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The Influence of Traumatic Brain Injury on Treatment Outcomes of Concurrent Treatment for PTSD and Substance Use Disorders Using Prolonged Exposure (COPE) in Veterans

Daniel F. Gros[*], **Cynthia L. Lancaster**, **Michael David Horner**, **Derek D. Szafranski**, and **Sudie E. Back**

Ralph H. Johnson Veterans Affairs Medical Center, Charleston, SC

Department of Psychiatry and Behavioral Sciences, Medical University of South Carolina, Charleston, SC

Abstract

Background—The co-occurrence of posttraumatic stress disorder (PTSD), substance use disorders (SUD), and traumatic brain injury (TBI) in veterans of Operations Enduring/Iraqi Freedom and New Dawn has received much attention in the literature. Although hypotheses have been presented and disseminated that TBI history will negatively influence treatment response, little data exist to support these claims. The present study investigates the influence of TBI history on response to COPE (Concurrent Treatment of PTSD and SUD Using Prolonged Exposure), a 12-session, integrated psychotherapy designed to address co-occurring PTSD and SUD.

Method—Participants were 51 veterans with current PTSD and SUD enrolled in a clinical trial examining COPE. Assessments of PTSD symptoms, substance use, and depression were collected at baseline and each treatment session. A TBI measure was used to dichotomize veterans into groups with and without a history of TBI (*n*s = 30 and 21, respectively).

Results—Participants with and without TBI history demonstrated significant improvements in PTSD and depression symptoms during the course of treatment. However, participants with TBI history experienced less improvement relative to participants without TBI history.

Conclusions—The present findings suggest that, although patients with a TBI history respond to treatment, their response to treatment was less so than that observed in patients without a TBI history. As such, identification, symptom monitoring, and treatment practices may require alteration and further special consideration in individuals with PTSD, SUD and TBI.

Keywords

COPE; TBI; PTSD; Alcohol; Comorbidity; Veterans

[*]Corresponding author. Mental Health Service 116, Ralph H. Johnson VAMC, 109 Bee Street, Charleston, SC 29401, United States. Tel.: +1 843 789 7311; fax: +1 843 805 5782. grosd@musc.edu (D.F. Gros).

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1. Introduction

Traumatic brain injury (TBI) has been labeled a signature injury of the wars involved in Operations Enduring Freedom/Iraqi Freedom/New Dawn (OEF/OIF/OND).[1–3] A TBI is defined as a temporary or permanent neurological dysfunction resulting from a trauma-induced external force, and can range from mild to severe in presentation.[3] Over the course of the OEF/OIF/OND conflicts, nearly 250,000 cases of TBI were reported among service members.[1] Of these cases, 75% were classified as mild (mTBI), characterized by confusion or disorientation for less than 24 hours, loss of consciousness (if present) for less than 30 minutes, memory loss for less than 24 hours, and normal brain imaging results.[4] Given the rates of TBI among veterans, the Department of Defense and Department of Veteran Affairs (VA) have prioritized the identification, assessment, and treatment of veterans with TBI.[5–6]

Further complicating the high incidence of mTBI in veterans has been the demonstrated relation between mTBI and psychiatric symptoms. For example, a positive screening of mTBI has been associated with higher rates of co-occurring posttraumatic stress disorder (PTSD), substance use disorders (SUD), and depression,[2,7–8] even after controlling for combat exposure.[2] Other studies have found that veterans with PTSD and mTBI history report more severe PTSD symptoms than veterans with PTSD and no history of mTBI.[4–5,9] In addition, studies of TBI and SUD suggest a trend of increased alcohol use over time in participants with TBI,[10–12] although the clinical significance of the increased severity remains unclear.[9]

Although many studies suggest that complications from mTBI resolve within three months of the event,[13] a recent meta-analysis has identified very mild but persistent cognitive impairments after mTBI (e.g., executive functioning, verbal delayed memory, and processing speed).[14] There also are concerns that complications from mTBI history may have a negative impact on the learning processes involved in EBPs for PTSD and other conditions, such as the processing of trauma-related thoughts and feelings.[15–17] In fact, specific treatment recommendations have been proposed for veterans with PTSD and mTBI history to address the increased complexity and potential severity of symptoms in this population.[6] Several preliminary studies have investigated the influence of mTBI history on PTSD treatment outcomes.[18–21] Most of these studies involved a PTSD-specific EBP, a small sample of patients with a history of mTBI, and demonstrated improvements during treatment. In the only two studies to include a control group without TBI history, no differences were found between groups in outcomes for an EBP for PTSD.[19,21] However, the authors acknowledge that the sample size limited their ability to adequately test for group differences.

With regard to SUD treatments, studies typically observe promising effects for a range of SUD interventions.[22–24] However, similar to the TBI/PTSD literature, comparison of treatment response among patients with and without TBI history is strikingly rare, and this literature has predominantly focused on differences in baseline severity rather than treatment response.[22–25] Interestingly, baseline studies have shown that patients with TBI histories tend to use alcohol and drug less than patients without TBI histories,[9,23] although

preliminary findings have found that SUD in patients with TBI, especially milder TBIs, may be more resistant to treatment.[23] Together, these findings indicate that more research is needed to investigate the potential influence of mTBI history on outcomes of EBPs for PTSD, SUD and related conditions.

The goal of the present study, therefore, was to investigate the relation among mTBI history and symptom reduction during the course of an EBP for comorbid PTSD and SUD. We hypothesized that participants with and without a history of mTBI would demonstrate significant improvements in PTSD, SUD, and depression during the course of treatment based on the similar findings for each group reported separately in the literature.[17–21] In addition, we also hypothesized that participants without a history of mTBI would demonstrate greater improvements in these outcomes as compared to participants with a history of mTBI.

2. Materials and Methods

2.1. Participants

Participants were 51 veterans of the United States military (92.2% male; M_{age} = 39.8, SD = 10.8) with co-occurring PTSD and substance use disorders. The data were collected as part of a NIDA-sponsored, randomized controlled trial investigating the efficacy Concurrent Treatment for PTSD and Substance Use Disorders Using Prolonged Exposure (COPE), an integrated, exposure-based psychosocial treatment for co-occurring PTSD and SUD.[26–27] Participants were recruited from VA treatment clinics, newspaper and internet advertisements, and flyers posted at local mental health clinics and colleges. Baseline inclusion criteria involved: 1) status as a combat or non-combat veteran, reservist, or member of the National Guard, 2) 18–65 years old, 3) meet DSM-IV criteria for a current substance use disorder and have some substance use consistent with diagnosis in the past 90 days for any of the assessed substances (e.g., alcohol, amphetamine, marijuana, cocaine, hallucinogen, inhalant, opioid, phencyclidine, and anxiolytic/sedative-hypnotic), 4) meet DSM-IV criteria for current PTSD and have a score of 50 or higher on the Clinician Administered PTSD Scale (CAPS),[28] and 5) fluency in English. Baseline exclusion criteria included: 1) current suicidal or homicidal ideation and intent, 2) current or history of psychotic or bipolar affective disorders; 3) current eating disorder or dissociative identity disorder; 4) individuals already participating in ongoing PTSD or SUD treatment; and 5) severe cognitive impairment as indicated by the Mini Mental Status Exam. Ongoing treatment via psychotropic medication was permitted.

2.2. Procedure

Potential participants were first screened on the phone or in person and were given a full description of the study. If eligible and interested, they were asked to come into the office for a baseline assessment to further evaluate eligibility. All participants were asked to read and sign an institutional review board-approved informed consent form before any study procedures or assessments were conducted. The baseline assessment involved semi-structured clinical interviews, including the CAPS to assess PTSD symptoms and the Mini International Neuropsychiatric Interview (MINI) to assess SUD and other disorders.[28–29]

Participants also completed a series of self-report measures, including the Structured Assessment for Evaluation of TBI (INTRuST, 2012),[9,30] PTSD Checklist (PCL),[31] Beck Depression Inventory II (BDI-II),[32] and Timeline Follow-Back (TLFB).[33] Participants were then randomized to receive either COPE or Relapse Prevention as part of a larger clinical trial. For the purposes of the present study, only participants in the COPE condition were investigated. The COPE treatment consists of 12 weekly, individual, 90-min sessions which are primarily focused on psychoeducation and methods for coping with cravings to use (sessions 1 – 3), in vivo exposure (sessions 3 – 12), and imaginal exposure (sessions 4 – 11).[26]

2.3. Measures

2.3.1. PTSD Symptoms—The CAPS is a semi-structured clinical interview considered the gold standard for PTSD assessment, was used to obtain a current diagnosis of PTSD and ensure a symptom severity score ≥ 50 at baseline.[28] The PCL-M was administered weekly to assess change in PTSD symptoms during treatment.[31] Internal consistency was good to excellent in the current sample (α = .86 – .96). Minimal missingness (< 1%) of individual items within available observations was imputed using last observation carried forward.

2.3.2. Substance Use—The Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998), a structured interview with strong psychometric properties, was used to identify the presence of a substance use disorder at baseline. We assessed the percent of days using alcohol (PDU) for two months prior to baseline and weekly during treatment using the TLFB.[33] Although other substances also were assessed by the TLFB, the frequency of use was insufficient to include the findings in the analyses and/or discussion (e.g., stimulant use in only 15.7% of sample, opiate use in only 10.0% of sample).

2.3.3. Traumatic Brain Injury—The SAFE-TBI was designed to identify the level of evidence for exposure to mTBI.[30] The SAFE-TBI includes a definition of mTBI as well as three multi-part screening questions, regarding: 1) exposed to, screened for, or put on restricted duty due to any head or brain injury from six examples (e.g., blast or explosion); 2) associated symptoms that occurred immediately after the head or brain injury (e.g., duration of loss of consciousness, feeling dazed or confused, amnesia); and 3) memory loss of events just before or after the injury. Evidence for mTBI was defined as having a credible incident in Question 1 and either one item in Question 2 marked “yes” or Question 3 marked “yes.” The SAFE-TBI has been investigated in three cohorts of military personnel, demonstrating moderate levels of agreement for inter-rater and test-retest reliabilities, which improved with greater strength/severity of evidence for TBI.[30] In addition, the SAFE-TBI demonstrated reasonable convergent validity with more extensive measures of TBI in a VA sample.[9,30] The SAFE-TBI has been used to assess mTBI in similar recent studies.[30]

2.4. Data Analysis

All participants were separated into two groups based on the scoring criteria of the SAFE-TBI [history of TBI with loss of consciousness (LOC), n = 30; no TBI/TBI without LOC, n = 21]. TBI+LOC-positive and control were used as the group labels (without specifiers of mild, moderate, or severe TBI), as both mTBI and more severe presentations were included

in this group (mTBI = 93.3%, $n = 28$; moderate-severe TBI = 6.7%, $n = 2$) as determined by the duration of the LOC (mTBI = less than 30 minutes; moderate-severe TBI = greater than 30 minutes). Upon identification of the two groups (TBI+LOC positive and control), χ^2 tests of independence (for categorical variables) and one-way analyses of variance (ANOVAs; for continuous variables) were used to investigate differences in demographic variables (gender, race, relationship status, employment, service in OEF/OIF/OND, VA disability, age, and years of education) as well as session attendance and treatment discontinuation. In addition, analyses of covariance (ANCOVAs) were used to investigate group differences in treatment outcome variables (PCL-M, TLFB-Alcohol, and BDI) with the matching baseline symptom measure entered as covariates (PCL-M, TLFB-Alcohol, and BDI). In cases that prematurely discontinued treatment, the last observation carried forward method was used in the ANCOVA analyses.

3. Results

3.1. Baseline Demographics

All demographic variables for the two groups are presented in Table 1. There were no group differences in any of the demographic variables.

3.2. Group Differences in Treatment Retention and Completion

Of the 51 participants enrolled in COPE, 36 (70.6%) completed the intervention. Participants were classified as treatment completers if they completed at least 8 of the 12 sessions and at least 3 imaginal exposure sessions (Brady et al., 2001). Despite observed group differences between the control (19.0%; $n = 4$) and TBI+LOC-positive (36.7%; $n = 13$) with regard to treatment completion, the findings were not statistically significant [$\chi^2 = 1.9$, $p = .17$]. There were no group differences between the control ($M = 10.0$ sessions; $SD = 3.7$) and TBI+LOC-positive ($M = 8.9$ sessions; $SD = 3.6$) in the number of sessions attended ($F = 1.1$, $p = .29$).

3.3. Group Differences in Treatment Outcome

The treatment outcome findings are presented in Table 2. As can be seen, significant group differences in post-treatment PTSD symptoms (PCL-M) and depression symptoms (BDI) were observed, when controlling for baseline symptomatology (Fs > 4.7, ps < .04). There were no group differences in post-treatment percent of days of alcohol use (TLFB; $F = 0.3$, $p = .86$). Pair-samples t-tests and Cohen's d effect sizes were computed to further investigate the group differences. As presented in Table 2, both groups demonstrated significant improvements in PTSD and depression symptoms (ts > 3.5; ps < .01; ds > 0.75). However, the control participants demonstrated larger effect sizes for improvements in PTSD ($d = 1.61$) and depression ($d = 1.46$), as compared to the TBI+LOC-positive participants (PTSD $d = 0.85$; depression $d = 0.66$) (ps < .035). No group differences were found in improvements in alcohol use ($F = 0.3$; $p = 0.86$).

4. Discussion

The present study investigated the relation between a history of TBI with LOC and treatment outcome of an integrated, exposure-based treatment for comorbid PTSD and SUD. In contrast to prior studies investigating the relation between mTBI and EBPs for PTSD,[19] the present study included a somewhat larger sample of patients with a history of TBI and LOC to more adequately test for differences in treatment response among those with and without TBI+LOC history. Although both groups demonstrated significant improvements in symptoms of PTSD and depression during the course of COPE, the control group evidenced significantly larger treatment improvements than the TBI+LOC-positive group. No differences were observed in the reduction of percent days of alcohol use, but with both groups demonstrating symptom improvements during the course of treatment. Together, these findings may have important implications for the diagnosis, assessment, and treatment of patients with history of TBI, and how they may differ in treatment response to those without a history of TBI.

One explanation for these findings is that group differences might be related to self-perception and expectations of recovery among patients with mTBI history. (Note that the majority of participants in the present study were positive for mTBI (93.3%) rather than moderate to severe TBI (6.7%)). For example, several studies among mTBI patients have identified that the perceptions of the consequences of the injury, rather than the actual injury and disability, may be contributing to challenges in recovery.[34–36] Whittaker and colleagues investigated a large sample of participants with mild head injuries and found that individuals that believed that their head injury would have severe, long-term consequences on their general well-being and functioning were at heightened risk of experiencing enduring post-concussional symptoms for months after the injury.[34] This perspective could suggest that participants endorsing TBI+ LOC and lingering symptoms at baseline were more concerned with the long-term consequences of their TBI and therefore were less optimistic of the benefits of COPE. This interpretation is complicated by the lack of baseline differences in PTSD and depression between the two groups, suggesting that beliefs regarding TBI history may have only influenced the perceived capacity to learn or improve, rather than perceptions of overall symptomatology.

There are several limitations that should be considered in future research. First, although the TBI screening was developed specifically for assessment TBI in a veteran population, a more thorough neurological and neuropsychological evaluation, possibly including neuroimaging, may have provided a more accurate and descriptive assessment of TBI, especially to address some of the structural differences hypothesized from other findings.[37] Second, the TBI screening was retrospective, allowing for a greater introduction of error/misremembering and does not assess/address current symptomatology. In addition, the assessment measures of PTSD and SUD were based on the DSM-IV criteria based the study being initiated prior to the publication of the DSM 5.0. And finally, the study focused on a treatment that included exposure-based practices as well as other interventions for SUD. It is curious that the pattern of reduced responsiveness to treatment among those with TBI+LOC history did not extend to SUD symptoms, possibly because of significant baseline differences in alcohol use. Given that reduced decreases in PTSD symptoms among TBI

+LOC positive patients is hypothesized to be related specifically to exposure-based elements of COPE (i.e., related to impaired fear extinction, or encoding of a fear memory more resistant to extinction), it may be useful to replicate the findings from this study in the context of a design that more specifically tests the impact of TBI history on extinction learning (e.g., a pure exposure based intervention such as Prolonged Exposure alone), as opposed to the combined intervention (Prolonged Exposure combined with Relapse Prevention treatment) used in the present study.[38–39] Unfortunately, the sample size in this study was not large enough to allow for more fine-grained analysis of TBI severity subgroups, nor analysis of rate of change over time (e.g., latent growth curve).[40]

4.1. Conclusions

The present study investigated the relation between co-occurring history of mTBI and the symptoms of PTSD, depression, and SUD during the course of an integrated, exposure-based psychotherapy for PTSD and SUD. In contrast to previous studies with smaller samples and PTSD-only interventions, participants with histories of TBI and LOC demonstrated significant, but relatively lower, reductions in PTSD and depression compared to participants without TBI+LOC history. These findings were explained by possible differences in perceived ability to respond to treatment. Although additional study is needed to further explore this hypothesis, the present findings highlight that differences in treatment response are present in co-occurring PTSD, SUD, and mTBI, suggesting that alterations may be needed in related identification, symptom monitoring, and treatment practices.

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References

1. Boyle E, Cancelliere C, Hartvigsen J, et al. Systematic review of prognosis after mild traumatic brain injury in the military: Results of the international collaboration of mild traumatic brain injury prognosis. Archiv Phys Med Rehabilitation. 2014; 95:S230–S237.
2. Hoge CW, McGurk D, Thomas JL, et al. Mild traumatic brain injury in U.S. soldiers returning from Iraq. New Eng J Med. 2008; 358:453–463. [PubMed: 18234750]
3. Marshall KR, Holland SL, Meyer KS, et al. Mild traumatic brain injury screening, diagnosis, and treatment. Militar Med. 2012; 177:67–75.
4. Ragsdale KA, Neer SM, Beidel DC, et al. Posttraumatic stress disorder in OEF/OIF veterans with and without traumatic brain injury. J Anx Disord. 2013; 27:420–426.
5. Barnes SM, Walter KH, Chard KM. Does a history of mild traumatic brain injury increase suicide risk in veterans with PTSD? Rehab Psychol. 2012; 57:18–26.
6. Capehart B, Bass D. Review: Managing posttraumatic stress disorder in combat veterans with comorbid traumatic brain injury. J Rehab Res Dev. 2012; 49:789–812.
7. Carlson KF, Nelson D, Orazem RJ, et al. Psychiatric diagnoses among Iraq and Afghanistan war veterans screened for deployment-related traumatic brain injury. J Traumat Stres. 2010; 23:17–24.
8. Corrigan JD, Cole TB. Substance use disorders and clinical management of traumatic brain injury and posttraumatic stress disorder. JAMA. 2008; 300:720–721. [PubMed: 18698070]
9. Gros DF, Korte KJ, Horner MD, et al. Co-occurring traumatic brain injury, PTSD symptoms, and alcohol use in veterans. J Psychopathol Behav Asses. 2016; 38:266–273.

10. Parry-Jones BL, Vaughan FL, Cox WM. Traumatic brain injury and substance misuse: A systematic review of prevalence and outcomes research (1994–2004). Neuropsycholog Rehab. 2006; 16:537–560.
11. Adams RS, Corrigan JD, Larson MJ. Alcohol use after combat-acquired traumatic brain injury: What we know and don't know. J Soc Work Pract Addictions. 2012; 12:28–51.
12. Brady KT, Tuerk P, Back SE, et al. Combat posttraumatic stress disorder, substance use disorders, and traumatic brain injury. J Addict Med. 2009; 3:179–188. [PubMed: 21769015]
13. Rohling ML, Binder LM, Demakis GJ, et al. A meta-analysis of neuropsychological outcome after mild traumatic brain injury: Re-analyses and reconsiderations of Binder et al.(1997), Frencham et al.(2005), and Pertab et al.(2009). Clin Neuropsychol. 2011; 25:608–623. [PubMed: 21512956]
14. Karr JE, Areshenkoff CN, Duggan EC, et al. Blast-related mild traumatic brain injury: a Bayesian random-effects meta-analysis on the cognitive outcomes of concussion among military personnel. Neuropsychol rev. 2014; 24:428–444. [PubMed: 25253505]
15. Bryant RA, Hopwood S. Commentary on "trauma to the psyche and soma". Cog Behav Pract. 2006; 13:17–23.
16. Elder GA, Mitsis EM, Ahlers ST, et al. Blast-induced mild traumatic brain injury. Psychiat Clin North Amer. 2010; 33:757–781.
17. Shu I, Onton JA, O'Connell RM, et al. Combat veterans with comorbid PTSD and mild TBI exhibit a greater inhibitory processing ERP from the dorsal anterior cingulate cortex. Psychiat Res Neuroimag. 2014; 224:58–66.
18. Chard KM, Schumm JA, McIlvian SM, Bailey GW, Parkinson RB. Exploring the efficacy of a residential treatment program incorporating Cognitive Processing Therapy-Cognitive for veterans with PTSD and traumatic brain injury. J Traum Stres. 2011; 24:347–351.
19. Sripada RK, Rauch SAM, Tuerk PW, Emith E, Defever AM, Mayer RA, Messina M, Venners M. Mild traumatic brain injury and treatment response in Prolonged Exposure for PTSD. J Traum Stres. 2013; 26:369–375.
20. Wolf GK, Kretzmer T, Crawford E, Thors C, Wagner HR, Strom TQ, Eftekhari A, Klenk M, Hayward L, Vanderploeg RD. Prolonged Exposure Therapy with veterans and active duty personnel diagnosed with PTSD and traumatic brain injury. J Traum Stres. 2015; 28:339–347.
21. Davis JJ, Walter KH, Chard KM, et al. Treatment adherence in cognitive processing therapy for combat-related PTSD with history of mild TBI. Rehab Psychol. 2013; 58:36–42.
22. Corrigan, J. Substance abuse. In: High, W.Sander, A.Struchen, M., Hart, K., editors. Rehabilitation for Traumatic Brain Injury. Oxford University Press; New York: 2005. p. 133-155.
23. Graham DP, Cardon AL. An update on substance use and treatment following traumatic brain injury. Ann New York Acad Sci. 2008; 1141:148–162. [PubMed: 18991956]
24. Bogner J, Corrigan JD. Interventions for substance misuse following TBI: A systematic review. Brain Impairment. 2013; 14:77–91.
25. Corrigan JD, Deutschle JJ Jr. The presence and impact of traumatic brain injury among clients in treatment for co-occurring mental illness and substance abuse. Brain Injur. 2008; 22:223–231.
26. Back, SE., Foa, EB., Killeen, TK., et al. Concurrent treatment of PTSD and substance use disorders using prolonged exposure (COPE): Therapist guide. New York, NY: Oxford Press; 2014.
27. Back SE, Killeen T, Foa EB, et al. Use of prolonged exposure to treat PTSD in an Iraq veteran with comorbid alcohol dependence. Am J Psychiat. 2012; 169:688–691. [PubMed: 22760188]
28. Blake DD, Weathers FW, Nagy LM, et al. The development of a clinician-administered PTSD scale. J Traum Stres. 1995; 8:75–90.
29. Sheehan DV, Lecrubier Y, Sheehan KH, et al. The Mini-International Neuropsychiatric Interview (MINI): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J Clin Psychiat. 1998; 59:22–33.
30. McAllister T, Temkin N, Dikmen S, et al. The structured assessment for evaluation of TBI (SAFE-TBI): A new instrument for assessing previous exposure to TBI. Brain Injur. 2016; 30:714–715.
31. Weathers, FW., Litz, BT., Herman, DS., et al. Annual meeting of the International Society for Traumatic Stress Studies. San Antonio, TX: 1993. The PTSD Checklist: reliability, validity and diagnostic utility.

32. Beck, A., Steer, R., Brown, G. Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation; 1996.
33. Sobell, LC., Sobell, MB. Timeline follow-back: a technique for assessing self-reported alcohol consumption. In: Litten, RZ., Allen, JP., editors. Measuring alcohol consumption: psychosocial and biochemical methods. Totowa, NJ: Humana Press; 1992. p. 41-73.
34. Whittaker R, Kemp S, House A. Illness perceptions and outcome in mild head injury: a longitudinal study. J Neurol Neurosurg Psychiat. 2007; 78:644–646. [PubMed: 17507448]
35. Ryan PB, Lee-Wilk T, Kok BC, Wilk JE. Interdisciplinary rehabilitation of mild TBI and PTSD: A case report. Brain Injur. 2011; 25:1019–1025.
36. Roth RS, Spencer RJ. Iatrogenic risk in the management of mild traumatic brain injury among combat veterans: a case illustration. J Palliative Care Med. 2013; 1:105.
37. Depue BE, Olson-Madden JH, Smolker HR, Rajamani M, Brenner LA, Banich MT. Reduced amygdala volume is associated with deficits in inhibitory control: A voxel- and surface-based morphometric analysis of comorbid PTSD/mild TBI. BioMed Res Inter. 2014:691505.
38. Foa, EB., Hembree, EA., Rothbaum, BO. Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences. Oxford: Oxford University Press; 2007.
39. Gros DF. Development and initial evaluation of Transdiagnostic Behavior Therapy (TBT) for veterans with affective disorders. Psychiat Res. 2014; 220:275–282.
40. Maas CJM, Hox JJ. Sufficient sample sizes for multilevel modeling. Methodol. 2005; 1:86–92.

Table 1

Baseline Demographics and Treatment Retention among Veterans with and without TBI history (N=51)

Variable	Control (*n* = 21)	TBI+LOC positive (*n* = 30)	χ^2 / *F*	*p*
Gender	85.7% (male)	96.7% (male)	2.1	.152
Race	61.9% (white)	73.3% (white)	1.9	.396
Relationship	19.0% (single); 38.1% (married)	33.3% (single); 16.7% (married)	4.5	.214
Employment	55.0% (employed)	35.7% (employed)	1.8	.184
OEF/OIF/OND	70.0% (OEF/OIF/OND)	66.7% (OEF/OIF/OND)	0.1	.804
Index Trauma	90.0% (military)	83.3% (military)	0.4	.506
VBA Disability	81.0% (disabled)	86.7% (disabled)	0.3	.581
Age	38.7 (10.4)	40.7 (11.1)	0.5	.505
Years of Education	14.7 (2.1)	13.6 (2.1)	3.0	.088
Completed Sessions	10.0 (3.7)	8.9 (3.5)	1.1	.292
Treatment Completion	81.0% (complete)	63.3% (complete)	1.9	.174

Note. Percentages are presented in the TBI+LOC negative and positive columns. χ^2s are presented in χ^2 / *F* in the first six rows as well as the last row. *F*s are presented in χ^2 / *F* in the seventh, eighth, and ninth rows with means (standard deviations) are presented in the TBI+LOC negative and positive columns. TBI = Traumatic Brain Injury. LOC = Loss of consciousness; VBA = Veterans Benefits Administration; OEF/OIF/OND = Operations Enduring/Iraqi Freedom/New Dawn; Treatment Completion = Completing at least 8 of 12 therapy sessions and at least 3 imaginal exposures.

Table 2

Effectiveness of COPE for PTSD and SUD symptoms among Veterans with and without mTBI (N=51)

	Control (*n* = 21) Within Group Outcome				mTBI+LOC positive (*n* = 30) Within Group Outcome				Between Groups	
Scale	**Baseline**	**Last Session**	***t***	***d***	**Baseline**	**Last Session**	***t***	***d***	***F***	η_p^2
PCL-M	61.0 (11.2)	37.4 (17.4)	6.2***	1.61	64.3 (10.4)	50.6 (20.2)	4.2***	0.85	4.8*	.250
BDI	29.2 (13.3)	11.6 (10.7)	5.6***	1.46	29.9 (11.9)	21.2 (14.5)	3.6**	0.66	7.4**	.133
TLFB-Alcohol	61.6 (27.2)	27.4 (27.1)	5.9***	1.26	32.4 (35.1)	18.2 (31.5)	2.0	0.43	0.3	.001

Note. PCL-M = PTSD Checklist – Military; BDI = Beck Depression Inventory – II; TLFB-Alcohol = Timeline Followback Alcohol Percent Days Using over the past week.

*** $p < .001$;

** $p < .01$;

* $p < .05$.