Self-Reported Sleep Disturbance Mediates the Relationship Between PTSD and Cognitive Outcome in Blast-Exposed OEF/OIF Veterans

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Objectives: To examine the contribution of sleep disturbance to cognitive performance following blast exposure. **Design:** Correlational research evaluating self-reported sleep disturbance as a mediator of the association between the primary blast-related comorbidities of mild traumatic brain injury (mTBI) and posttraumatic stress disorder and cognitive outcome. **Participants:** One hundred sixty Operation Enduring Freedom/Operation Iraqi Freedom Veterans with a history of blast exposure assigned to 1 of 3 groups (no TBI, mTBI without loss of consciousness, and mTBI with loss of consciousness). **Main Outcome Measures:** Neuropsychological measures and self-report of sleep disturbance. **Results:** Increased posttraumatic stress disorder symptomatology was associated with worse performance in multiple cognitive domains. This association was mediated in part by self-reported sleep disturbance. Traumatic brain injury with loss of consciousness was associated with lower manual dexterity, but this association was not mediated by sleep disturbance. **Conclusions:** Our results highlight the importance of sleep disturbance as a factor contributing to cognitive outcome in individuals with posttraumatic stress disorder symptoms. They point to the importance of considering sleep problems in the diagnosis and treatment of cognitive deficits in veterans with blast exposure. **Key words:** *blast injuries*, *cognition*, *mediation*, *posttraumatic stress disorder*, *sleep*, *traumatic brain injury*

THE MILITARY CONFLICTS in Iraq and Afghanistan as well as recent terrorist events such as the Boston Marathon bombing have focused attention on the health outcomes associated with blast exposure. Sleep disturbances are prominent among deployed military personnel[1–3] and are one of the most common complaints of Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF) veterans, with findings suggesting that three-quarters or more of treatment-seeking individuals complain of sleep difficulty.[4–6] These high prevalence rates can be understood in light of the fact that the signature injuries of these conflicts—mild traumatic brain injury (mTBI) and posttraumatic stress disorder (PTSD)—each can be associated with sleep disturbances.[7] Complaints of poor sleep are common in the chronic stage of mTBI,[8–11] and objective sleep recordings in patients with traumatic brain injury (TBI), regardless of severity, have documented a variety of long-term sleep abnormalities including sleep apnea, hypersomnia, insomnia, and narcolepsy.[12–15] Sleep disturbance is considered a core feature of PTSD, with insomnia[16,17] and nightmares[16,18] as key components. Although polysomnography studies have not consistently documented sleep abnormalities in PTSD,[19] recent meta-analyses confirm the presence of reduced sleep efficiency and heightened awakenings in PTSD.[20,21] In light of these findings, the presence and impact of sleep abnormalities in individuals with blast injury deserves further attention.

Sleep disruption affects a range of health outcomes, including neurocognitive functioning.[22–25] Individuals with sleep disorders demonstrate deficits on neuropsychological tasks measuring information processing

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The research was supported by Merit Review grants awarded by the Clinical Science Research and Development Service and Rehabilitation Research and Development Service of the Department of Veterans Affairs to Dr Verfaellie. Drs Verfaellie and Spiro are supported by the Clinical Science Research and Development Service of the Department of Veterans Affairs. The contents do not represent the views of the U.S. Department of Veterans Affairs or the US Government.

The authors declare no conflicts of interest.

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DOI: 10.1097/HTR.0000000000000197

speed, attention, episodic memory, and working memory.[23,26,27] Evidence suggests that sleep problems contribute to the cognitive difficulties observed in both TBI[28–30] and PTSD.[31,32] Moreover, improvement in sleep can lead to improvement in cognition in both disorders.[33,34]

The above findings motivate the examination of the contribution of sleep disturbances to the cognitive difficulties experienced by OEF/OIF veterans exposed to blast. Previous studies examining associations of chronic mTBI and PTSD with neuropsychological outcomes have highlighted PTSD symptoms as a primary contributor to cognitive impairment.[35–38] Although there is some evidence that sleep duration may moderate the association between PTSD and cognitive function,[32] no study has directly assessed the overlap among PTSD, sleep disturbance, and cognitive outcomes, while taking into account mTBI-comorbidity. Here, we evaluate whether self-reported sleep disturbance mediates the relationship between PTSD and cognitive outcomes. Such a finding not only would help elucidate the nature of PTSD-associated cognitive difficulties but also would have important implications for treatment.

METHODS

Participants

The sample consisted of 160 OEF/OIF veterans who reported being within 100 m of a blast during deployment and were at least 6 months post–blast exposure. They were recruited through the VA Boston Polytrauma Network and through flyers and outreach events in the community. Using the definition of mTBI of the American Congress of Rehabilitation Medicine,[39] participants were assigned to 1 of 3 groups: no TBI, mTBI without loss of consciousness (LOC), and mTBI with LOC. Fifty-five participants comprised the no TBI group, 63 the mTBI without LOC group (mTBI-LOC), and 42 the mTBI with LOC group (mTBI+LOC). See Table 1 for participant demographics.

Participants were excluded from the study if they suffered a TBI of greater than mild severity ($n = 2$), scored below 45 on the retention trial of the Test of Memory Malingering[40] demonstrating questionable effort ($n = 10$), or if they reported a history of predeployment TBI of greater than mild severity ($n = 3$). Results of neuropsychological assessment for a subset of the sample have been reported elsewhere.[38]

Measures

mTBI interview

Diagnosis of TBI was based on an extensive clinical interview that consisted of 4 parts: (1) determination of the most severe blast exposure based on the participant's description of the event and associated symptoms; (2) in-depth description of the index event, including description of events preceding the blast, the blast event itself, and events subsequent to the blast to determine presence and duration of alteration of consciousness (ie, disorientation, posttraumatic amnesia, LOC); (3) probing of the presence immediately following the blast of neurological symptoms consistent with TBI; and (4) query regarding medical examination or reports by a witness that could corroborate the presence of TBI. In all but 6 instances, presence and duration of LOC were based on information provided by a medic or combat peers who witnessed the event and often were the first to assess responsiveness.

Interviews were transcribed and evaluated by 2 of the authors, who then sought consensus in a joint session as to whether a minimal biomechanical threshold for TBI had plausibly been met, and any reported disorientation was due to TBI rather than situational chaos or confusion. Traumatic brain injury group status was represented by 2 dummy variables that indicate membership in the mTBI-LOC group, and membership in the mTBI+LOC group, respectively.

Posttraumatic stress disorder

The Clinician Administered PTSD Scale for *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (*DSM-IV*)[41] was used to assess PTSD. This structured clinical interview is the criterion standard for assessing PTSD and measuring the severity of PTSD symptoms and has excellent reliability and validity.[42] Symptoms were probed associated with any potential index trauma (not only the blast event). Continuous Clinician Administered PTSD Scale scores were used as a measure of severity of PTSD symptoms. Scores were divided by 10 to facilitate interpretation.

Sleep disturbance

The Pittsburgh Sleep Quality Index (PSQI)[43] was used as a global measure of sleep. It consists of 18 items that probe for different aspects of sleep over the past month: sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Scoring is based on a scale from 0 to 3, with higher scores reflecting worse sleep. The measure is psychometrically reliable, as indicated by high internal consistency and test-retest reliability.[43] Scores on each item were summed to obtain a global PSQI score, which can range from 0 to 21. Scores greater than 5 are indicative of poor sleep,[43] and in an outpatient TBI sample, scores greater than 8 have been shown to identify clinically significant insomnia symptoms with excellent sensitivity and specificity.[44]

TABLE 1 *Descriptive statistics*[a]

M (SD)/*n* (%)	Overall (*N* = 160)	No TBI (*n* = 55)	mTBI-LOC (*n* = 63)	mTBI+LOC (*n* = 42)	$F_{2,157}$ / χ^2_2	*P*
Age	30.02 (7.43)	30.25 (6.74)	30.57 (8.39)	28.88 (6.80)	0.69	.50
Education	13.43 (1.88)	13.65 (1.93)	13.48 (1.82)	13.10 (1.90)	1.05	.35
Male	154 (96%)	52 (95%)	61 (96%)	41 (98%)	0.72	.70
CAPS total score	56.82 (25.97)	49.40[b,c] (26.59)	59.32[b] (24.75)	62.79[c] (25.24)	3.77	.03
Sleep disturbance	11.25 (4.34)	9.84[b,c] (4.14)	11.62[b] (4.20)	12.54[c] (4.38)	5.26	.01
PSQI: 5-8 (poor sleep)	29 (18%)	15 (27%)	11 (17%)	3 (7%)	6.53	.04
PSQI: >8 (clinically significant symptoms)	119 (74%)	35 (64%)	48 (76%)	36 (86%)	6.27	.04
Sleep medications (1 = Y, 0 = N)	30 (19%)	9 (16%)	13 (21%)	8 (19%)	0.35	.84
Cognitive factors *z* scores:						
Processing speed	0.01 (0.93)	0.22[c] (0.94)	0.02 (0.88)	−0.26[c] (0.94)	3.32	.04
Verbal memory	0.04 (0.95)	0.09 (0.87)	0.08 (1.02)	−0.08 (0.95)	0.46	.63
Visual memory	0.03 (0.94)	0.18 (0.78)	−0.01 (0.98)	−0.12 (1.06)	1.24	.29
Motor speed	0.01 (0.88)	0.04 (0.89)	0.04 (0.72)	−0.05 (1.07)	0.15	.86
Manual dexterity	−0.01 (0.90)	0.23[c,d] (0.77)	−0.06[d] (0.92)	−0.24[c] (0.96)	3.60	.03
Cognitive control	0.002 (0.85)	0.07 (0.88)	0.04 (0.83)	−0.14 (0.84)	0.83	.44
Months since blast exposure	49.33 (28.99)	53.56 (33.37)	43.59 (26.85)	52.40 (24.87)	2.09	.13

Abbreviations: CAPS, Clinician Administered PTSD Scale; LOC, loss of consciousness; mTBI, mild traumatic brain injury; N, no; PSQI, Pittsburgh Sleep Quality Index; TBI, traumatic brain injury; Y, yes.

[a]For descriptive purposes, CAPS scores are shown in the original metrics (ie, not divided by 10) in this table.

[b]$P < .05$.

[c]$P \leq .01$.

[d]$P < .10$.

Internal consistency in the current sample as measured by Cronbach α was 0.72.

Neurocognitive function

Participants were given a battery of neuropsychological tests selected for their sensitivity to mTBI, including tests of processing speed and executive functioning, verbal and visual memory, and motor ability.[38] Processing speed was measured using Wechsler Adult Intelligence Test - III (WAIS-III) Digit Symbol Coding,[45] requiring participants to write symbols corresponding to digits according to a key presented in front of them; the number sequencing, letter sequencing, and motor speed subtests of the D-KEFS Trail Making Test,[46] which require connecting in sequence letters, numbers, or circles following a dotted line; and the word and color naming subtests of the DKEFS Color-Word Interference Test.[46] Working memory was assessed using WAIS-III Digit Span Backwards,[45] which requires recall of digits in reverse; and Auditory Consonant Trigrams,[47] which requires recall of 3 consonants after variable delays of counting backward by 3 from a specified number. Mental flexibility was assessed using the D-KEFS Verbal Fluency Test,[46] which requires generating words to a specified letter; the number-letter switching subtest of the D-KEFS Trail Making Test,[46] which requires alternating between letters and numbers to connect them in order; and the inhibition subtest of the D-KEFS Color-Word Interference Test,[46] which requires inhibiting reading of color words to report the color in which the words are presented. Memory was assessed using the California Verbal Learning Test,[48] which requires learning of 16 words across 5 trials, as well as immediate and delayed retention; and the Brief Visual Memory Test-Revised,[49] which requires participants to learn and reproduce 6 figures across 3 trials, as well as immediate and delayed retention. Motor ability was assessed using the Finger Tapping Test,[50] which requires tapping a key as quickly as possible; and the Grooved Pegboard Test,[51] a dexterity test that requires manipulation of small pegs to be put into grooves. These tests are considered to have good reliability and validity.

Data on Digit Span and Digit Symbol-Coding were not available for 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.

Covariates

We considered age, education, and use of sleep medications as covariates. Education was indicated by years

*There were 4 other instances in which a respondent had missing or invalid scores on a test, but we did not impute in these cases.

of schooling. Use of sleep medications was coded as a binary variable (1 = Yes, 0 = No) to indicate whether participants were currently using 1 or more sleep-related medications, including trazodone, zolpidem, and a variety of benzodiazepines.

Procedure

Study procedures took place within a research context and were unrelated to diagnostic or treatment purposes. A licensed clinical neuropsychologist administered all clinical interviews, questionnaires, and neuropsychological assessments. Study procedures typically took 4 to 5 hours, which were completed in 1 or 2 sessions according to participant preference. Hospital institutional review board approval and written informed consent from participants were obtained.

Data analysis

Descriptive statistics were compared as a function of TBI group status using 1-way analysis of variance for continuous variables and χ^2 test for categorical variables.

To identify cognitive domains, we performed a factor analysis with oblique (promax) rotation on the cognitive measures. Factor scores were estimated via the regression method as *z* scores and served as dependent variables in subsequent analyses (see Appendix).

To evaluate sleep disturbance as a mediator of the association between the independent variables of PTSD severity and TBI status and the dependent variable of cognitive functioning, we conducted path analysis using Mplus version 7.1.[52] As illustrated by Figure 1, the saturated model included the regression of 6 cognitive factors on sleep disturbance, PTSD severity, TBI group status, and covariates, and the regression of sleep disturbance on PTSD severity and TBI group status and covariates.

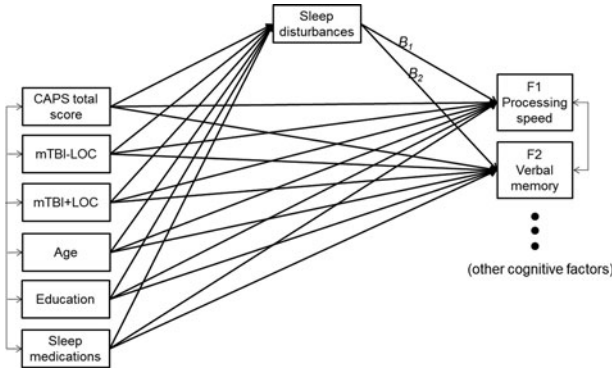

Figure 1. Conceptual representation of the saturated mediation model. Single-headed arrows represent regression paths. Double-headed arrows represent correlations. Only 2 of 6 cognitive factors (F1 and F2) are shown for simplicity. CAPS indicates Clinician Administered PTSD Scale; LOC, loss of consciousness; mTBI, mild traumatic brain injury.

For model trimming, we first considered for removal nonsignificant paths (ie, $P > .10$) emitting from the covariates. Next, we considered nonsignificant regression paths that make up the indirect effects. Paths were eliminated only if their removal did not produce a significant deterioration in model fit. Direct paths from PTSD severity and TBI group status to the cognitive factors were retained regardless of effect size because their elimination could result in biased estimates of indirect effects.[53] We also evaluated whether the indirect effects from PTSD severity and TBI group status via sleep disturbance were equivalent across the 6 domains of cognitive functioning. This was accomplished by testing equality constraints applied to the regression paths from sleep disturbance to the 6 cognitive factors. For 2 indirect paths predicting 2 cognitive factors (eg, PTSD→sleep disturbance→factor 1, PTSD→sleep disturbance→factor 2), the indirect effects were considered identical if the path for the regression of each cognitive factor on sleep disturbance could be equated without a decrement in model fit. Beginning with the 6 regression paths of the cognitive factors on sleep disturbance, we equated 2 paths that were closest in magnitude and compared the model fit. This step was repeated across all 6 regression paths to yield a final, best-fitting model to the data.

Models were evaluated with the comparative fit index,[54,55] Tucker-Lewis fit index,[56] root mean square error of approximation,[55,56] standardized root mean square residual,[56] and Bayesian Information Criterion.[57] Nested models were also evaluated with the χ^2 difference test. In all models, we used the bias-corrected bootstrapping method on the basis of 5000 bootstrap draws to estimate the 95% confidence intervals for indirect effects. Indirect effects are considered statistically significant if the 95% bias-corrected bootstrap confidence interval does not contain 0. Model estimation was based on full information maximum likelihood.

RESULTS

Descriptive statistics by TBI group status are shown in Table 1. The groups did not differ on age, education, gender, and functioning in the domains of verbal memory, visual memory, motor speed, and cognitive control. Group differences were significant for PTSD symptom severity, sleep disturbance, processing speed, and manual dexterity. Post hoc contrast analyses were conducted to examine pairwise differences in these variables by TBI group status. Results indicated that compared with participants without TBI, those who suffered mTBI without LOC had more severe PTSD symptoms, greater sleep disturbance, and a trend for worse manual

TABLE 2 *Summary of model trimming steps and fit indices*[a]

Model description	χ^2 (*df*)	*P*	$\Delta\chi^2/\Delta$par	BIC	ΔBIC	RMSEA (90% CI)	SRMR	CFI	TLI
(a) Saturated model	0 (0)	—	—	6533	—	—	0.000	1.000	1.000
(b) Removed 16 nonsignificant paths emitting from covariates	17.07 (16)	.38	+17.07/–16	6469	−64	0.020 (0.000-0.077)	0.026	0.997	0.988
(c) Set $B_5 = 0$	18.19 (17)	.38	+1.12/–1	6465	−4	0.021 (0.000-0.076)	0.026	0.997	0.988
(d) Set mTBI-LOC → sleep = 0, mTBI+ LOC → sleep = 0	22.20 (19)	.27	+4.01/–2	6459	−6	0.032 (0.000-0.079)	0.030	0.996	0.989
(e) Set $B_4 = B_6$	22.22 (20)	.33	+0.02/–1	6454	−5	0.026 (0.000-0.075)	0.030	0.994	0.981
(f) Set $B_2 = B_3$	22.29 (21)	.38	+0.07/–1	6449	−5	0.020 (0.000-0.071)	0.030	0.996	0.989
(g) Set $B_1 = B_4 = B_6$	22.45 (22)	.43	+0.16/–1	6444	−5	0.011 (0.000-0.067)	0.030	0.999	0.996
(h) Set $B_1 = B_2 = B_3 = B_4 = B_6$	23.74 (23)	.42	+1.30/–1	6440	−4	0.014 (0.000-0.067)	0.031	0.998	0.994

Abbreviations: BIC, Bayesian Information Criterion; CFI, comparative fit index; CI, confidence interval; LOC, loss of consciousness; mTBI, mild traumatic brain injury; RMSEA, root mean square error of approximation; SRMR, standardized root mean square residual; TLI, Tucker-Lewis fit index.

[a]par indicates estimated parameters. B_1 to B_6 represent the coefficients for the regression paths from sleep disturbances to cognitive factors 1 (processing speed), 2 (verbal memory), 3 (visual memory), 4 (motor speed), 5 (manual dexterity), and 6 (cognitive control), respectively. $\Delta\chi^2$ (Δpar) and ΔBIC indicate differences compared with the prior model.

dexterity; those who suffered mTBI with LOC had more severe PTSD symptoms, greater sleep disturbance, slower processing speed, and worse manual dexterity. It can be seen that the proportion of participants with PSQI greater than 8, which is indicative of clinically significant insomnia symptoms, was positively associated with TBI severity.

Path analysis began with a saturated model* (see Table 2, model a). Eliminating 16 nonsignificant paths emitting from age, education, and use of sleep medications did not produce a decrement in model fit (see Table 2, model b). The regression path from sleep disturbance to the manual dexterity factor was nonsignificant, $B_5 = -0.02$, $SE_5 = 0.02$ (subscript "5" corresponds to the cognitive factor, see Table 2 footnote). Eliminating this path did not worsen model fit (see Table 2, model c). The regression path from mTBI-LOC to sleep disturbance was also nonsignificant, $B = 0.70$, $SE = 0.58$. We subsequently eliminated the paths from TBI group status to sleep disturbance, which did not worsen model fit (see Table 2, model d). In this model, all remaining regression paths from PTSD severity to sleep disturbance, and from sleep disturbance to the 5 remaining cognitive factors were statistically significant at $P < .10$. The effects of sleep disturbance on the cognitive factors were as follows: $B_1 = -0.04$, $SE_1 = 0.02$ for processing speed; $B_2 = -0.06$, $SE_2 = 0.02$ for verbal memory; $B_3 = -0.06$, $SE_3 = 0.02$ for visual memory; $B_4 = -0.04$, $SE_4 = 0.02$ for motor speed; and $B_6 = -0.04$, $SE_6 = 0.02$ for cognitive control.

Next, we compared a series of models to evaluate the equality of indirect effects across the cognitive factors. This was accomplished by testing equality constraints among the paths emitting from sleep disturbance to various cognitive factors. First, we equated the paths from sleep disturbance to motor speed (B_4) and cognitive control (B_6). The resulting model did not suffer a loss in fit (see Table 2, model e). We further equated the paths from sleep disturbance to verbal memory (B_2) and visual memory (B_3). Model fit was preserved (see Table 2, model f). In the next step, we equated the paths from sleep disturbance to processing speed (B_1), motor speed (B_4), and cognitive control (B_6), which did not result in deterioration in model fit (see Table 2, model g). We proceeded to test a model wherein the paths from sleep disturbance to all cognitive factors (except manual dexterity, B_5) were equated, that is, $B_1 = B_2 = B_3 = B_4 = B_6$. Model fit was again preserved (see Table 2, model h). This was the most parsimonious model tested, and we selected it to be our final, best-fitting model.

Table 3 displays parameter estimates for the best-fitting model and summarizes the mediation results. Results for the regression of sleep disturbance and the cognitive factors on covariates are shown at the top portion of Table 3; mediation results are shown at the bottom. The indirect effects of PTSD suggest that greater sleep disturbance mediated the association between higher

*We reran the model excluding sleep items from the CAPS score as well as sleep medications from the PSQI score. The same pattern of findings was observed as in the initial model with the exception that the regression of PSQI scores on sleep medications was no longer significant. Given that we removed the sleep medication item from the PSQI, the diminished association between the PSQI total score and the sleep medication variable was expected.

TABLE 3 *Results from best-fitting path model*[a]

(a) Parameter estimates for regression of sleep disturbance on covariates and of cognitive factors on sleep disturbance and covariates

	Sleep disturbance	F1: Processing speed	F2: Verbal memory	F3: Visual memory	F4: Motor speed	F5: Manual dexterity	F6: Cognitive control
Sleep disturbance	—	**−0.05 (−0.07, −0.02)**	**−0.05 (−0.07, −0.02)**	**−0.05 (−0.07, −0.02)**	**−0.05 (−0.07, −0.02)**	=0	**−0.05 (−0.07, −0.02)**
Age	=0	=0	**0.03 (0.01, 0.05)**	**0.02 (0.01, 0.04)**	=0	=0	=0
Education	=0	**0.07 (0.004, 0.13)**	=0	=0	=0	=0	**0.08 (0.02, 0.15)**
Sleep medications	**1.68 (0.61, 2.78)**	=0	=0	=0	=0	=0	=0

(b) Direct and indirect effects of PTSD and TBI group status on cognitive outcomes

	F1: Processing Speed	F2: Verbal memory	F3: Visual memory	F4: Motor speed	F5: Manual dexterity	F6: Cognitive control
CAPS score						
Indirect	**−0.05 (−0.08, −0.02)**	**−0.05 (−0.08, −0.02)**	**−0.05 (−0.08, −0.02)**	**−0.05 (−0.08, −0.02)**	=0	**−0.05 (−0.08, −0.02)**
Direct	−0.02 (−0.08, 0.05)	−0.02 (−0.09, 0.05)	−0.01 (−0.07, 0.05)	−0.01 (−0.07, 0.04)	−0.03 (−0.08, 0.03)	0.002 (−0.06, 0.06)
Total	**−0.07 (−0.12, −0.01)**	**−0.07 (−0.13, −0.01)**	**−0.06 (−0.11, −0.01)**	**−0.06 (−0.12, −0.01)**	−0.03 (−0.08, 0.03)	−0.05 (−0.10, 0.01)
mTBI-LOC						
Direct only	−0.09 (−0.41, 0.25)	0.08 (−0.25, 0.44)	−0.10 (−0.40, 0.22)	0.10 (−0.19, 0.40)	−0.26 (−0.57, 0.06)	0.07 (−0.23, 0.37)
mTBI+LOC						
Direct only	−0.30 (−0.64, 0.07)	0.01 (−0.35, 0.38)	−0.13 (−0.50, 0.23)	0.06 (−0.34, 0.45)	**−0.44 (−0.81, −0.08)**	−0.04 (−0.38, 0.32)

Abbreviations: CAPS, Clinician Administered PTSD Scale; LOC, loss of consciousness; mTBI, mild traumatic brain injury; PTSD, posttraumatic stress disorder; TBI, traumatic brain injury.

[a]Results correspond to model (h) in Table 2. Unstandardized regression coefficients (95% bootstrap confidence intervals) are shown. Values in boldface indicate parameter estimates for which the 95% confidence interval does not contain 0. CAPS scores were divided by 10 to facilitate interpretation. Regression of sleep disturbance on CAPS score yielded a parameter estimate of $B = 1.064$ (0.87, 1.24), $P < .05$. Regression paths from TBI dummy variables to sleep disturbance were set to 0.

PTSD severity and worse performance in the domains of processing speed, verbal memory, visual memory, motor speed, and cognitive control, adjusting for the association between mTBI and cognitive performance. For each cognitive factor, a large proportion of the total effect of PTSD was due to the indirect effect of sleep disturbance: 73.8% for processing speed, 72.7% for verbal memory, 82.8% for visual memory, 78.7% for motor speed, and 96% for cognitive control. After adjusting for PTSD symptom severity, mTBI without LOC was not associated with any type of cognitive functioning; mTBI with LOC was linked only to worse manual dexterity, but this relation could not be explained by sleep disturbance. The proportion of explained variance accounted for by the final model was 48.2% for sleep disturbance, 11.8% for processing speed, 9.3% for verbal memory, 8.2% for visual memory, 6.3% for motor speed, 4.9% for manual dexterity, and 9.7% for cognitive control.

DISCUSSION

In a large cohort of OEF/OIF veterans exposed to blast, we examined the relations among the primary deployment-related comorbidities of mTBI and PTSD, sleep disturbance, and neuropsychological performance. There were 3 main findings. First, self-reported sleep disturbance was positively associated with PTSD symptom severity but not with chronic mTBI. Second, sleep disturbance was related to worse cognitive functioning across multiple domains. Third, a mediation analysis revealed that PTSD symptom severity was associated with poorer cognitive performance in part through its relation to sleep disturbance.

Our findings converge with others in showing the high prevalence of self-reported sleep problems in the OEF/OIF veteran population.[2,5,58] These disturbances were linked to PTSD rather than TBI, consistent with other reports that have highlighted the contribution of mental health symptoms to sleep quality following chronic mild TBI.[5,59] Poor sleep in PTSD is likely multidetermined, as nightmares, insomnia, and sleep-disordered breathing all are prevalent.[19,21,60] An important mechanism underlying sleep disturbance in PTSD is thought to be hyperactivity of the noradrenergic system.[61] In addition, maladaptive behavioral patterns, possibly compounded by irregular sleep schedules developed during deployment and the perceived need to be constantly vigilant,[58] could further exacerbate sleep disturbance.

While previous research has suggested that reports of poor sleep in individuals with PTSD contribute to negative physical[62,63] and mental health outcomes,[21,61] our study demonstrates for the first time that sleep disturbance is also associated with reduced cognitive performance. The effect of sleep disturbance on neuropsychological test performance was wide-ranging, as it accounted for significant variance in all cognitive factors with the exception of manual dexterity. Furthermore, its effect was equivalent across cognitive domains, consistent with the notion that sleep disruption affects cognition by virtue of a global impact on alertness and attention, which are important for performance across cognitive domains.[64] However, an extensive literature also suggests specific mechanisms, beyond a general role in attention, by which sleep contributes to processes involved in memory[65–67] and cognitive control.[68–70] Future studies using cognitive tasks designed to specifically assess component processes will be necessary to disentangle global from process-specific effects of sleep disturbance in this population.

The lack of effect of sleep disturbance on manual dexterity is in line with evidence that performance on such tasks is resistant to the effect of reduced sleep.[27,71] These tasks place minimal cognitive demands, and it is possible that the motoric demands of the task as well as the built-in feedback regarding one's performance help maintain alertness. On the contrary, manual dexterity was affected by mTBI, particularly when associated with LOC. Impaired manual dexterity has been noted in other studies of mTBI,[72,73] including blast-related injury.[74] It is notable that the cerebellum, known to be important for motor coordination, has been shown to be particularly vulnerable to the effects of blast.[75–77] It could be argued that the clinical significance of the LOC-associated reduction in manual dexterity is limited given that scores remained largely within the normal range. However, this finding is noteworthy in light of the fact that motor skills are a strong predictor of functional outcome after TBI.[78] Moreover, it highlights the importance of assessing manual dexterity in individuals with blast-induced mTBI.

Our finding that PTSD was indirectly associated with cognitive performance through its relation to reported sleep disturbance suggests that sleep disturbance may partially account for the relationship between PTSD and cognitive outcomes. One other study[79] has examined relations among sleep, psychiatric symptoms, and cognitive function in OEF/OIF veterans. The authors also found that sleep and emotional distress make unique contributions to cognitive performance but postulated that lack of sleep could influence cognition by disrupting systems involved in emotional regulation.[64] Although this conclusion appears at odds with the findings of our mediation analysis, it is important to note that the relationship between PTSD and sleep is likely reciprocal. Just as sleep disturbance is thought to be a core symptom of PTSD,[21,80] disrupted sleep can exacerbate PTSD symptoms.[17] As such, sleep and PTSD appear intrinsically linked, and sleep disturbance may initiate a downward spiral that enhances the risk of poor cognitive performance.

In this context, the potential long-term effects of disturbed sleep also should be noted. A growing literature suggests that reduced sleep quality and duration may be risk factors for subsequent cognitive decline and dementia.[81,82] Moreover, sleep disruption has been found to contribute to the onset and accumulation of Alzheimer pathology.[83] Given the elevated risk of dementia associated with PTSD[84,85] as well as with TBI,[86–88] consideration of sleep as a mediating factor of long-term cognitive outcomes will be of critical importance.

Our study focused on the associations among PTSD, sleep, and cognition, but other mental health symptoms may also contribute to cognitive outcome. Measures of PTSD and depression were highly correlated in our sample. This likely reflects overlap in symptoms between the 2 disorders but is also consistent with the proposal that PTSD and depression in the aftermath of trauma are best conceptualized as a single, general response to traumatic stress.[89] In our previous work,[38] we evaluated PTSD and depression in separate models as predictors of cognitive outcome and found that both were significant predictors, although the model fit was better with PTSD as the predictor, hence the focus on PTSD in this study. However, additional mental health variables such as substance use should also be considered in future work.

The study was cross-sectional in nature and does not allow inferences about causality. Although we observed that sleep disturbance mediated the relationship between PTSD and cognitive performance, future studies will need to examine whether improved sleep leads to cognitive benefits. In recent years, there has been heightened awareness of the importance of targeted sleep therapies as an integral part of treating PTSD.[21] Our findings raise the possibility that sleep restoration may directly improve cognitive function, a conjecture for which there is some preliminary support.[34] To the extent that cognitive factors may influence the maintenance or resolution of PTSD,[90] improved sleep may also indirectly contribute to better affective regulation and more adaptive appraisal of the trauma and its sequelae.

A limitation of the current study is that assessment of sleep difficulties was based on self-report, and we do not know to what extent these self-report measures map onto objective measures of sleep functioning. It has been suggested that persons with PTSD may have a distorted perception of their sleep, overestimating sleep onset latency, and underestimating total sleep time,[91,92] but these discrepancies may also reflect the fact that individuals with PTSD sleep better in a sleep laboratory where physiological sleep measures are obtained, because the environment is experienced as being safe.[21] Moreover, recent findings point to the importance of the perception of one's sleep quality in its own right and its impact on cognitive performance.[93] Thus, future studies examining the role of sleep disturbance as mediator of the association between PTSD and clinical outcomes should use a multimodal sleep assessment that incorporates objective and subjective measures.

In conclusion, our study points to the role of sleep disturbance in the relationship between PTSD symptoms and cognitive performance in OEF/OIF veterans. These findings have several clinical implications. First, they highlight the importance of assessing sleep symptoms as part of any comprehensive evaluation in this cohort. Second, they should alert the clinician to the potential contribution of sleep in interpreting neuropsychological impairments. Finally, they point to the importance of treating sleep disturbances. However, a better understanding of which sleep treatments are most effective and how they may impact cognitive outcome awaits further research.

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APPENDIX *Factor Loadings and Correlations for Cognitive Measures*[a]

	F1: Processing speed	F2: Verbal memory	F3: Visual memory	F4: Motor speed	F5: Manual dexterity	F6: Cognitive control
Factor loadings						
Verbal fluency	**.53**	.04	−.05	−.11	−.06	.10
Digit symbol-coding	**.55**	−.03	.00	−.16	.25	.18
Trails motor speed	**.72**	−.08	.13	−.03	−.10	−.21
Trails letter + number sequencing	**.78**	.13	.02	.06	−.04	−.16
Trails number/letter switching	**.60**	−.03	.20	−.03	−.03	.04
Stroop word + color naming	**.52**	−.01	−.13	.27	.03	.19
Stroop inhibition	**.43**	−.05	−.10	.19	.16	.29
Consonant trigrams	.01	.08	.17	.10	−.08	**.58**
Digit span backwards	−.02	−.01	.04	−.12	−.08	**.66**
CVLT total trials 1-5	.03	**.78**	−.01	−.09	.00	.08
CVLT delayed recall	.11	**.86**	.04	.02	−.05	.01
CVLT delayed recognition	−.12	**.82**	.01	.05	.09	−.04
BVMT-R total trials 1-5	.05	.01	**.76**	.03	.09	.01
BVMT-R delayed recall	−.02	−.03	**.90**	.00	.00	.10
BVMT-R delayed recognition	.05	.09	**.44**	.00	−.04	.02
Finger tapping dominant	.00	.02	−.01	**.83**	−.01	−.08
Finger tapping nondominant	−.06	−.03	.07	**.84**	.00	.01
Grooved pegboard dominant	.01	.02	.09	.00	**.80**	−.15
Grooved pegboard nondominant	−.04	.03	−.04	.00	**.77**	.00
Factor correlations						
F1: Processing speed	—					
F2: Verbal memory	**.39**	—				
F3: Visual memory	**.45**	**.45**	—			
F4: Motor speed	**.37**	.07	**.25**	—		
F5: Manual dexterity	**.42**	**.18**	**.27**	**.38**	—	
F6: Cognitive control	**.60**	**.30**	**.40**	**.24**	.10	—

Abbreviations: BVMT-R, Brief Visual Memory Test-Revised; CVLT, California Verbal Learning Test.
[a]Values in boldface indicate standardized factor loadings > .35 and factor correlations with $P \leq .05$.