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Neuropsychological Outcomes in OEF/OIF Veterans With Self-Report of Blast Exposure: Associations With Mental Health, but not mTBI

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Objective: To examine neuropsychological outcomes in veterans of Operations Enduring and Iraqi Freedom (OEF/OIF) with self-reported histories of blast exposure and determine the contribution of deployment-related mild traumatic brain injury (mTBI), and posttraumatic stress disorder (PTSD) and depression to performance. The effect of number of blast exposures and distance from the blast was also assessed. ***Method:*** OEF/OIF veterans who reported exposure to blast underwent structured interviews and were assigned to no-TBI ($n = 39$), mTBI without loss of consciousness (LOC; $n = 53$), or mTBI with LOC ($n = 35$) groups. They were administered tests of executive function, memory, and motor function at least 6 months after the index event. ***Results:*** Neuropsychological outcomes did not differ as a function of mTBI group. Blast load and distance from the blast also did not affect neuropsychological performance. Both PTSD and depression symptoms were significantly associated with neuropsychological outcomes. ***Conclusions:*** A history of mTBI with or without LOC during deployment does not contribute to objective cognitive impairment in the chronic phase post injury. In contrast, PTSD and depression symptoms are associated with cognitive performance decrements. This finding is thought to reflect at least in part the impact of psychiatric distress on neuropsychological performance.

Keywords: TBI, blast injuries, PTSD, depression, neuropsychological outcome

The high prevalence of combat-related traumatic brain injury in military personnel of Operations Enduring and Iraqi Freedom (OEF/OIF) has focused attention on the long-term consequences of mild traumatic brain injury (mTBI). In civilian samples, cognitive deficits and symptoms are transient in a vast majority of patients, and typically resolve within days to weeks (Carroll et al., 2004; Iverson, 2005), but in a minority of patients a constellation of physical, cognitive, and emotional symptoms persist at 3 months postinjury (Pertab, James, & Bigler, 2009; Ponsford et al., 2000). These lingering postconcussion symptoms are nonspecific, and evidence suggests that psychosocial and psychiatric factors play a significant role in their maintenance (McCrae, 2008). With regard to neuropsychological performance, evidence from meta-analytic studies generally supports complete recovery following noncomplicated mTBI (Rohling et al., 2011). However, in individuals with a history of multiple concussions, residual deficits in the domains of executive functioning and memory have been identified (Belanger, Spiegel, & Vanderploeg, 2010).

Extrapolation of these findings regarding recovery to deployment-related mTBI is complicated, not only by the potential for cumulative injury resulting from repetitive blast exposure, but also by the high comorbidity of mental health disorders (Hoge et al., 2008; Schneiderman, Braver, & Kang, 2008; Tanielian & Jaycox, 2008). Postconcussion symptoms are common in OEF/OIF veterans who report mTBI, even years after injury. Yet, evidence suggests that these symptoms in the chronic stage are overwhelmingly associated with posttraumatic stress disorder (PTSD; Belanger, Kretzmer, Vanderploeg, & French, 2010; Drag, Spencer, Walker, Pangilinan, & Bieliauskas, 2012; Hoge et al., 2008; Lippa, Pastorek, Benge, & Thornton, 2010; Polusny et al., 2011; Verfaellie, Lafleche, Spiro, Tun, & Bousquet, 2013) and depression (Hoge et al., 2008; Verfaellie et al., 2013), with minimal evidence for an independent contribution of mTBI.

The evidence concerning the status of objective cognitive functioning in OEF/OIF veterans with mTBI, and the potential source of impairment, is less clear. Focusing on the fact that mTBI and

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PTSD may both impact neuropsychological functioning, several studies (Campbell et al., 2009; Nelson, Yoash-Gantz, Pickett, & Campbell, 2009) have compared the performance of patients with mTBI-only to those with mTBI and PTSD in order to examine the possibility that these conditions may have synergistic negative effects on cognitive functioning. Interpretation of these studies is complicated, however, by the fact that the two groups were not matched in severity or chronicity of TBI. Further, several studies have focused on the comparison of subgroups of mTBI patients with different injury characteristics (Brenner et al., 2010; Cooper, Chau, Armistehad-Jehle, Vanderploeg, & Bowles, 2012; Drag et al., 2012), but these studies have not included a no-TBI control group, and thus leave open the question as to whether there are residual mTBI-associated deficits in this population.

Ivins, Keane, and Schwab (2009) directly addressed the effect of mTBI by administering the Automated Neuropsychological Assessment Metrics (ANAM) to an unselected cohort of soldiers who had served in OEF/OIF up to 2 years prior to study participation. The likelihood of "possible impairment" on the ANAM, defined as performance at least 2 *SD* below the mean on one test or at least 1.5 *SD* below the mean on two tests, did not differ as a function of whether or not soldiers screened positive on a brief mTBI screen. The impact of PTSD on performance was not assessed in that study. Also using the ANAM, Vasterling et al. (2012) reported results of a longitudinal study in which they examined the effect of mTBI associated with loss of consciousness (LOC) in a large sample of U.S. Army soldiers. mTBI was not associated with neuropsychological performance deficits, whereas both PTSD and depression symptoms were associated with decrements in simple reaction time (RT) and efficiency of learning and recall. A major asset of this study is that performance deficits could be estimated directly based on a comparison of pre- and postdeployment performance; a limitation, however, was that the study did not capture individuals with mTBI without LOC. Their inclusion in the control group may have artificially attenuated group differences.

In light of concerns about the sensitivity of the ANAM to mTBI when administered in the postacute phase (Coldren, Russell, Parish, Dretsch, & Kelly, 2012), there is a need for studies using clinical neuropsychological tests of memory and executive function—the domains most sensitive to the acute effects of mTBI. We are aware of only two studies that have compared directly individuals with and without deployment-related TBI. In one study that focused primarily on neural changes associated with blast-induced TBI, Levin et al. (2010) found that veterans with self-report of mild to moderate TBI performed worse than veterans without TBI on a measure of verbal memory. Memory performance in the TBI group was not related to PTSD severity. In contrast, Nelson et al. (2012) reported no differences in performance on a battery of neuropsychological tests as a function of history of blast-related mTBI, but individuals with LOC were minimally represented in the mTBI group. Irrespective of mTBI, however, groups with Axis I psychopathology (including primarily PTSD and depression) performed worse on measures of processing speed and verbal learning and recall.

In light of the limited and somewhat contradictory evidence regarding the chronic effects of deployment-related mTBI on neuropsychological performance, the present prospective study examined the performance of OEF/OIF veterans who report a history of blast exposure on a battery of neuropsychological tests sensitive to the acute effects of mTBI, with the goal of evaluating (a) the association between neuropsychological outcomes and a clinical diagnosis of mTBI with or without LOC; and (b) the influence of PTSD and depression symptoms on neurocognitive performance.

Method

Participants

Participants were 136 OEF/OIF veterans who reported being within 100 m of a blast, and who were recruited through the VA Boston Polytrauma Network and through flyers and outreach events in the community. Participants were recruited for a research study that took place outside the clinical context and was unrelated to diagnostic or treatment purposes. The study consisted of clinical interviews, neuropsychological testing, and self-report measures. Results of self-report measures for a subset of the sample have been reported elsewhere (Verfaellie et al., 2013).

As part of the study, participants were given a symptom validity test (Tombaugh, 1996). Seven participants were excluded from the study because they scored below 45 on the retention trial, demonstrating questionable effort. Two others were excluded with a history of predeployment TBI that was greater than mild in severity. The remaining participants were assigned to one of three groups (no-TBI, TBI without LOC, and TBI with LOC), using the definition of mTBI put forth by the American Congress of Rehabilitation Medicine (1993).

Assessment of TBI was based on an extensive clinical interview, and is described in detail in Verfaellie et al. (2013). In brief, participants were queried about their blast exposure(s) so as to determine the most severe event, which functioned as the index event. They then provided a detailed description of the index event, based on questions regarding their memory of events preceding the blast, experience of the blast itself, and memories of events subsequent to the blast. This description was used to infer the presence and duration of alteration of consciousness (i.e., disorientation, posttraumatic amnesia, LOC). Participants were also queried about the presence of neurological symptoms immediately after the blast that were consistent with TBI. Finally, we inquired whether TBI could be corroborated by medical examination or by witness report. Collateral reports were rarely available; information about injury characteristics depended on participant report. In all but six instances, information regarding the presence and duration of LOC was based on what participants were told by a medic or combat peers who witnessed the event and often were the first to assess a participant's responsiveness.[1]

Participant interviews were transcribed and evaluated by two of the investigators who then sought consensus as to whether a minimal biomechanical threshold for concussion had plausibly been met, and any reported disorientation was the result of concussion rather than situational chaos and confusion. Such decisions are challenging, but unavoidable given that assessment of mTBI in this population is overwhelmingly based on self-report. Of the 127 veterans included in the analysis, 39 were in the no-TBI group, 53 in the TBI without LOC group, and 35 in the TBI with LOC group.

[1] Analysis of the data excluding these six participants yielded identical results.

LOC was estimated to be < 2 min in 22 participants and 2–5 min in 13 participants. Although blast was the immediate antecedent to TBI in all participants with mTBI, we could not ascertain whether primary blast or secondary/tertiary injury mechanisms were primarily responsible for TBI.

Procedures

The neuropsychological battery consisted of tests selected on the basis of their sensitivity to mTBI in the civilian literature, and focused on the domains of attention/executive functioning, verbal and visual memory, and motor ability. As noted above, we also assessed effort, and we used a measure of word reading to estimate premorbid IQ.

Table 1 provides an overview of the tests and variables of interest. Data on two of the attentional measures, Digit Span and Digit Symbol-Coding, were not available for a subset of 25 participants, as these measures were added after initiation of the study. We used the EM algorithm to impute missing data on these cognitive measures. There were several other instances where respondents had missing or invalid scores on a test, but we did not impute in these cases (see Appendix). Reasons for invalid scores included shoulder injury interfering with pegboard performance and English as a second language interfering with WTAR performance.

The Clinician Administered PTSD Scale (CAPS) for *DSM–IV* (Blake et al., 1995) was used to assess PTSD. Continuous CAPS scores were used as a measure of PTSD severity.[2] The SCID overview module (First, Spitzer, Gibbon, & Williams, 2002) was used to assess predeployment psychiatric history. The Beck Depression Inventory (Beck, 1996) was administered to assess current depressive symptoms. Participants were additionally queried about their alcohol use, if they had sustained any head injury prior to deployment, and if they had ever been diagnosed with a learning disability and/or ADHD.

All data were obtained in compliance with regulations of the Institutional Review Boards at VA Boston Healthcare System and Boston University School of Medicine, and all participants provided written informed consent.

Statistical Analyses

For demographic and injury characteristics, one-way analysis of variance (ANOVA) with Duncan's post hoc follow-up (for continuous data) and χ^2(for categorical data) were employed to compare means across groups.

To reduce the cognitive measures and to identify factors corresponding to neuropsychological domains of functioning, factor analysis with oblique (promax) rotation was performed on the cognitive measures. Factor scores were estimated via the regression method as z-scores and served as dependent variables in subsequent analyses examining differences among TBI groups. For completeness, we also report group means on individual neuropsychological test variables (see Appendix).

To assess whether neuropsychological factor scores differed among the three TBI groups, we used multivariate regression analyses. The six cognitive factor scores were simultaneously considered as outcome measures and an unstructured covariance matrix was assumed among them. The initial model tested whether the outcome set differed among the TBI groups. Additional predictors (CAPS and BDI) were included in two separate models that focused on the contribution of PTSD (Model 2) and depression (Model 3), respectively. Entering CAPS and BDI scores as mental health scores in separate models was motivated by their high collinearity. In an initial step of each model, mental health score was entered as a predictor. In a second step, the interaction of TBI group and mental health score was entered in each model. We used the Akaike Information Criterion (AIC) to compare successive models and to evaluate whether including additional predictors improved the model fit. Finally, two additional models focused on the role of injury variables. Here, number of blast exposures (blast load: 1, 2–4, 5 or more) and distance from the index blast (≤ 5 m, > 5 m–50 m, > 50 m–100 m) were the predictors entered as a second step. Age, education, and current alcohol use were entered as covariates in all models but were nonsignificant in all cases and are therefore not reported.

Results

Table 2 presents demographic and clinical information as a function of TBI group. There were no differences among the three groups in age, education, or estimated premorbid IQ (WTAR scores; Fs < 1). The presence of predeployment history of psychiatric disorder, mTBI, and learning/attentional disability also did not differ across groups (χ^2s < 3.8; ps > .15).

With regard to injury and clinical characteristics, there were no differences among groups in time since index blast, $F(2, 126) = 1.1$, $p = .33$, or number of blast exposures, $\chi^2 = 5.4$, $p = .25$. The groups did differ in terms of distance from the blast, $\chi^2 = 31.7$, $p < .001$, Cramer's V = .35, with the no-TBI group more likely to have been at a greater distance from the blast than the other two groups. PTSD scores were marginally different across groups, $F(2, 125) = 2.7$, $p = .07$, $\eta^2 = .04$, with a stepwise increase in CAPS scores from the no-TBI group to the TBI with LOC group. There were no group differences in depression, $F(2, 126) = 1.4$, $p = .24$ or current alcohol consumption, $F(2, 126) < 1$.

Table 3 shows the results of the factor analysis. Based on the scree plot, we extracted six factors, which accounted for 59% of the common variance. These factors were labeled as processing speed, verbal memory, visual memory, motor speed, manual dexterity, and cognitive control.

Table 4 presents mean factor scores by TBI group and Table 5 summarizes the results of the different statistical models. A multivariate regression treating the multiple cognitive factor scores as the outcome and TBI group as the predictor revealed no significant effects. Both CAPS scores and BDI scores were significantly associated with cognitive factor scores. A 10-point increase in CAPS score yielded an estimated decrease in performance across cognitive domains ranging from −0.04 to −0.10 z-score units; a 5-point increase in BDI yielded an estimated decrease from −0.05 to −0.11. In both cases, the estimated decrease was largest for processing speed. Adding the interaction of TBI group and mental health scores to the model further enhanced the model fit. The effect of CAPS score and BDI score remained significant, but in each case the main effect was modified by a significant three-way interaction between TBI group, mental health score, and cognitive

[2] One individual in the no-TBI group did not receive the CAPS.

Table 1
Neuropsychological Tests and Measures

Test	Measures used
D-KEFS Trail Making Test (Delis et al., 2001)	Combined number + letter sequencing time, number-letter switching, motor speed
D-KEFS Color-Word Interference Test (Delis et al., 2001)	Color naming speed, word reading speed, inhibition
D-KEFS Verbal Fluency Test (Delis et al., 2001)	Letter fluency total correct
Digit span (Wechsler Adult Intelligence Scale–III) (Wechsler, 1997)	Digits backward score
Digit Symbol–Coding (Wechsler Adult Intelligence Scale–III) (Wechsler, 1997)	Total correct responses
Auditory Consonant Trigrams (Peterson and Peterson, 1959)	Total number correct 9, 18, and 36 s interference
California Verbal Learning Test (Delis et al., 2000)	Trials 1–5 total, long delay free recall total, total recognition discriminability (*d'*)
Brief Visuospatial Memory Test-Revised (Benedict, 1997)	Total recall, delayed recall, recognition discrimination index
Finger Tapping Test (Halstead, 1947)	Mean number of taps in dominant and nondominant hand
Purdue Pegboard Test (Tiffin, 1968)	Completion time for dominant and nondominant hand
Test of Memory Malingering (Tombaugh, 1996)	Cutoff = score < 45 on retention trial
Wechsler Test of Adult Reading (Wechsler, 2001)	Total correct response

factor. These interactions reflected an interaction between TBI group and each of the mental health scores selectively for the Verbal Memory factor, CAPS: $F(2, 120) = 7.62$, $p < .01$; BDI: $F(2, 121) = 5.14$, $p < .01$. CAPS as well as Beck scores had a detrimental effect on verbal memory in the no-TBI and TBI with LOC groups, but not in the TBI without LOC group.

In two additional models examining the impact of injury variables, blast load and distance from the blast, respectively, were entered in a second step after TBI group. Neither model revealed any significant effects.

Discussion

The present results demonstrate that a remote history of self-reported blast-induced mTBI is not associated with inferior performance on a battery of neuropsychological tests of memory, executive, and motor function—domains sensitive to the effects of acute mTBI. In contrast, mental health problems such as PTSD and depression were associated with small but measureable neuropsychological performance decrements. These findings accord well with those in civilian TBI, which suggest that mTBI has a negligible effect on neuropsychological outcome more than 3 months postinjury (Rohling et al., 2011). They also extend previous results in deployment-related mTBI, and suggest that months or years after injury, objective neuropsychological outcomes, like subjective symptom reports, are primarily associated with psychiatric variables.

This pattern of findings replicates and extends those of Nelson et al. (2012) who also concluded that cognitive impairments in OEF/OIF veterans several years post mTBI, when present, are most likely attributable to PTSD and other psychiatric conditions. Given that their study sample included only a few patients with LOC, their results left open the possibility that mTBI with LOC yields chronic impairment in neuropsychological performance. The present study suggests that this is not the case, as participants

Table 2
Demographic and Clinical Information

	No TBI (n = 39)	TBI w/o LOC (n = 53)	TBI with LOC (n = 35)	Overall (n = 127)
Age, *M* (*SD*)	29.8 (6.6)	30.7 (8.7)	29.5 (7.9)	30.1 (7.8)
Education, *M* (*SD*)	13.2 (1.9)	13.5 (2.0)	13.3 (2.2)	13.4 (2.0)
Males, # (%)	37 (95)	51 (96)	33 (94)	121 (95)
Prior psychiatric diagnosis, # (%)	7 (17.9)	6 (11.3)	4 (11.4)	17 (13.4)
Prior attention/learning deficit, # (%)	10 (25.6)	9 (17.0)	3 (8.6)	22 (17.3)
Predeployment mTBI, # (%)	6 (15.4)	10 (18.9)	4 (11.4)	20 (15.7)
Time since blast in months, *M* (*SD*)	40.1 (25.3)	38.5 (22.7)	45.9 (22.2)	41.0 (23.4)
Distance from blast, # (%)				
≤ 5 m	5 (12.8)	30 (56.7)	25 (71.4)	60 (47.2)
5 m–50 m	22 (56.4)	18 (34.0)	9 (25.7)	49 (38.6)
50 m–100 m	12 (30.8)	5 (14.3)	1 (2.9)	18 (14.2)
Blast exposures, # (%)				
1	7 (17.9)	5 (9.4)	3 (8.6)	15 (11.8)
2–4	12 (30.8)	26 (49.1)	19 (54.3)	57 (44.9)
5 of more	20 (51.3)	22 (41.5)	13 (37.1)	55 (43.3)
CAPS total, *M* (*SD*)	51.9 (24.5)	56.5 (26.3)	65.9 (27.7)	57.7 (26.5)
BDI total, *M* (*SD*)	17.5 (9.6)	19.6 (10.9)	21.7 (10.8)	19.5 (10.5)
# alcoholic drinks/wk, *M* (*SD*)	6.7 (8.8)	6.7 (13.1)	5.7 (8.6)	6.4 (10.7)

Note. TBI = traumatic brain injury; LOC = loss of consciousness; CAPS = Clinician-Administered PTSD Scale; BDI = Beck Depression Inventory.

Table 3
Factor Loadings and Correlations for Cognitive Measures

	Proc. speed	Verbal memory	Visual memory	Motor speed	Manual dext.	Cogn. control
Factor loadings						
Verbal fluency	**.53**	.05	−.04	−.14	−.04	.15
Digit symbol–coding	**.62**	−.10	.05	−.14	.19	.22
Trails motor speed	**.91**	−.03	.08	.04	−.14	−.29
Trails letter + number sequencing	**.67**	.14	.04	.04	.01	−.07
Trails number-letter switching	**.53**	0	.22	−.05	−.04	.10
Stroop word + color naming	**.47**	−.01	−.14	.19	.07	**.36**
Stroop inhibition	**.38**	−.08	−.09	.13	.14	**.46**
Consonant trigrams	.08	.15	.16	.09	−.14	**.53**
Digit span backwards	−.08	.01	.09	−.10	−.09	**.69**
CVLT total trials 1–5	.04	**.74**	.01	−.06	−.01	.05
CVLT delayed recall	.10	**.86**	.03	−.01	−.01	.03
CVLT delayed recognition	−.09	**.77**	−.04	.05	.08	−.02
BVMT-R total trials 1–5	.06	.01	**.79**	.03	.10	−.01
BVMT-R delayed recall	−.02	−.04	**.89**	0	−.01	.09
BVMT-R delayed recognition	.10	.02	**.41**	.04	−.03	.04
Finger tapping dominant	−.04	.01	0	**.91**	−.02	−.07
Finger tapping nondominant	−.04	−.03	.07	**.83**	.02	.04
Grooved pegboard dominant	.01	.05	.07	.02	**.85**	−.16
Grooved pegboard nondominant	−.05	.02	−.02	−.02	**.74**	.02
Factor correlations						
Processing speed	1.00	1.00	1.00	1.00	1.00	1.00
Verbal memory	.33	.40	.20	.36	.20	
Visual memory	.45	.15	.16	.25		
Motor speed	.32	.19	.27			
Manual dexterity	.31	.26				
Cognitive control	.50					

Note. Proc. speed = processing speed; Dext. = dexterity; Cogn. control = cognitive control; CVLT = California Verbal Learning Test; BVMT = Brief Visuospatial Memory Test. Factor loadings > .35 for each test are shown in **bold**.

with mTBI, with or without LOC, performed no differently from those without mTBI. Our results further suggest that the impairment in verbal memory observed by Levin et al. (2010) may be due to the inclusion of patients with moderate TBI in their sample.

Our results add to a growing number of studies of OEF/OIF military personnel that have reported a significant association between neuropsychological outcomes and symptoms of PTSD and/or depression (Campbell et al., 2009; Nelson et al., 2012; Vasterling et al., 2012). Although one study failed to find a significant association with PTSD, this likely reflects limited power due to that study's small sample size (Brenner et al., 2010). These findings are in agreement with a considerable body of research documenting mild impairments in processing speed, memory, and executive functioning in patients with PTSD (Brewin, Kleiner, Vasterling, & Field, 2007; Buckley, Blanchard, & Neill, 2000; Vasterling & Brailey, 2005) or depression (Burt, Zembar, & Niederehe, 1995; Porter, Bourker, & Gallagher, 2007). Interpretation of these findings is hampered by the fact that, in the absence of baseline measures, it is difficult to know the causal relationship between reduced neuropsychological performance and psychiatric distress. This concern is particularly marked for PTSD, given the growing literature identifying lower neuropsychological scores as a preexisting characteristic that may constitute a risk factor for PTSD (Gilbertson et al., 2006; Parslow & Jorm, 2007). Nonetheless, convincing evidence that inferior neuropsychological performance is at least in part attributable to PTSD comes from longitudinal studies documenting changes in cognitive functioning in association with the development (Marx et al., 2009; Vasterling et al., 2012) and resolution of stress-related emotional symptoms (Fann, Uomoto, & Katon, 2001; Walter, Palmieri, & Gunstad, 2010).

Table 4
Neuropsychological Factor Scores by TBI group, M (SD)

	No-TBI (n = 39)	TBI w/o LOC (n = 53)	TBI with LOC (n = 35)
Processing speed	.12 (1.00)	.03 (.86)	−.17 (.99)
Verbal memory	.05 (.90)	.04 (.95)	−.12 (.99)
Nonverbal memory	.10 (.81)	.02 (.91)	−.15 (1.10)
Motor speed	.03 (.92)	.04 (.80)	−.10 (1.14)
Manual dexterity	.06 (.82)	.14 (.94)	−.28 (.93)
Cognitive control	−.04 (.89)	.04 (.86)	−.01 (.90)

Note. Factor scores are in z-units (M = 0, SD = 1). TBI = traumatic brain injury; LOC = loss of consciousness.

There was limited evidence in our study for an interaction between mTBI and PTSD or depression on neuropsychological performance, with the possible exception of verbal memory, where mental health symptoms were associated with worse performance both in the no-TBI and TBI with LOC group, but not in the TBI without LOC group. The results in the no-TBI group and the TBI with LOC group are consistent with a large literature demonstrating a robust association between PTSD and verbal memory (Brewin et al., 2007) as well as between depression and verbal memory (Burt et al., 1995). Why this association was not present

Table 5
Results of the Statistical Models Examining Associations of TBI and Cognitive Factor Scores (Model 1), Adding CAPS Scores (Model 2) and BDI Scores (Model 3) as Predictors

	Model 1			Model 2 (CAPS)			Model 3 (BDI)		
	AIC	*F*	*p*	AIC	*F*	*p*	AIC	*F*	*p*
TBI	1925								
TBI		.92	ns						
TBI × factor		.36	ns						
TBI + MH				1913			1924		
TBI					0.98	ns		0.99	ns
TBI × factor					0.43	ns		0.37	ns
MH					8.38	0.005		9.84	0.002
MH × factor					0.84	ns		1.03	ns
TBI + MH + TBI x MH				1908			1917		
TBI					0.98	ns		0.99	ns
TBI × factor					0.44	ns		0.37	ns
MH					8.50	0.005		9.89	0.002
MH × factor					0.85	ns		1.03	ns
TBI × MH					0.49	ns		0.03	ns
TBI × MH × factor					3.03	0.002		3.46	0.001

Note. CAPS = Clinician-Administered PTSD Scale; BDI = Beck Depression Inventory; AIC = Akaike Information Criterion; TBI = traumatic brain injury; MH = mental health.

in the TBI without LOC group is a puzzle, and future studies will be needed to determine whether this finding can be replicated. The lack of interaction between mTBI and PTSD in other cognitive domains, however, should not lead to the conclusion that mTBI and trauma-related mental health conditions are unrelated. There is considerable evidence that mTBI confers additional risk for common trauma-related psychopathologies such as PTSD and depression (Bryant et al., 2010; Hoge et al., 2008; Schneiderman et al., 2008; Vasterling, Constans, & Hanna-Pladdy, 2000). In our study as well, PTSD symptoms tended to be more pronounced in individuals who suffered mTBI with LOC. Alterations in executive function and memory in the acute phase of mTBI may affect posttrauma psychological adjustment, and several mechanisms by which this may occur have been proposed (Verfaellie, Amick, & Vasterling, 2012). Neurocognitive inefficiencies have been demonstrated in the acute phase of blast-induced mTBI (Cooper et al., 2010; Luethcke, Bryan, Morrow, & Isler, 2011), and as such, these may influence the development and course of PTSD. Despite resolution of these mTBI-associated deficits, neurocognitive inefficiencies may persist months or years later as a result of neurobiological alterations associated with PTSD (Rao, Suvrathan, Miller, McEwen, & Schattarji, 2009; Southwick et al., 2007).

Although a history of blast-related mTBI was not associated with neuropsychological outcome in this or in previous studies (Nelson et al., 2012; Vasterling et al., 2012), the possibility remains that clinical neuropsychological tests are not sufficiently sensitive to detect residual cognitive inefficiencies in individuals with a history of remote mTBI. In the literature on civilian mTBI, several studies using specialized cognitive measures have documented chronic impairments in more taxing tasks of complex attention and cognitive control (Ellemberg, Leclerc, Couture, & Daigle, 2007; Pontifex, O'Connor, Broglio, & Hillman, 2009), even in the absence of deficient performance on standard neuropsychological tests. Further, altered cerebral activation patterns have been observed in the context of normal behavioral performance, suggesting that additional processing resources may be required to support performance following mTBI (Chen, Johnston, Petrides, & Ptito, 2008; Witt, Lovejoy, Pearlson, & Stevens, 2010). Future studies targeting working memory and cognitive control in the context of functional imaging will be important in blast-induced mTBI as well (for an example, see Scheibel et al., 2012).

Given the lack of a significant association between blast-related mTBI and neuropsychological outcome, it may not be surprising that distance from the blast did not impact performance. Yet, experimental studies in animals indicate that distance from the blast (or its correlates, intensity, and duration of the blast wave) affects physiological and cognitive changes (Cernak & Noble-Haeusslein, 2010). An important consideration in understanding this discrepancy is the fact that in animal studies, outcomes are measured much closer to the time of injury. Additionally, experimental and clinical findings may be difficult to compare given the complexity of blast waves and the variability in the nature of the injury in-theater. Finally, self-reported estimates of distance may not be reliable, although this concern is reduced by virtue of the fact that distance was meaningfully related to severity of mTBI.

Number of blast exposures also did not impact neuropsychological outcome in this study. Based on a meta-analysis of results from the sports literature, Belanger, Spiegel, & Vanderploeg (2010) concluded that although the overall effect of multiple concussions was minimal, report of multiple concussions was associated with poorer performance on measures of delayed memory and executive functioning. Our failure to find a dose-response relationship may be due to the fact that not all blast exposures lead to concussion, although a study that directly measured lifetime concussion in OEF/OIF soldiers also failed to find an effect of number of TBIs (Ivins, Keane, & Schwab, 2009). The long time since injury and the reliance on self-report data to assess the number of blast or TBI events may be other reasons for the null effect, both in our study and in that of Ivins et al. (2009). Based on a retrospective review of records of military personnel who completed the military version of the Immediate Post-Concussion Assessment Cognitive Test, Kontos et al. (2013) found that num-

ber of blast mTBIs was associated with slower RT, but not with performance in memory or visual processing speed. However, a similar dose-response function was observed for PTSD symptoms, and thus it is possible that the relationship between number of mTBIs and RT was mediated by PTSD.

Similar to many previous studies of deployment-related mTBI, assessment of TBI in this study relied on retrospective self-report, because medical records are rarely available. This limitation merits highlighting, especially given that the TBI assessment occurred well after the injury, and therefore is subject to misremembering and reporting bias. Within the context of this limitation, however, participant report was guided by an in-depth structured interview, which is considered the gold standard for diagnosis (Corrigan & Bogner, 2007). Additionally, the fact that the large majority of individuals who were classified as having mTBI with LOC provided information conveyed by a witness alleviates concerns about exclusive reliance on patients' memory for the event.

In comparison with other studies of deployment-related mTBI, our study has a number of unique strengths. We examined performance on neuropsychological tests selected to be sensitive to the effects of mTBI in a relatively large sample of subjects, which included participants with LOC as well as without LOC. The inclusion of a control group of participants who were exposed to blast but did not suffer mTBI ensured comparable deployment experience among the groups and allowed control for potential effects of deployment per se on neuropsychological performance (Vasterling et al., 2006). Further, by treating PTSD and depression symptoms as continuous variables, we had greater power to isolate the effects of mTBI and mental health factors, respectively.

Our findings not only lead to a better understanding of the complex comorbidity of mTBI and psychiatric symptoms, they also have important clinical implications. On the one hand, the fact that a history of remote blast-induced mTBI has minimal impact on current neuropsychological functioning validates the implementation of educational approaches early following mTBI that support the expectation of full recovery (Mittenberg, Tremont, Zielinski, Fichera, & Rayls, 1996; Paniak, Toller-Lobe, Reynolds, Melnyk, & Nagy, 2000; Ponsford et al., 2002). On the other hand, the association between neuropsychological outcome and psychiatric symptoms reinforces the importance of early mental health intervention for returning veterans who have suffered blast exposure, with the possibility that interventions targeting emotional symptoms may alleviate neuropsychological impairment as well. Preliminary results in this regard are promising (Fann et al., 2001; Walter et al., 2010), but further investigation is clearly needed.

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(*Appendix follows*)

Appendix

Summary of Neuropsychological Test Results, *M* (*SD*)

	No-TBI (n = 39)	TBI w/o LOC (n = 53)	TBI with LOC (n = 35)
WTAR	0.26 (0.88)	0.35 (0.70)	0.36 (0.77)
Trails motor speed	0.70 (0.52)	0.65 (0.38)	0.63 (0.43)
Trails letter + number sequencing	0.59 (0.87)	0.53 (0.96)	0.19 (1.04)
Trails number-letter switching	0.16 (0.96)	0.11 (0.82)	−0.14 (1.03)
Stroop word + color naming	0.08 (0.78)	0.00 (0.88)	0.00 (0.96)
Stroop inhibition	−0.41 (1.17)	−0.12 (1.17)	−0.24 (1.23)
Digit symbol	0.06 (1.04)	−0.22 (0.92)	−0.44 (0.95)
Verbal fluency	0.20 (1.14)	0.14 (1.20)	−0.05 (1.09)
Digit span backwards	0.02 (0.85)	0.04 (1.00)	0.17 (0.90)
Consonant trigrams mean[a]	−0.86 (1.18)	−0.82 (1.06)	−1.14 (1.12)
CVLT total trials 1–5	−0.04 (1.00)	0.01 (0.87)	0.12 (1.00)
CVLT delayed recall[b]	−0.22 (1.09)	−0.26 (1.25)	−0.52 (1.27)
CVLT delayed recognition[b]	0.02 (0.90)	−0.07 (1.02)	−.23 (0.90)
BVMT total trials 1–5	−0.55 (1.19)	−0.66 (1.26)	−1.00 (1.53)
BVMT delayed recall	−0.77 (1.29)	−0.88 (1.39)	−1.01 (1.68)
BVMT delayed recognition	−0.53 (1.68)	−0.38 (1.73)	−0.56 (1.77)
Finger tapping dominant	0.47 (0.94)	0.43 (0.78)	0.32 (1.10)
Finger tapping nondominant	0.59 (0.89)	0.62 (0.82)	0.50 (1.14)
Grooved pegboard dominant[c,d]	−0.19 (0.82)	−0.20 (0.78)	−0.62 (0.81)
Grooved pegboard nondominant[c,d]	−0.32 (0.74)	−0.07 (0.87)	−0.34 (0.87)

Note. TBI = traumatic brain injury; LOC = loss of consciousness. Scores are expressed in *z*-units (M = 0, SD = 1).
[a] 1 TBI without LOC missing. [b] 1 no-TBI missing. [c] 1 TBI with LOC excluded. [d] 1 TBI with LOC missing.

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