocabulary subtests of the WAIS-R, a multimodal test of intellectual functioning. Thought to measure general fund of information and word knowledge, respectively, the Information and Vocabulary subtests are commonly used as "hold" tests from which estimates of premorbid ability are derived in brain-impaired populations (Lezak, 1995). Subtest data were first transformed from raw to scaled scores using age-appropriate norms. Next, a single variable was created by means of principal components analysis performed on the Information and Vocabulary age-scaled scores yielded by the combined sample. As outlined by Tabachnick and Fidell (1989), Mahalanobis distance with $p < .01$ was calculated to determine whether the combined value of the variables for a given case was unacceptably different from the centroid of the remaining cases, and no multivariate outliers were found. The analysis yielded one primary component, which explained 90.7% of the variance (eigenvalue = 1.81). The only other component was characterized by an eigenvalue of less than one. Each of the two variables included in the analysis contributed positively to the primary component, which was labeled *estimated premilitary IQ* (EPIQ), with component loadings of 0.95 for each variable.

Data Analyses

Group comparisons of education completed, combat exposure, PTSD symptom severity, and EPIQ were accomplished with a univariate analysis of variance approach. A single factor of Group (i.e., PTSD, no mental disorders comparison sample) was examined.

Analysis of attention, learning, and memory data incorporated multivariate omnibus tests of significance to protect against inflation of Type I error, as recommended by Cliff (1987). Because attention, learning, and memory were conceptualized as distinct cognitive domains, separate multivariate analyses of variance (MANOVAs) were performed for each domain. Significant multivariate F ratios were followed by corresponding univariate comparisons. Dependent variables for the attention MANOVA were the eight attention measures described earlier; dependent variables for the learning MANOVA included AVLT List A, sum of correctly recalled words, Trials 1–5, and CVMT total correct in acquisition phase; dependent variables for the memory MANOVA included AVLT retroactive interference and savings ratios and CVMT total correct in delayed recall. To examine the potential influence of medication and comorbid depression on cognitive and intellectual performances, we conducted a series of univariate

comparisons of cognitive performances and EPIQ measures within the PTSD sample. The analyses were performed using medication status (i.e., either taking psychoactive medication or not taking psychoactive medication) as the between-subjects factor and then were repeated using depression diagnoses (i.e., presence or absence of major depressive disorder or dysthymia) as the between-subjects factor.

To examine whether group differences found on cognitive performance measures were artifacts of lower intellectual ability, we conducted a series of correlational analyses. First, standard multiple regression equations were conducted in which EPIQ served as the independent variable and cognitive performance variables found to differ between the two groups served as dependent variables. The standardized residuals from these multiple regression equations were retained as variables, each reflecting a specific cognitive variable adjusted for EPIQ. Second, correlations between IQ-adjusted cognitive performance variables and PTSD severity, as measured by the Mississippi Scale (Keane et al., 1988) were computed.

To examine whether premilitary IQ was related to PTSD severity independently of cognitive performance, similar to the analyses described above, we created a standard multiple regression equation in which cognitive performance variables found to differ between the two groups served as independent variables, and EPIQ served as the dependent variable. The standardized residuals of this equation were retained as a variable reflecting EPIQ adjusted for the cognitive variables. Next, because trauma severity has been shown in prior research to be a significant predictor of PTSD severity (cf. Foy, Osato, Houskamp, & Neumann, 1992), CES scores and EPIQ were entered sequentially as independent variables into a hierarchical multiple regression equation, with PTSD severity as the dependent variable. The sequential entry procedure ensures that any variance in PTSD severity that is accounted for by combat exposure is removed prior to the entry of EPIQ. Thus, any remaining contributions of EPIQ to PTSD severity are an extremely conservative estimate of the relationship between EPIQ and PTSD severity.

Results

Group Comparison: Education, Combat Exposure, and PTSD Symptom Variables

Veterans with PTSD diagnoses completed fewer years of education prior to Vietnam service than veterans without PTSD diagnoses (see Table 1). As expected, PTSD-diagnosed veterans also reported more extensive combat exposure ($M = 27.77$, $SD = 8.09$) on the CES than veterans without mental disorder diagnoses ($M = 13.71$, $SD = 9.49$), $F(1, 45) = 30.05$, $p < .01$, and more severe PTSD symptomatology ($M = 129.62$, $SD = 21.76$) on the Mississippi Scale than the no mental disorders comparison sample ($M = 68.81$, $SD = 13.03$), $F(1, 45) = 126.88$, $p < .01$.

Group Comparisons: Attention, Learning, and Memory Measures

MANOVA revealed that Vietnam veterans suffering PTSD differed significantly from those without mental disorders on tasks of attention, $F(8, 38) = 2.35$, $p = .04$. As shown in Table 2, examination of univariate analyses indicated that, as compared with the no mental disorders sam-

Table 2
Comparison of PTSD-Diagnosed Veterans With Veterans Without Mental Disorders on Attention Measures

Variable	PTSD (<i>n</i> = 26)		No mental disorders (<i>n</i> = 21)		<i>F</i> (1, 45)	<i>p</i> <
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
Focus–execute						
Letter cancellation omissions	2.48	2.43	1.56	2.13	1.87	.18
Stroop Interference <i>T</i> scores	45.81	10.66	49.80	6.81	2.21	.15
Sustain						
CPT commissions	2.81	3.74	1.67	2.01	1.55	.22
CPT hits	73.65	9.71	78.29	1.55	4.68	.04
Shift						
WCST % correct	73.28	15.74	70.76	15.18	0.31	.53
WCST % perseverative	14.94	11.11	19.40	12.46	1.68	.21
Encode						
WAIS–R Digit Span age SS	9.27	2.89	11.10	2.53	5.17	.03
WAIS–R Arithmetic age SS	9.38	2.17	10.62	3.47	2.22	.15

Note. PTSD = posttraumatic stress disorder; CPT = Continuous Performance Test; WCST = Wisconsin Card Sorting Test; WAIS–R = Wechsler Adult Intelligence Scale—Revised; SS = scaled scores.

ple, veterans with PTSD diagnoses correctly responded to fewer CPT stimuli and performed less proficiently on the WAIS–R Digit Span subtest. Thus, PTSD-diagnosed veterans showed relative impairments on the sustain and encode tasks but did not differ from their psychopathology-free counterparts on the focus or shift tasks (all *ps* > .14).

As revealed by multivariate *F* ratios, PTSD and no mental disorders samples also differed on basic learning measures, $F(2, 44) = 3.90, p = .03$. Inspection of univariate comparisons revealed that PTSD-diagnosed veterans correctly recalled fewer words during AVLT learning trials than veterans without mental disorder diagnoses. The two groups did not differ in correct responses during the acquisition phase of the CVMT (see Table 3). In contrast, the MANOVA comparing group performances on measures of memory retention failed to reach statistical significance, $F(3, 43) = 0.17, p = .92$.

Group Comparison: EPIQ

Examination of group means and standard deviations of WAIS–R Information and Vocabulary subtest scores revealed that both samples performed within the average

range (PTSD diagnosed: Information, $M = 10.50, SD = 2.40$; Vocabulary, $M = 10.30, SD = 2.40$; No mental disorders: Information, $M = 12.20, SD = 2.40$; Vocabulary, $M = 11.80, SD = 2.40$). However, compared with their psychopathology-free counterparts, PTSD-diagnosed veterans exhibited less sophisticated intellectual resources as measured by the EPIQ factor, $F(1, 45) = 5.84, p < .02$.

Influence of Psychoactive Medication, Depression, and Prior Alcohol Use on Cognitive and Intellectual Measures

Comparisons within the PTSD sample of the neurocognitive and intellectual performances of veterans taking psychoactive medications ($n = 16$) with veterans not taking psychoactive medication ($n = 10$) revealed that medicated veterans performed more poorly on WAIS–R Arithmetic, $F(1, 24) = 4.95, p = .04$, and made more commission errors on the CPT, $F(1, 24) = 4.29, p = .05$, than their unmedicated counterparts. Nonsignificant trends were also observed within the PTSD sample for medicated veterans to exhibit lower percentages of correct responses, $F(1, 24) = 4.10, p = .05$, and higher percentages of persevera-

Table 3
Comparison of PTSD-Diagnosed Veterans With Veterans Without Mental Disorders on Memory Measures

Variable	PTSD (<i>n</i> = 26)		No mental disorders (<i>n</i> = 21)		<i>F</i> (1, 45)	<i>p</i> < ^a
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
AVLT						
Total recall, Trials 1–5	40.46	8.91	46.33	4.60	7.48	.01
Retroactive interference ratio	0.76	0.21	0.80	0.13	0.45	.51
Savings ratio	0.98	0.29	0.98	0.16	0.00	.97
CVMT						
Total correct, learning	71.69	8.39	72.05	5.80	0.03	.88
Total correct, delay	3.54	1.42	3.59	1.28	0.02	.90

Note. PTSD = posttraumatic stress disorder; AVLT = Auditory Verbal Learning Test; CVMT = Continuous Visual Memory Test.
^a Bonferroni corrected significance level = .01.

tive responses on the WCST, $F(1, 24) = 3.34, p = .08$, than unmedicated veterans. However, medicated and unmedicated PTSD-diagnosed veterans did not differ on any of the cognitive comparisons for which significant differences were found (CPT hits, Digit Span, and AVLT learning trials total score) between the PTSD and the no mental disorders comparison samples ($ps > .17$) or on EPIQ factor scores ($p > .16$). Thus, PTSD-related deficits on these tasks do not appear to be a function of psychoactive medication usage.

Veterans in the PTSD sample with comorbid depression diagnoses ($n = 13$) made more commission errors on the CPT, $F(1, 24) = 4.74, p = .04$, and performed less proficiently on the Stroop Test, $F(1, 24) = 5.54, p = .03$, than did PTSD-diagnosed participants without comorbid depression ($n = 13$). However, the depressed and nondepressed PTSD-diagnosed veterans did not differ on their EPIQ factor scores ($p > .90$) or on any of the cognitive comparisons for which significant differences were found (CPT hits, Digit Span, and AVLT learning trials total score) between the PTSD and the no mental disorders comparison sample ($ps > .42$).

PTSD-diagnosed veterans with a lifetime history of alcohol-use disorders ($n = 19$) did not differ either on any of the cognitive variables or on the EPIQ factor scores ($ps > .13$).

Relationships Among Cognitive Performances, EPIQ, and PTSD Severity

Regression analyses conducted on CPT hits, Digit Span age-scaled scores, and AVLT List A total correct Trials 1–5 to derive cognitive performance residuals adjusted for EPIQ revealed that EPIQ was significantly related to Digit Span age-scaled scores, $r(45) = .40, F(1, 45) = 8.44, p < .01$, explaining approximately 15.8% of the variance in Digit Span scores and significantly related to the sum of correctly recalled words on AVLT List A, Trials 1–5, $r(45) = .42, F(1, 45) = 9.49, p < .01$, explaining approximately 17.4% of the variance in AVLT learning performance. However, EPIQ was not significantly related to CPT hits, $r(45) = .25, F(1, 45) = 2.91, p = .10$, explaining only 6.1% of the variance in CPT hits. The standardized residuals from the three regression equations were retained as variables, each reflecting a cognitive variable with the variance accounted for by EPIQ removed. Two-tailed correlations conducted between EPIQ-adjusted cognitive performance variables and PTSD severity revealed that PTSD severity was negatively correlated with CPT hits, $r(45) = -.43, p < .01$, and with AVLT recall, Trials 1–5, $r(45) = -.30, p = .04$, but not with Digit Span age-scaled scores, $r(45) = .25, p < .10$. Thus, it appears that EPIQ alone does not account for the specific deficits in sustained attention and acquisition of verbal material displayed in the sample.

Table 4 presents results of a standard multiple regression analysis conducted to derive an EPIQ residual adjusted for CPT hits, Digit Span age-scaled scores, and AVLT List A total correct, Trials 1–5. The equation yielded a multiple correlation of $R = .48$, with the three independent cognitive variables collectively accounting for 23.0% of the variance in EPIQ scores. The standardized residual of this equation

Table 4

Relationship of CPT Hits, Digit Span Scores, and AVLT Trials 1–5 to Estimated Premilitary IQ Using Regression Analysis

Variable	<i>B</i>	<i>SEB</i>	β
CPT Hits	.009	.019	.07
Digit Span age scaled scores	.089	.053	.25
AVLT, List A, Trials 1–5	.035	.020	.28

Note. $R^2 = .23, F(3, 43) = 4.33, p < .01$. All β s were nonsignificant. CPT = Continuous Performance Test; Digit Span = Wechsler Adult Intelligence Scale—Revised Digit Span subtest; AVLT = Auditory Verbal Learning Test.

was retained as a variable reflecting EPIQ, with the variance accounted for by the three cognitive variables removed. As displayed in Table 5, results of the hierarchical multiple regression analysis conducted to examine the unique contributions of combat exposure and the EPIQ residual to PTSD severity revealed that each step of the equation was significant, yielding a multiple correlation of $R = .68$ in the final step, with the CES scores and the EPIQ residual accounting for 47.0% of the variance in Mississippi Scale scores. As expected, combat exposure explained a significant proportion (40.0%) of the variance in PTSD severity. Nonetheless, the change in explained variance from Step 1 to Step 2, when the EPIQ residual was added, was also significant, $F(1, 44) = 5.57, p = .02$, and indicated that EPIQ adjusted for cognitive performance contributed 6.7% for the variance in Mississippi Scale scores above that attributable to combat exposure.

Discussion

This study explored the relationship of cognitive performances in attention, learning, and memory domains to intellectual functioning in Vietnam veterans with chronic presentation of combat-related PTSD. Consistent with previous research examining attention, learning, and memory functioning subsequent to acute onset PTSD (Vasterling et al., 1998), Vietnam veterans with PTSD diagnoses performed less proficiently on tasks assessing sustained attention, working memory, and initial registration of verbal information compared with Vietnam veterans without mental disorder diagnoses. Bolstering a growing body of research suggesting that intellectual resources may buffer development of PTSD (Macklin et al., 1998; McNally & Shin, 1995; Vasterling et al., 1997), Vietnam veterans without mental disorders performed more proficiently than veterans diagnosed with PTSD on intellectual tasks thought to reflect native intellectual potential. Results revealed that neurocognitive and intellectual task performances were independently related to PTSD after accounting statistically for shared variance between the two domains.

Consistent with previous research (Bremner et al., 1993; Sutker et al., 1995; Uddo et al., 1993; Vasterling et al., 1998; Yehuda et al., 1995), observed deficits on specific attention and anterograde memory tasks are suggestive of abnormalities in frontal-limbic circuits. The pattern of

Table 5
Relationship of Estimated Premilitary IQ (EPIQ) and Combat Exposure to Mississippi Scale Scores Using Hierarchical Multiple Regression Analysis

Variable	<i>R</i>	β	<i>t</i>	<i>p</i> <
Step 1	.63*			
CES		.63	5.48	.01
Step 2	.68*			
CES		.64	5.80	.01
EPIQ		-.26	-2.36	.03

Note. For Step 1: $R^2 = .40$, $F(1, 45) = 30.00$, $p < .01$; for Step 2: $R^2 = .47$, $F(2, 44) = 19.31$, $p < .01$; and $\Delta R^2 = .07$, $F(1, 44) = 5.57$, $p = .023$. CES = Combat Exposure Scale.

* $p < .01$.

weaknesses involving sustained attention, working memory, and new learning may be attributed to disordered arousal and dysfunction of the prefrontal cortex and possibly, to a lesser extent, the hippocampus (see Vasterling et al., 1998, for a more comprehensive discussion). That significant relationships remained between neurocognitive variables (CPT hits and AVLT learning) and PTSD severity after shared variance with an estimate of native intellectual sophistication was removed suggests that neurocognition is disordered in PTSD independently of intellectual functioning and cannot readily be dismissed as an artifact of reduced cognitive inefficiency related to overall brain integrity.

Findings in the present study differed slightly from those produced by previous research using a GW sample (Vasterling et al., 1998). Results revealed that PTSD-diagnosed Vietnam veterans exhibited a poorer overall hit rate on a continuous performance task than veterans without mental disorder diagnoses, suggesting that omission errors were primarily responsible for CPT performance deficiencies. In contrast, PTSD diagnosis in GW veterans was associated with more errors of commission, but not omission, on the continuous performance task (Vasterling et al., 1998). Although these population-specific performance differences could be attributed to chance, the failure to find a significant difference in the number of commissions in the present study raises the possibility that PTSD-related arousal dysregulation may shift from a pattern of predominant hyperarousal to one of more generally disordered arousal and sustained attention as the disorder becomes more chronic. Alternatively, such cohort-related differences could be related to other population-specific factors, including trauma characteristics, age, and other sociodemographic variables.

The less proficient performance of PTSD-diagnosed veterans relative to their psychopathology-free counterparts on tasks thought to reflect premilitary intellectual ability are consistent with vulnerability models of PTSD that posit intellectual sophistication as a potential vulnerability or buffering factor. When shared variance with neurocognitive functioning, as measured by attention and learning performances, was removed statistically from the estimated premilitary IQ variable, a significant relationship between estimated native intellectual potential and PTSD severity remained. Moreover, consistent with the findings of McNally

and Shin (1995) and Macklin et al. (1998), our results revealed that estimated premorbid intellectual resources contributed significantly and uniquely to the variance in the Mississippi Scale scores after controlling statistically for the level of combat exposure. Taken together, these findings suggest that the relationship between intellectual functioning and PTSD symptom severity cannot be explained solely either by acquired deficits in attention and memory or by veterans with lower IQ being assigned to heavier combat duties.

Although the specific mechanism by which intellectual skills exert protection against psychopathology development following exposure to trauma are unknown, one could speculate that intellectual sophistication serves as a potential protective resource. For example, verbal intelligence may enhance verbal mediation integral to effective forms of active coping, such as the formation of narratives in assimilation of traumatic memories and emotions (Harber & Pennebaker, 1992) or the establishment of more elaborate social support networks (Cohen & Willis, 1985). Similarly, greater intellectual sophistication may facilitate the development of personal resources such as educational attainment, occupational achievement, and other socioeconomic factors, which appear to buffer stress impact (Hobfoll, 1989; Sutker & Allain, 1995; Ursano, Wheatley, Sledge, Rahe, & Carlson, 1986). Additionally, if exposure to stress is associated with neurobiological alterations, as animal models suggest (see Bremner et al., 1999; Rasmusson & Charney, 1997, for reviews), then higher levels of premilitary intelligence may reflect reduced central system vulnerability to stress. Indirect support for this hypothesis can be found among studies documenting increased psychological symptoms in trauma survivors with a history of brain injury (Chemtob et al., 1998; R. F. Mollica, personal communication, August 20, 1998; Vasterling, Constans, & Hanna-Pladdy, 2000).

Several limitations of the present study should be noted. First, it was not feasible to obtain actual premilitary measures of intellectual functioning. Such measures do not readily exist in the military records of current populations of veterans who have been exposed to combat, and even when such records do exist, the context of these military test administrations was not necessarily well controlled. However, our retrospectively derived estimate of premilitary intelligence consisted of tasks commonly used as "hold" tests of intellectual functioning in neuropsychological clinical settings and research designs (Lezak, 1995). That PTSD-diagnosed veterans also completed fewer years of education than those without mental disorders diagnosis supports the validity of our EPIQ factor. Moreover, results of the present study are consistent with previous research incorporating indirect preexposure IQ estimates (Centers for Disease Control Vietnam Experiences Study, 1988; Green et al., 1990; Harel et al., 1988; Kulka et al., 1990; Macklin et al., 1998; Pitman et al., 1991; Sutker et al., 1990).

Second, the study design incorporated a quasi-experimental, descriptive approach. Thus, although intellectual performance and neurocognitive performance were found to be independently related to PTSD severity, causal infer-

ences must necessarily be regarded with caution. However, the hypothesis that general intelligence is a vulnerability factor for PTSD and that PTSD is characterized by neurobiological alterations associated with cognitive impairment is consistent with current neurobiological models of the pathogenesis and maintenance of the disorder and is more parsimonious than the supposition that general intelligence and specific cognitive impairments in attention and memory are either both independent risk factors for the development of PTSD or both independent sequelae of the disorder. Nonetheless, prospective longitudinal designs or studies with sample sizes sufficient to perform path analysis or other causal modeling procedures may be necessary to sort out causal pathways more definitively.

Third, the PTSD-diagnosed and no mental disorders samples differed in current medication usage and historic alcohol-use patterns. However, comparisons of PTSD-diagnosed veterans with and without current psychoactive medication and PTSD-diagnosed veterans with and without past history of alcohol-use disorders revealed that the two groups did not differ on neurocognitive and intellectual variables of interest. Thus, group differences in intellectual and neurocognitive functioning are difficult to attribute either to current medications or to residual neurotoxic effects of prior alcohol abuse or dependence.

Finally, because we did not incorporate a non-PTSD, psychopathology comparison sample, the degree to which intellectual and neurocognitive performance deficits in the present study were either related specifically to stress-related psychopathology or reflected generalized emotional distress or comorbid psychopathology use could be questioned. However, the finding that PTSD-diagnosed veterans with comorbid depression did not differ from PTSD-diagnosed veterans without comorbid depression on key intellectual or cognitive variables suggests that group differences in cognitive and intellectual performance cannot be attributed exclusively to depression. Moreover, although attention deficits have been described in psychologically distressed samples (Breslow, Kocsis, & Belkin, 1980; Sackheim et al., 1992) and in affective disorders (cf. R. A. Cohen & O'Donnell, 1993), our findings did not suggest pervasive attention and memory performance deficits as might be expected if concentration were more generally limited as a result of global distress. Similarly, the PTSD-related deficits on the IQ factor derived from the WAIS-R Information and Vocabulary subtests are atypical of intellectual deficits documented in non-PTSD psychiatric samples (cf. Breslow et al., 1980; Kluger & Goldberg, 1990), suggesting that these deficits may be disorder specific.

Taking into account these methodological weaknesses, this study nonetheless extends previous research by providing preliminary evidence that neurocognitive and intellectual performance deficits are independently related to PTSD. Moreover, by replicating the measurement techniques used in a previous study of neurocognitive functioning in GW veterans, this is the first study to demonstrate systematically that specific attention and memory performance deficits generalize across trauma populations.

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