



## Posttraumatic stress disorder in OEF/OIF veterans with and without traumatic brain injury



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### ABSTRACT

Veterans of Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF) are presenting with high rates of co-occurring posttraumatic stress disorder (PTSD) and traumatic brain injury (TBI). The purpose of this study was to compare the clinical presentations of combat-veterans with PTSD and TBI ( $N=40$ ) to those with PTSD only ( $N=56$ ). Results suggest that the groups present two distinct clinical profiles, with the PTSD + TBI group endorsing significantly higher PTSD scores, higher overall anxiety, and more functional limitations. The higher PTSD scores found for the PTSD + TBI group appeared to be due to higher symptom intensity, but not higher frequency, across PTSD clusters and symptoms. Groups did not differ on additional psychopathology or self-report of PTSD symptoms or executive functioning. Further analysis indicated PTSD severity, and not TBI, was responsible for group differences, suggesting that treatments implicated for PTSD would likely be effective for this population.

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### 1. Introduction

According to the Department of Veterans Affairs (2012), approximately 2.4 million troops deployed to Iraq and Afghanistan between October 2001 and March 2012. Of the nearly 1.5 million OEF/OIF/OND (Operation Enduring Freedom [OEF]; Operation Iraqi Freedom [OIF]; Operation New Dawn [OND]) veterans who have left active duty, approximately 54% have already sought Veterans Affairs (VA) health care since 2002, with 228,361 receiving diagnoses of posttraumatic stress disorder (PTSD; Veterans Affairs, 2012). Prevalence studies of combat-related PTSD among OEF/OIF/OND service personnel indicate a range of 2.2–17.3% (Hermann, Shiner, & Friedman, 2012; Richardson, Frueh, & Acierno, 2010) with best estimates coming in at around 8% (Richardson et al., 2010; Smith et al., 2008).

Additionally, and of equal concern, a number of veterans are suffering from physical trauma resulting in traumatic brain injury (TBI). TBI is defined by temporary or permanent neurological dysfunction (i.e., either loss of consciousness or altered mental status)

resulting from either a closed or penetrating head injury (Marshall et al., 2012). Advances in military protective equipment and medical care, coupled with the proliferation of high explosive weaponry used by military combatants (e.g., improvised explosive devices and rocket-propelled grenades), have resulted in increasing rates of both survival and traumatic brain injury (TBI) (Shively & Perl, 2012; Vasterling, Verfaellie, & Sullivan, 2009). Incidence rates of TBI garnered from electronic medical records by the Department of Defense (2012) report that over 253,000 United States military personnel have received medical diagnoses of TBI from 2000 to August 2012, with nearly 77% of diagnoses falling in the mild range (mTBI). mTBI is characterized by confusion or disorientation for less than 24 h, loss of consciousness for up to 30 min, memory loss for less than 24 h, and normal brain imaging results (Department of Defense, 2012).

Veterans who screen positive for TBI are more likely to receive VA benefits (Carlson et al., 2010), with annual costs reported to be four times higher than veterans without TBI diagnoses (Taylor et al., 2012). Due to the high prevalence of TBI among returning veterans, and the resulting increased demand for treatment and healthcare, understanding the sequelae and long-term implications of TBI have become critical. The resulting clinical presentation can vary significantly based on a number of factors and may include cognitive, physical, and psychological difficulties (Hoge et al., 2008). The persistent symptoms of TBI, or postconcussive symptoms (PCS), are self-reported somatic, cognitive, and affective symptoms occurring post injury (Morissette et al., 2011). PCS of mTBI include

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headaches, poor sleep, dizziness, balance problems, irritability, and concentration and memory difficulties (Hoge et al., 2008; Shively & Perl, 2012). Understanding the relationships between PCS, TBI, and PTSD is complicated by significant symptom overlap (e.g., irritability, sleep difficulty, and impaired concentration; for review, see Morissette et al., 2011).

In addition to PCS, comorbid psychiatric diagnoses are often present in individuals suffering from TBI. Carlson et al. (2010) found that 80% of OEF/OIF veterans with positive TBI screens had additional psychiatric diagnoses. Specifically, veterans with positive TBI screens were three times more likely to have an additional diagnosis of PTSD and two times more likely to have additional diagnoses related to substances or depression compared to those with negative TBI screens. In Army Active Duty and Reservist personnel who deployed to Iraq and experienced mTBI with loss of consciousness, 43% endorsed co-occurring PTSD and 23% endorsed depression (Hoge et al., 2008). Higher rates of PTSD in those with comorbid TBI have also been reported in civilian samples (e.g., Bryant et al., 2009). In a recent review of co-occurring PTSD and mTBI in both military and civilian samples (Carlson et al., 2011), frequency of comorbidity varied from 0% to 89%; however, the frequency of comorbidity of probable PTSD in veterans with probable mTBI ranged from 33% to 39% among the three largest studies evaluating Afghanistan and Iraq veterans. It is important to note that all three of these studies utilized self-report screening measures distributed by mail or conducted by telephone interview, highlighting the need for studies using validated assessment methods (Carlson et al., 2011). Despite these assessment limitations, it is clear that veterans returning from recent wars are presenting with high rates of this comorbidity.

Although research has clearly demonstrated higher rates of PTSD diagnoses in veterans with deployment-related TBI compared to veterans without TBI (Carlson et al., 2010, 2011; Hoge et al., 2008; Morissette et al., 2011; Walker, Clark, & Sanders, 2010), researchers are just beginning to understand the differential clinical presentation of PTSD with and without TBI, as few studies to date have directly compared these discrete populations. What is known, however, is that both groups report comparable levels of suicide risk as measured by suicidal ideation, hopelessness, pain, emotional support, marital and employment status, and other axis I disorders (Barnes, Walter, & Chard, 2012), as well as comparable psychosocial outcomes (e.g., self-report of social adjustment, somatic symptoms, and quality of life; Polusny et al., 2011). Research on the presence/absence of PCS varies, as one study found comparable rates in an Army Fort Carson Brigade Combat team (Polusny et al., 2011), whereas another study found a stronger association of PCS and PTSD in US National Guard Soldiers with PTSD with mTBI than either those with mTBI alone or PTSD alone (Brenner et al., 2010). Both studies utilized large samples and were based on self-report of PCS one year post deployment.

Conversely, veterans suffering from PTSD with mTBI are reported to have significantly more severe PTSD symptoms than those with PTSD alone as assessed by the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995) total score (Barnes et al., 2012). Although Barnes et al. (2012) also reported a nonsignificant trend of higher reexperiencing symptom scores for those with PTSD and TBI compared to those with PTSD alone, authors did not comment on additional symptomology assessed by the CAPS (e.g., additional clusters or specific symptom intensity or frequency). Extant literature also suggests that PTSD severity appears to confound or mediate the treatment outcome of comorbid mTBI and PTSD, as mTBI and associated PCS appear to have little impact on outcome measures after adjustment for PTSD severity (Hoge et al., 2008; Polusny et al., 2011; Schneiderman, Braver, & Kang, 2008; Wilk, Herrell, Wynn, Riviere, & Hoge, 2012).

Since PTSD symptoms appear to lead to various negative outcomes, and comorbid mTBI increases the severity of these symptoms, understanding the specific clinical presentation of PTSD in those with TBI is an important area of study. Expanding the literature to include a comparison of each symptom cluster, as well as the specific frequency and intensity of each individual PTSD symptom, will elucidate the individual differences among these two populations, clarifying why veterans with PTSD and TBI have higher PTSD scores. Veterans returning from recent wars are presenting with high rates of both TBI and PTSD, highlighting the imperative need to better understand the compounding impact of the comorbidity upon the treatment needs of returning veterans. In this investigation, we provide a first attempt to compare the specific clinical presentation of PTSD in OEF and OIF veterans suffering from PTSD with and without TBI, highlighting the clinical, functional, and potential treatment outcome implications of this increasingly common comorbidity.

## 2. Methods

### 2.1. Participants

The sample consisted of individuals who had served in Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF) and who were seeking treatment in a randomized controlled trial of Trauma Management Therapy for PTSD. The research trial, funded by the US Army Military Operational Medical Research Program, was located at two sites: the University of Central Florida (UCF) and the Medical University of South Carolina/Ralph Johnson Veterans Affairs Medical Center (MUSC). All procedures were approved by the Institutional Review Boards at both UCF and MUSC. The treatment program consisted of virtual-reality assisted exposure therapy to address PTSD and group therapy designed to address depression, anger and social isolation. Participant recruitment consisted of advertising through clinician referral, radio, various websites, and public events, and in the case of MUSC, through the PTSD clinic at the Ralph Johnson VAMC in Charleston, South Carolina. Following a telephone screen, participants came to the clinic for the pre-treatment diagnostic interview. All data reported in this investigation are from the pre-treatment assessment.

Ninety-six participants (90.6% male) with a mean age of 35.48 ( $SD=9.64$ ; range = 21–63) were included in this study. The sample was 55.2% Caucasian, 24% Hispanic/Latino, 14.6% African American, 2.1% Asian/Pacific Islander, 1.0% Biracial, and 3.1% other. Forty-nine percent of participants were married, 32.3% were single, 12.5% were divorced, 5.2% were separated, and 1.5% reported domestic partners. Twenty-five percent of participants completed high school only, 54.2% completed some college, 16.7% had earned a Bachelor's degree, and 5.2% had earned a Master's degree. The majority of the sample served in the Army (69.8%), followed by the Marine Corps (19.8%), Air Force (7.3%), Navy (2.1%), and as a Civilian Contractor (1.0%). At the time of assessment, 27.1% were active duty and 44.8% were service-connected for disability.

All participants met *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., Rev.; DSM-IV-TR; American Psychiatric Association, 2000) criteria for PTSD as assessed by the CAPS (Blake et al., 1995). Additionally, 41.7% of participants reported receiving diagnoses of TBI. Finally, 63% of participants met criteria for a comorbid mood disorder, 21% met criteria for a comorbid substance use disorder, and 15% met criteria for an additional anxiety disorder. There were no significant group differences on any participant or clinical characteristics with the exception of age. Veterans with PTSD and TBI were significantly younger than veterans with PTSD only,  $t(93.79)=-2.92$ ,  $p<.01$ , a consistent finding in the literature (Carlson et al., 2010; Hoge et al., 2008; Taylor et al., 2012).

## 2.2. Assessment battery

### 2.2.1. PTSD and TBI

The CAPS (Blake et al., 1995) is a clinician-administered interview to assess for the presence of PTSD by evaluating both the frequency and intensity of the 17 DSM-IV PTSD symptoms on a zero to four Likert-scale. The symptom scores of the three subscales (Criterion B reexperiencing, Criterion C avoidance and numbing, and Criterion D hyperarousal) are derived by summing the frequency and intensity scores for relevant individual items. Summing the subscale scores provides overall frequency, intensity, and total PTSD scores. The CAPS is a widely used standard measure of traumatic stress with excellent reliability, convergent and discriminant validity, diagnostic utility, and sensitivity to clinical change (Weathers, Keane, & Davidson, 2001).

The CAPS was administered to participants by licensed clinical psychologists, post-doctoral fellows, or supervised senior doctoral students. For the majority of participants, the CAPS was administered in the clinic. In 18% of participants, the CAPS was administered over the telephone. These participants lived at a significant distance from the UCF clinic and a decision was made to determine the likelihood of a PTSD diagnosis prior to having them incur travel expenses to Orlando only to be told that they did not qualify for participation in the treatment study. Twenty percent of interviews were randomly selected and scored by a blinded clinician for inter-rater reliability (ICC = .993).

The PTSD Checklist-Military Version (PCL-M; Weathers, Litz, Herman, Huska, & Keane, 1993) is a commonly used 17-item self-report measure of military-related PTSD symptoms. Participants rate the degree to which they were bothered by each symptom over the past week, using a five point Likert-scale of 1 (not at all) to 5 (extremely). The PCL-M evidences good reliability and validity (Wilkins, Lang, & Norman, 2011) and is highly correlated with the CAPS (0.93; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996).

TBI status was determined based on self-report by the participant, in most instances based on the presence of a previous diagnosis. Participants were asked whether they had been diagnosed with a TBI as a result of their service during both the phone screen and assessment battery. The PTSD only group had no history of a TBI diagnosis, whereas the PTSD + TBI group responded affirmatively to prior diagnosis of TBI.

### 2.2.2. Other aspects of psychopathology

Clinicians screened for presence of additional axis I disorders using the computer-assisted Structured Clinical Interview for DSM-IV-TR, Clinician Version (SCID-CV; First, Spitzer, Gibbon, & Williams, 1996). Twenty percent of interviews were randomly selected and scored by a blinded clinician for inter-rater reliability of PTSD diagnosis ( $\kappa = 1.00$ ).

The clinician version of the Hamilton Rating Scale for Anxiety was utilized to assess overall anxiety in the past month (HAM-A; Hamilton, 1959). Clinicians rate the anxiety symptoms on a 0 (none) to 4 (very severe) Likert-scale. The measure consists of 14 items with a total score range of 0–56. Twenty percent of interviews were randomly selected and scored by a blinded clinician for inter-rater reliability (ICC = .953).

Health status was assessed using the Version 2 of the SF-36 Health Survey (SF-36v2; Ware, Kosinski, & Dewey, 2003). This 36-item self-report measure assesses health status and functioning over the past month including: Physical Functioning (PF), limitations in physical activities due to health problems, Role Physical (RP), problems with work or daily activities as a result of physical health, Bodily Pain (BP), a measure of pain and limitations due to pain, General Health (GH), personal evaluation of health, Vitality (VT), energy and fatigue, Social Functioning (SF), limitations in social activities due to physical or emotional problems, Role

**Table 1**  
CAPS scores for PTSD and PTSD + TBI groups.

Variable	PTSD N=56 M (SD)	PTSD + TBI N=40 M (SD)	t	p
Overall PTSD symptoms				
Total Score	77.36 (20.40)	88.63 (16.75)	2.87	.005
Total Frequency Score	40.71 (11.22)	45.15 (9.29)	2.05	.043
Total Intensity Score	36.64 (10.01)	43.40 (8.19)	3.51	.001
Reexperiencing symptoms				
Criterion B Total Score	20.61 (8.09)	24.60 (7.00)	2.52	.013
Criterion B Overall Frequency	10.07 (4.57)	11.65 (3.81)	1.79	.077
Criterion B Overall Intensity	10.54 (3.92)	12.95 (3.75)	3.03	.003
Avoidance and numbing symptoms				
Criterion C Total Score	30.73 (9.64)	34.95 (8.60)	2.21	.030
Criterion C Overall Frequency	16.63 (5.38)	18.50 (4.73)	1.77	.080
Criterion C Overall Intensity	14.11 (4.77)	16.45 (4.24)	2.48	.015
Hyperarousal symptoms				
Criterion D Total Score	26.02 (6.40)	29.08 (5.54)	2.44	.017
Criterion D Overall Frequency	14.02 (3.81)	15.08 (3.42)	1.40	.165
Criterion D Overall Intensity	12.00 (3.04)	14.00 (2.55)	3.40	.001

Note. Mean scores for individual items are available from the authors by request.

Emotion (RE), problems with work or daily activities as a result of emotional health, and Mental Health (MH), personal evaluation of emotional health (Ware, Kosinski, & Dewey, 2000; Ware & Sherbourne, 1992). Scores are reported as norm-based *T*-scores with lower scores representing more difficulty. The measure has high reliability coefficients (0.93–0.95) and small confidence intervals (Ware et al., 2003).

### 2.2.3. Executive functioning

The Behavior Rating Inventory of Executive Function – Adult Version (BRIEF-A) self-report form (Roth, Isquith, & Gioia, 2005) measures an individual's perception of their executive functions. The 75-item measure comprises nine clinical scales: Inhibit, Self-Monitor, Plan/Organize, Shift, Initiate, Task Monitor, Emotional Control, Working Memory, and Organization of Materials. These nine clinical scales form two broader indexes, Behavioral Regulation (BRI), a measure of regulatory control of behavior and emotional responses, and Metacognition (MI), a measure of cognitive attention and problem solving. These two indexes form the overall summary score, the Global Executive Composite (GEC). Scores are reported as norm-based *T*-scores, with higher scores reflecting more difficulty, and a *T*-score of 65 or greater indicating significant difficulty. The BRIEF-A adequately assesses patients with TBI and evidences strong reliability in this particular population (0.94–0.96; Waid-Ebbs, Wen, Heaton, Donovan, & Vellozo, 2012).

## 3. Results

An independent-samples *t*-test reported significant group differences for the CAPS total score (combining symptom frequency and intensity),  $t(94) = 2.87$ ,  $p < .01$ , CAPS total symptom frequency,  $t(94) = 2.05$ ,  $p = .04$ , and CAPS total symptom intensity  $t(94) = 3.51$ ,  $p = .001$ . In each case, scores were significantly higher for the PTSD + TBI group compared to the PTSD only group (see Table 1 for group scores).

In order to analyze which factors may have contributed to the overall group difference, CAPS criterion subscale scores (Criteria B, C, and D) were analyzed with a one-way between groups (PTSD vs. PTSD + TBI) multivariate analysis of variance (MANOVA). The results of the MANOVA revealed a significant main effect of TBI on the combined dependent variables,  $F(3,92) = 2.75$ ,  $p = .04$ ,  $\eta^2 = .08$ , Wilks' Lambda = .92. See Table 1. Significant overall effects were followed with analysis of simple effects for subscores and

individual item scores. Results for each of the individual criterion subscale scores are reported below.

Follow-up analysis for Criterion B (reexperiencing symptoms) indicated that the PTSD + TBI group ( $M = 12.95$ ,  $SD = 3.75$ ) endorsed greater overall symptom intensity compared to the PTSD group ( $M = 10.54$ ,  $SD = 3.92$ ),  $t(94) = 3.03$ ,  $p < .01$ . Significant group differences were found for the individual Criterion B symptoms of flashbacks and psychological distress. Specifically, PTSD + TBI group reported more intense flashbacks ( $M = 2.00$ ,  $SD = 1.22$ ) than the PTSD group ( $M = 1.25$ ,  $SD = 1.41$ ),  $t(94) = 2.72$ ,  $p < .01$ . Also, the PTSD + TBI group ( $M = 2.93$ ,  $SD = 0.80$ ) reported more intense psychological distress than the PTSD group ( $M = 2.13$ ,  $SD = 1.11$ ),  $t(94) = 3.89$ ,  $p < .001$ . Participants with PTSD + TBI ( $M = 1.53$ ,  $SD = 1.09$ ) also endorsed a significantly greater frequency of flashbacks compared to the PTSD group ( $M = 0.96$ ,  $SD = 1.19$ ),  $t(94) = 2.36$ ,  $p = .02$ . There were no significant group differences for intensity or frequency on the other re-experiencing symptoms (intrusive recollections, distressing dreams, or physiological reactivity on exposure to cues) or on the overall Criterion B symptom frequency score.

Consistent with Criterion B results, participants with comorbid TBI ( $M = 16.45$ ,  $SD = 4.24$ ) endorsed greater overall intensity for Criterion C symptoms (avoidance and numbing) compared to participants with PTSD ( $M = 14.11$ ,  $SD = 4.77$ ),  $t(94) = 2.48$ ,  $p = .02$ , but no group difference on overall symptom frequency. With respect to individual Criterion C symptoms, participants with PTSD + TBI ( $M = 1.33$ ,  $SD = 1.23$ ) reported more frequent inability to recall important aspects of the trauma as compared to the PTSD group ( $M = 0.75$ ,  $SD = 1.08$ ),  $t(94) = 2.42$ ,  $p = .02$ . The PTSD + TBI group ( $M = 1.90$ ,  $SD = 1.68$ ) also reported more intense inability to recall important aspects of the trauma as compared to the PTSD group ( $M = 0.86$ ,  $SD = 1.29$ ),  $t(94) = 3.30$ ,  $p < .01$ . There were no significant group differences for the intensity or frequency of any other avoidance and numbing symptoms (avoidance of thoughts and feelings, avoidance of activities, places, or people, diminished interest in activities, detachment or estrangement, restricted range of affect, or a sense of a foreshortened future).

With respect to Criterion D (hyperarousal symptoms), participants with PTSD + TBI ( $M = 14.00$ ,  $SD = 2.55$ ) again had significantly higher overall symptom intensity compared to the PTSD group ( $M = 12.00$ ,  $SD = 3.04$ ),  $t(94) = 3.40$ ,  $p < .01$ , but not higher overall symptom frequency. With respect to the individual Criterion D symptoms, participants with PTSD + TBI ( $M = 2.48$ ,  $SD = 0.91$ ) endorsed significantly higher intensity for difficulty concentrating compared to the PTSD group ( $M = 1.95$ ,  $SD = 1.07$ ),  $t(94) = 2.54$ ,  $p = .01$ . The PTSD + TBI group ( $M = 3.13$ ,  $SD = 0.72$ ) also reported more intense hypervigilance than the PTSD group ( $M = 2.71$ ,  $SD = 0.80$ ),  $t(94) = 2.58$ ,  $p = .01$ . Finally, the PTSD + TBI group ( $M = 2.55$ ,  $SD = 1.04$ ) reported more intense exaggerated startle responses compared to those with PTSD ( $M = 1.95$ ,  $SD = 1.23$ ),  $t(94) = 2.53$ ,  $p = .01$ . There were no significant group differences for intensity on the other hyperarousal symptoms (difficulty sleeping or irritability), nor were there any group differences on frequency of any Criterion D symptom.

As age was significantly different between the two groups, a one-way between-groups analysis of covariance (ANCOVA) was conducted to rule out age as contributory to group differences on the CAPS total score. Results found no significant main effect for the age covariate,  $F(1,94) = 1.32$ ,  $p = .25$ ,  $\eta^2 = .01$ , but a significant main effect for TBI,  $F(1,94) = 9.49$ ,  $p < .01$ ,  $\eta^2 = .09$ , suggesting that the CAPS total score was significantly different between groups when controlling for age. Additionally, to rule out increased memory impairment (as a result of TBI) as contributory to group differences on the CAPS total score, CAPS Inability to Recall total score served as a covariate in an additional ANCOVA. Results found a significant main effect for the memory covariate,  $F(1,94) = 6.94$ ,  $p = .01$ ,  $\eta^2 = .07$ ,

and a significant main effect for TBI,  $F(1,94) = 4.07$ ,  $p = .05$ ,  $\eta^2 = .04$ , suggesting that the CAPS total score was still significantly different between groups when controlling for the Inability to Recall total score.

Additional independent-samples *t*-tests examined potential group differences on the PCL-M and HAM-A total scores, as well as the BRIEF and SF-36 factor scores. The PTSD + TBI group reported higher HAM-A total scores,  $t(93) = 2.39$ ,  $p = .02$ ; however, there was no significant group difference on the PCL-M total score. With respect to the SF-36v2, the PTSD + TBI group endorsed lower *T*-scores on the Role Physical,  $t(92) = 2.75$ ,  $p = .02$ ; and Role Emotional,  $t(92) = 1.99$ ,  $p = .05$ , factors. No group differences were reported for any factors of the BRIEF-A. See Table 2 for mean scores for all variables.

To rule out PTSD severity as contributory to the HAM-A total score, and SF-36 Role Physical and SF-36 Role Emotional scores, data were re-analyzed using a one-way between-groups multivariate analysis of covariance (MANCOVA), where total CAPS score served as the covariate. Results revealed a significant main effect for the PTSD covariate,  $F(3,88) = 23.78$ ,  $p < .001$ ,  $\eta^2 = .45$ , Wilks' Lambda = .55, but there was no main effect for TBI status after controlling for PTSD severity,  $F(3,88) = 0.65$ ,  $p = .59$ ,  $\eta^2 = .02$ , Wilks' Lambda = .98, suggesting that PTSD severity, and not TBI per se, was the primary reason for the group differences on these measures.

#### 4. Discussion

This study compared the clinical presentation of military personnel with PTSD, with and without comorbid TBI. Consistent with prior research (Barnes et al., 2012), the PTSD + TBI group was rated as having more severe PTSD (measured by the CAPS total score) compared to participants with PTSD only. A unique finding from our investigation is that the elevated CAPS total score found for the comorbid group was a product of higher symptom intensity ratings. In other words, the PTSD + TBI group endorsed higher PTSD scores as a result of more intense, but not more frequent symptoms. With two exceptions, both groups endorsed comparable symptom frequency; however, individuals with comorbid TBI perceived the symptoms as more extreme or distressing.

Higher rates of PTSD following TBI may be attributable to mechanisms of brain injury (Bryant, 2008; Bryant et al., 2009), which may also explain the perception of more intense PTSD symptoms in individuals with TBI. For instance, biological models suggest that PTSD involves an exaggerated amygdala response and impaired functioning in the ventral/medial prefrontal cortex and hippocampus, which may result in inability to inhibit fear reactions, increased focus on trauma related stimuli, and impaired learning and memory (Rauch, Shin, & Phelps, 2006). Relatedly, mTBI can often damage the prefrontal cortex as a result of shearing forces of the frontal regions of the brain against the skull (Bryant, 2008). Therefore, damage to these and other relevant brain networks following TBI may impair brain functioning involved in the regulation of anxiety (Bryant, 2008; Kennedy et al., 2007), which may lead to more intense PTSD symptoms than would be expected in PTSD alone.

Similarly, cognitive models posit that maladaptive cognitive strategies following trauma can lead individuals to persistently evaluate the traumatic event as a threat, which can lead to PTSD (Ehlers & Clark, 2000). This perception of constant threat is derived from excessively negative appraisal (e.g., overgeneralization of danger or exaggeration of probability of additional traumatic events) and memory difficulties (e.g., poor elaboration and integration which can lead to strong perceptual priming and associative memories). Furthermore, mTBI has been associated with poorer cognitive functioning (e.g., poorer processing speed, attention, concentration, mental flexibility, and memory; Landre, Poppe, Davis,

**Table 2**  
Additional Comparisons for PTSD and PTSD + TBI groups.

Variable	PTSD M (SD)	PTSD + TBI M (SD)	<i>t</i>	<i>p</i>
PCL-M total score (N=95)	61.64 (13.48)	62.54 (10.30)	0.35	.727
HAM-A total Score (N=95)	23.04 (8.37)	27.08 (7.69)	2.39	.019
SF-36v2 Physical Function T Score (N=93)	46.17 (10.06)	42.95 (9.77)	1.54	.127
SF-36v2 Role Physical T Score (N=94)	42.27 (11.38)	36.67 (10.22)	2.75	.016
SF-36v2 Bodily Pain T Score (N=94)	39.76 (11.32)	36.67 (9.78)	1.38	1.71
SF-36v2 General Health T Score (N=93)	43.39 (11.43)	42.23 (9.94)	0.51	.612
SF-36v2 Vitality T Score (N=94)	40.29 (8.37)	40.69 (9.80)	0.21	.832
SF-36v2 Social Functioning T Score (N=94)	33.64 (11.79)	30.59 (10.00)	1.31	.192
SF-36v2 Role Emotional T Score (N=94)	35.75 (12.18)	30.79 (11.39)	1.99	.049
SF-36v2 Mental Health T Score (N=94)	33.65 (10.88)	32.62 (8.85)	0.49	.624
BRIEF-A Inhibit T Score (N=38)	64.20 (10.38)	63.15 (15.52)	0.22	.829
BRIEF-A Shift T Score (N=38)	65.40 (13.91)	62.38 (11.15)	0.68	.504
BRIEF-A Emotional Control T Score (N=38)	66.36 (10.51)	61.92 (10.85)	1.22	.230
BRIEF-A Self-Control T Score (N=38)	60.36 (10.33)	61.08 (11.84)	0.19	.848
BRIEF-A Initiate T Score (N=38)	65.56 (10.22)	61.77 (14.10)	0.95	.348
BRIEF-A Working Memory T Score (N=38)	66.92 (12.32)	71.85 (16.26)	1.05	.302
BRIEF-A Plan/Organize T Score (N=38)	59.12 (10.74)	61.38 (13.93)	0.56	.581
BRIEF-A Task Monitor T Score (N=38)	60.20 (10.61)	59.85 (14.91)	0.09	.933
BRIEF-A Organization of Materials T Score (N=38)	64.77 (12.22)	50.00 (12.55)	1.26	.215

Note. PCL-M, HAMA, and SF-36v2 sample sizes vary due to missing data. The BRIEF-A was administered to subsample only.

Schmaus, & Hobbs, 2006), which perhaps may impede individuals from appropriately utilizing their cognitive resources, resulting in maladaptive appraisal of the trauma (Bryant, 2008). This suggests that the brain injury of TBI may result in cognitive disruption, which when coupled with the presence of PTSD, may exacerbate the inability to manage the trauma, leading to symptoms that are more intense or distressing.

Relatedly, research on the effect of TBI on emotion processing has generally focused on recognition of emotion, typically assessed through the classification of facial expressions. However, recent research suggests that TBI patients suffering frontal-lobe damage are not only impaired in labeling facial expressions, but also rate the emotions as more intense than matched controls, regardless of whether the emotion was correctly or incorrectly identified (Callahan, Ueda, Sakata, Plamondon, & Murai, 2011). Perhaps this negative response bias generalizes to individuals' perception of the emotional intensity of their own psychiatric symptoms, regardless of their frequency. Overall, these findings suggest that individuals with TBI may experience psychiatric symptoms as more intense due to a liberal or negative bias, that may in part, be a function of their brain injury. Whether the more intense emotional response found among participants with TBI is specific to PTSD or also exists among individuals with TBI and other psychiatric disorders (such as depression) merits further research.

Given the combat experiences of this sample, the presence of TBI in individuals with PTSD may indicate greater trauma exposure (Bryant et al., 2009), as well as more severe traumatic events when compared to traumatic events that do not include brain injury. Extant literature suggests that the intensity of PTSD symptomatology is affected by both the total number and intensity of traumatic events in civilians (Lauterbach & Vrana, 2001; Zawadzki & Popiel, 2012) and combat veterans (Ferrier-Auerbach, Erbes, Polusny, Rath, & Sponheim, 2010; Forbes et al., 2012; Foy, Sippelle, Rueger, & Carroll, 1984). Thus, the presence of TBI may indicate more frequent or more severe traumatic events, which then may lead to increased symptom intensity.

In summary, it is likely that some combination of brain disruption, cognitive impairment, altered emotional processing, and trauma frequency/intensity are implicated in the increased symptom intensity found among individuals with PTSD + TBI. Having now identified the potentially relevant factors, future research should carefully examine the role of these factors. Such studies

should include examining structural and functional aspects of brain injury, cognitive strategies, subjective emotional experiences, and both quantitative and qualitative aspects of traumatic events to elucidate the mechanisms of action responsible for more severe PTSD symptoms.

Although the PTSD + TBI group was rated as experiencing more severe PTSD as assessed by the CAPS, there was no significant difference between groups on the PCL-M total score, a finding consistent with prior research (Barnes et al., 2012). This finding suggests a discrepancy between measures, despite the strong correlation between both measures' total scores in our sample ( $r = .066$ ,  $p < .001$ ). There are at least two possible reasons for this discrepancy. First, the discrepancy may be due to the difference of the time frame assessed by the measures, as the CAPS assesses the past month and the PCL-M assesses the past week. Second, the CAPS assesses for both intensity and frequency of PTSD symptoms, whereas the PCL-M assesses how much participants have been bothered by the symptoms (representing a generic measure of symptom intensity). As a result, the CAPS may provide better specificity of the clinical picture of each PTSD symptom, as the PCL-M appears to assess for a more subjective rating of disturbance. These findings highlight the incongruity of self-report perceptions and clinician ratings of PTSD symptoms, highlighting the need for multimodal assessments of psychopathology (De Los Reyes, Bunnell, & Beidel, 2013).

With respect to other measures of psychopathology, the PTSD + TBI group reported higher overall anxiety but not higher overall levels of other psychopathology. This suggests that the significantly higher HAM-A score reflects the generally higher level of arousal found among individuals with PTSD. The finding that groups did not differ on the presence of additional axis I disorders is consistent with other investigations (Barnes et al., 2012). Although higher rates of depression have been reported for veterans with either PTSD or TBI (e.g., Carlson et al., 2010; Hoge et al., 2008; Morissette et al., 2011; Taylor et al., 2012) our results suggest that the presence of TBI does not increase the probability of depression over and above the presence of PTSD.

Consistent with the hypothesis that the HAM-A total score reflected the generally higher arousal associated with PTSD, all clinically significant group differences on additional measures of psychopathology disappeared when controlling for PTSD severity, a finding also reported by others (Hoge et al., 2008; Polusny et al.,

2011; Schneiderman et al., 2008; Wilk et al., 2012). This finding suggests that clinical and functional outcomes which appear to be of poorer prognosis for those in the PTSD + TBI group may be accounted for by the presence of more intense PTSD symptoms. However, as TBI status in this investigation was retrospectively assessed, these conclusions should be interpreted with the appropriate caution.

Interestingly, despite an association between impaired executive functions and TBI (e.g., Bogdanova and Verfaellie, 2012), there was no significant group difference on any BRIEF-A clinical scale, suggesting that military personnel with TBI and PTSD do not perceive themselves to experience greater executive dysfunction than those with PTSD only. However, a perusal of raw scores suggested that both groups evidenced clinical levels of difficulty on at least one clinical scale, and borderline clinical levels on others. For instance, both groups reported experiencing clinically significant difficulty with working memory, and those with PTSD only reported clinically significant emotional control and initiation and shift difficulties. The lack of significant group differences may reflect reduced statistical power, as the BRIEF-A was administered to a subsample of participants only ( $N = 38$ ). Additionally, as the BRIEF-A measures self-perception of difficulty, significant differences in executive functioning may exist but not be apparent to the individual. To explore this hypothesis, sensitive neuropsychological tests and a larger sample would be needed.

The PTSD + TBI group endorsed significantly more problems and difficulty with work and daily activities as a result of both physical and mental health as assessed by the SF-36v2. Examination of individual items on these subscales suggest that participants with PTSD + TBI reported decreased time in productivity, reduced accomplishments, more limitations, more difficulty performing, and less careful execution of work or other activities as a result of both emotional and physical health. Initially suggesting that the presence of TBI may lead to functional limitations above and beyond the presence of PTSD, this finding was no longer significant once accounting for PTSD severity. This suggests that individuals with more severe PTSD, regardless of TBI status, may experience increased functional limitations as a result of more severe PTSD.

In light of these findings, limitations of the current study should be addressed. First, the authors did not independently assess for TBI, but rather relied on retrospective self-report of previous diagnosis. Although this method may result in false positives and false negatives within both groups, Carlson et al. (2011) found no clear differences in frequency rates of TBI and PTSD based on assessment methods (i.e., structured interviews versus self-report screening measures). Second, due to the nature of the TBI screening, the severity of TBI was unable to be determined, hindering examination of how TBI severity affects PTSD severity. Even so, it is likely that the majority of our sample presented with mTBI due to the physical and cognitive demands required to present for this study. With these limitations in mind, the study has several strengths. Specifically, structured clinical interviews (i.e., the SCID and CAPS) were utilized in the assessment of PTSD and additional axis I disorders, whereas the majority of previous studies have relied on self-report measures. Additionally, all participants in the sample met diagnostic criteria of PTSD providing a homogeneous sample for the comparison of veterans with and without TBI.

Taken together, the findings of this study shed light on the clinical presentation of PTSD in veterans with and without TBI, elucidating the specific symptom profile of comorbid PTSD and TBI. Extant literature has substantiated more severe PTSD in those with TBI, and the findings of the present study provide evidence that more severe PTSD is a result of more intense, and not generally more frequent, symptoms. As combat veterans returning from recent wars are presenting with this “signature injury” at an alarming rate, treatment implications for this population are in urgent

demand. These findings suggest that empirically supported treatments implicated for PTSD would likely be effective in reducing the more intense PTSD symptoms, as well as associated difficulties, which present in individuals with TBI.

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