WAR, TRAUMA, AND SLEEP ACROSS THE LIFESPAN
ORIGINAL ARTICLE

# The association of polysomnographic sleep on posttraumatic stress disorder symptom clusters in trauma-exposed civilians and veterans

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## Abstract

**Study Objectives:** Self-reported sleep disturbance has been established as a risk factor and predictor for posttraumatic stress disorder (PTSD); however, less is known about the relationship between objective sleep and PTSD symptom clusters, and the specific role of hyperarousal. The present study examined the relationships between sleep continuity and architecture on PTSD symptom clusters.

**Methods:** Participants underwent two in-laboratory sleep studies to assess sleep continuity and architecture. They also completed the Clinician-Administered PTSD-IV scale and the Structured Clinical Interview for the DSM-IV to assess for PTSD diagnosis and other psychiatric disorders.

**Results:** Sleep continuity (i.e. total sleep time, sleep efficiency percent, wake after sleep onset, sleep latency) was significantly related to PTSD Cluster B (reexperiencing) symptom severity ($R^2 = .27$, $p < .001$). Sleep architecture, specifically Stage N1 sleep, was significantly associated with PTSD Cluster B ($t = 2.98$, $p = .004$), C (Avoidance; $t = 3.11$, $p = .003$), and D (Hyperarousal; $t = 3.79$, $p < .001$) symptom severity independently of Stages N2, N3, and REM sleep. REM sleep variables (i.e. REM latency, number of REM periods) significantly predicted Cluster D symptoms ($R^2 = .17$, $p = .002$).

**Conclusions:** These data provide evidence for a relationship between objective sleep and PTSD clusters, showing that processes active during Stage N1 sleep may contribute to PTSD symptomatology in civilians and veterans. Further, these data suggest that arousal mechanisms active during REM sleep may also contribute to PTSD hyperarousal symptoms.

### Statement of Significance

Few studies have examined the relationships between objectively measured sleep and posttraumatic stress disorder (PTSD) symptom clusters, precluding the understanding of how sleep continuity and architecture may maintain unique PTSD presentations. In this sample of racially diverse trauma-exposed US military veterans and civilians, we found that more time spent in stage N1 sleep was associated with greater levels of all PTSD symptom clusters, and that greater REM latency and fewer REM periods were associated with hyperarousal symptoms. These results highlight the role of arousal and sleep fragmentation in PTSD. Work is needed to delineate the unique impact that sleep fragmentation has on maintaining each PTSD symptom cluster, and to investigate whether modifying sleep architecture could reduce symptoms.

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## Introduction

Approximately 90.0% of the US population will experience traumatic stress during their lifetime, and over 8.0% of those individuals will develop posttraumatic stress disorder (PTSD) [1]. According to the Diagnostic Statistical Manual (DSM), PTSD is broadly characterized by symptoms of intrusive memories or reexperiencing, avoidance of stimuli or reminders of the trauma, negative changes to thoughts and mood associated with the trauma, and changes in arousal or physical emotional reactions [2, 3]. These symptoms can vary in their frequency and intensity.

Recurrent nightmares and insomnia are encompassed by the PTSD diagnostic criteria, within the intrusion and hyperarousal symptom clusters, respectively. Disturbed sleep, specifically insomnia symptoms, is common following a traumatic event (e.g. combat, physical and sexual assault), and has been linked to the development of PTSD [4, 5]. Further, in veterans it has been well established that disturbed sleep prior to trauma exposure may serve as a risk factor [6] and/or predictor [7–9] for PTSD.

Subjectively, disturbed sleep (i.e. insomnia, nightmares) has been associated with PTSD [5, 10]. Although, no consensus on objective sleep abnormalities in PTSD has been established, a meta-analytic report of 20 polysomnographic (PSG) studies provided evidence for lighter sleep in those with PTSD compared to controls, specifically, increased Stage N1 sleep and reduced Stage N3 sleep [11, 12]. Kobayashi et al [13] also showed elevated nocturnal autonomic nervous system (ANS) arousal and dissociation between ANS activation and objective sleep duration in PTSD participants.

Polysomnographic studies conducted in adults with PTSD have been more consistent in identifying alterations related to rapid-eye movement (REM) sleep than overall sleep architecture. REM sleep, relative to other sleep stages, is a state of central nervous system arousal, and fragmented REM sleep following trauma predicts later development of PTSD [14]. Further, there is evidence for both heightened and attenuated REM sleep in PTSD. Specifically, REM sleep disturbances include increased phasic motor activation and disruptions in continuity, frequent transitions from REM sleep to Stage N1 or wake, shorter continuous period of REM sleep prior to stage shifts or arousals, increased REM interruptions, and less REM and Lo-Deep (<1 Hz) sleep [15–18].

The concept of trauma-associated sleep disorders (TASD) is a relatively new idea in the sleep/PTSD field. Mysliwiec and colleagues [19, 20] define TASD as a parasomnia that encompasses nightmares, disruptive nocturnal behaviors, in addition to REM sleep without atonia (RWA). While TASD encompasses the clinical criteria for REM behavior disorder (RBD), three additional diagnostic criteria differentiate TASD from RBD. Specifically, (1) an inciting traumatic experience, (2) a history of dream mentation related to the traumatic experience, and (3) evidence of autonomic hyperarousal not due to sleep-disordered breathing [20].

Consistent with TASD, one study found self-reported dream enactment in about 40% of a large veteran sample [21]. However, using objective measures, one study found no RWA in 68% of the sample with PTSD only, suggesting a non-REM sleep parasomnia opposed to a REM sleep phenomenon [22]. Given the recent mixed findings, additional data is needed to ascertain whether TASD represents a separate sleep disorder [23–25].

Given the evidence of sleep disruption following trauma exposure, it is believed that traumatic stress events may interfere with the normal sleep–wake regulatory processes by sensitizing the central nervous system's arousal centers, which may lead to pronounced central and physiologic hyperarousal [26]. Further, the primary concept of hyperarousal has been linked to both the pathophysiology of insomnia and to the neurobiological alterations in the aftermath of traumatic stress events and may be a neurobiological commonality underlying traumatic stress and insomnia [26]. Thus, these data imply that trauma-induced insomnia may exhibit in the absence of diagnosed PTSD and may also present as an antecedent to the development of PTSD.

Studies have begun to explore the relationship between PTSD-related hyperarousal and sleep. For instance, Van Wky et al. [27] examined four groups [i.e. PTSD+hyperarousal (HYP), PTSD-HYP, PTSD+Depression, healthy controls (HC)] to test the hypothesis that PTSD participants with prominent hyperarousal symptoms would have more disturbed sleep than PTSD participants without hyperarousal symptoms. Using a median split of the clinician-administered PTSD scale (CAPS) hyperarousal scores (PTSD+HYP defined as scores ≥ 25; PTSD-HYP defined as scores < 25), they found that patients with PTSD+HYP experienced reduced sleep efficiency percent, had greater wake after sleep onset, and reported poorer sleep quality compared to patients with PTSD-HYP. In another study using DSM-IV criteria, Babson et al. [28] found that PTSD Cluster B symptoms were related to nightmare reports and trouble initiating and maintaining sleep, and PTSD Cluster D symptoms were related to nightmare reports and difficulty maintaining sleep. Recurrent nightmares are part of the intrusion (Cluster B) symptoms and are hypothesized to be the result of an imbalanced hyperarousal system contributing to abnormal cortical hyperarousal during sleep [6]. Collectively, these studies provide some preliminary evidence for a relationship between sleep characteristics and PTSD hyperarousal; however, in the Van Wky et al.'s study [27], participants had only one sleep lab night, thus they did not account for an adaptation night. Further, the method used to assess PTSD symptoms in the Babson et al.'s study [28] did not contain the full range of probable PTSD symptom levels. Finally, both studies were skewed toward women, further limiting the generalizability of their study findings. In a more recent study [29], significant associations were found between veterans with posttraumatic stress symptoms combined with hyperarousal symptoms and sleep quality problems 30 years after trauma exposure. They also found that these sleep problems resulted in 2.5 greater odds of having hyperarousal symptoms throughout the follow-up period compared to veterans without sleep quality problems. While this study adds to the scarce data on PTSD hyperarousal and sleep disturbances, the findings are limited due to self-reported posttraumatic stress symptoms and sleep problems.

Overall, data are scant regarding the relationship between objective sleep and PTSD symptom clusters, and to our knowledge limited data have specifically focused on understanding the role of hyperarousal. Therefore, using DSM-IV criteria, the present study had two aims: (1) To examine the associations between sleep continuity, sleep architecture, and PTSD symptom Clusters (reexperiencing or B; Avoidance or C; Hyperarousal or D); and (2) To determine which sleep parameters (sleep continuity, sleep architecture, REM sleep variables) were contributors to total PTSD hyperarousal symptoms. Given the present

sample includes both trauma-exposed US military veterans and civilians, exploratory aims included examining group differences (i.e. civilian versus veteran, and female versus male) between clinical measures of sleep and PTSD symptomatology.

## Methods

### Participants

A total of seventy-one (27 veterans, 44 civilians) participants were included in the present analyses and were drawn from two separate studies. In the first study, 27 Operation Enduring Freedom/Operating Iraqi Freedom (OEF/OIF) veterans were recruited from 2013 to 2015 to examine neurobiological and neuropsychological domains, and biomarkers of arousal to identify those factors that distinguished combat-exposed veterans with PTSD to those without PTSD. They were recruited from the Corporal Michael J. Crescenz Veterans Affairs Medical Center (CMCVAMC). To be deemed eligible, participants had to be currently enrolled in treatment at the CMCVAMC with a primary care provider and/or a mental health provider. Additional inclusion criteria were: male and female veterans of OEF and/or OIF (all combat-exposed); meeting DSM-IV criteria for current PTSD related to their combat experience within the past month (PTSD group only) or no lifetime history of PTSD (control group); ability to read and speak English; concurrent anxiety or depressive diagnoses were allowed. The Structured Clinical Interview for DSM-IV (SCID) was used to assess for psychiatric comorbidities. Participants were ineligible if: there was evidence of substance dependence during the preceding six months and evidence of "at risk" drinking behavior over the past month. Specifically, for men: more than 4 drinks on a given night, drinking on more than 3 nights a week, or more than 14 total drinks in a week; for women: more than 3 drinks in a given night, drinking on more than 3 nights a week, or more than 7 total drinks in a week. Other exclusion criteria were: bipolar disorder, delirium, dementia, amnesic disorder, schizophrenia, and other psychotic disorders. Participants were also excluded if they endorsed severe TBI (i.e. loss of consciousness or alteration of mental status greater than 24 h, or peri-traumatic memory loss, or any posttraumatic amnesia greater than 7 days). Medication use was not an exclusion criterion for this sample. This study was approved by the CMC VAMC Institutional Review Board. All participants provided written informed consent.

In the second study, 44 trauma-exposed participants were recruited from a larger study (2008–2012) designed to examine associations between PTSD, sleep, and nocturnal blood pressure. Participants were recruited from the Washington, DC metropolitan region. Exclusion criteria were chronic medical conditions (i.e. severe asthma, cancer, diabetes, emphysema, thyroid disease, kidney and liver disease, heart disease, seizures); psychiatric illnesses to include psychotic disorders, major depressive disorder, and bipolar disorder, and any other condition that required consistent use of medications and/or affected blood pressure (i.e. antihypertensive medication) per study protocol; a body mass index ≥ 40, excessive use of caffeine (>5 cups of coffee/tea/soda per day or the equivalent); heavy smoking (>20 cigarettes per day); hazardous (per NIAAA guidelines) alcohol use (>14 drinks/week in men, >7 drinks/week in women); and regular night shift work or an unusual sleep-wake schedules.

Other exclusion criteria were current drug abuse or dependence, a positive urine toxicology screen for illicit drugs, and a history of head injury that resulted in loss of consciousness. This study was approved by the Howard University Institutional Review Board. Written informed consent was obtained from all study participants.

Additional exclusion criteria for both studies included sleep-related breathing and sleep-related movement disorders (screened using the first-night polysomnographic recording).

### Measures

#### Psychiatric assessment

The Clinician Administered PTSD Scale (CAPS) [30] was used to determine whether participants met diagnostic criteria for PTSD. The CAPS is considered the "gold standard" in PTSD assessment. It is a structured clinical interview designed to determine lifetime and current PTSD diagnostic status [2]. For the present study, DSM-IV criteria were used because the DSM-5 criteria had not been published prior to the start of study recruitment. The CAPS-IV provides a continuous score of symptom severity based on frequency (F) and intensity (I). The F1/I2 scoring rule was employed. According to this rule, a PTSD symptom is considered present if the frequency of the corresponding CAPS item is rated as 1 or higher and intensity is rated as 2 or higher [31]. The CAPS total severity score is calculated by summing the frequency and intensity scores, resulting in a range of 0–136. In addition to a total severity score, symptom cluster scores were computed as follows: items 1–5 for criterion B; items 6–12 for criterion C; item 13–17 for criterion D. The CAPS-IV has demonstrated good psychometric properties across a wide variety of clinical and research populations, with test–retest reliability ranging from .90 to .98 [30]. The present sample had an excellent reliability coefficient (α = .96).

The Structured Clinical Interview for DSM-IV (SCID-IV) [32] is a semi-structured interview designed to allow for making DSM-IV Axis I diagnoses. The SCID was used to determine lifetime and current mood disorders, psychotic disorders, anxiety disorders, and substance abuse/dependence for the purpose of determining study eligibility.

#### Sleep assessment

Participants underwent two in-laboratory sleep studies to assess for sleep continuity and sleep architecture. Standard polysomnographic (PSG) procedures were used to record the electroencephalogram (EEG), electrooculography (EOG), electromyography (EMG), and electrocardiogram (EKG) using the Sandman (veterans) or Embla (civilians) Systems. Electrode placements of Fp3, Fp4, C3, C4, O3, and O4 were used according to the International 10/20 system. Two EOG electrodes were placed, positioned 1 centimeter (cm) below and lateral to the outer canthus of the left eye and 1 cm above and lateral to the outer canthus of the right eye. Two surface EMG electrodes were taped onto the chin 2 cm apart. Two electrodes were taped over the anterior tibialis muscle of each leg to detect leg movements during the night. Flexible Resp-EZ belts were placed around the abdomen and chest to measure breathing-related movements during the night. A nasal cannula was used to detect pressure and an oximeter probe placed on the finger to measure blood oxygen saturation.

The criteria for defining sleep disorders were an apnea-hypopnea index (AHI) greater than 15 events per hour for sleep apnea and a periodic limb movement disorder (PLMD) index greater than 15 events per hour for periodic limb movements. Records were scored in 30-second epochs according to standard criteria [33]. PSG data were used to compute standard sleep architecture variables of the time spent in each stage of sleep in terms of minutes of total sleep time. In addition, the following sleep continuity variables were computed: sleep latency (SL; time in minutes from lights out to the first epoch of stage 2 or higher), total sleep time in minutes (TST), wake after sleep onset (WASO; number of minutes spent awake between lights out and lights on), and sleep efficiency percent (SE%; total sleep time divided by the total recording period).

The first night study was used to adapt participants to the sleep laboratory environment and to determine if there were any unknown sleep disorders. The second study night occurred within one month of study night one for the veteran sample and the night after the first sleep study for the civilians. All analyses reported used the second night recordings. PSG sleep variables for the present study were: TST minutes, SE percent, WASO minutes, Stages N1, N2, and N3 sleep minutes, and REM minutes. Additional REM variables included REM latency minutes and total count/number of REM periods.

### Data analysis

Statistical Package for the Social Sciences (SPSS; version 24) was used to analyze the data. Descriptive statistics were conducted to determine the prevalence rates or means and standard deviations of all study measures. Initial analyses included correlations to determine bivariate relationships between all study variables. T-tests and chi-squared tests were conducted to examine group differences (veterans vs civilians, and male vs females) on demographic variables and all clinical measures and sleep variables. CAPS sleep items were removed from the correlational analysis and all regression models to account for potential confounds. Separate multiple linear regression models were conducted to determine the effects of sleep continuity and architecture on PTSD symptom Clusters (B, C, D). An additional model was also generated to determine the effects of REM sleep variables specifically on PTSD hyperarousal symptoms (Cluster D). To account for multiple comparisons, Bonferroni corrections were made for all regression models, such that the statistical significance was set at $p < .005$.

## Results

Participants were civilians (62%) and veterans (38%) between the ages of 18 and 53 ($M = 27.39$, $SD = 9.69$). Approximately 20% of the participants were married, 76% identified as African American, 65% completed some college/associate degree. A complete description of study demographics and group differences are presented in Table 1.

### Associations between sleep and PTSD symptom clusters

All correlational analyses are presented in Table 2. Results showed that all symptom clusters were negatively associated with total sleep time minutes. Additionally, apart from sleep latency, sleep continuity variables (i.e. TST, SE%, WASO)

**Table 1.** Demographic characteristics of study participants

| | Total Sample (N = 71) | Veterans (n = 27) | Civilians (n = 44) | t-test or Chi-square | P | Phi or CI |
|---|---|---|---|---|---|---|
| Age, mean (SD) | 27.39 (9.69) | 36.93 (8.69) | 21.55 (3.88) | 10.23 | <.001 | [12.38, 18.38] |
| Sex (n, % female) | 29 (40.8) | 3 (11.1) | 26 (59.1) | 15.94 | <.001 | -.474 |
| **Highest education level completed** (n, %) | | | | 6.20 | .102 | .296 |
| High school/GED | 11 (15.5) | 2 (7.4) | 9 (20.5) | | | |
| Some college/associate | 46 (64.8) | 16 (59.3) | 30 (68.2) | | | |
| College | 9 (12.7) | 6 (22.2) | 3 (6.8) | | | |
| Graduate school | 5 (7.0) | 3 (11.1) | 2 (4.5) | | | |
| **Race** (n, %) | | | | 36.43 | <.001 | .716 |
| African American | 54 (76.1) | 10 (37.0) | 44 (100) | | | |
| Caucasian | 15 (21.1) | 15 (55.6) | – | | | |
| Other/mixed/unknown | 2 (2.8) | 2 (7.4) | – | | | |
| **Marital Status** (n, %) | | | | 41.52 | <.001 | .765 |
| Never married | 50 (70.4) | 7 (25.9) | 43 (97.7) | | | |
| Married | 15 (21.2) | 14 (51.9) | 1 (2.3) | | | |
| Separated/divorced | 6 (8.4) | 6 (22.2) | – | | | |
| **Trauma Type** (n, %) | | | | 71.00 | <.001 | 1.00 |
| Transportation Accident | 1 (1.4) | – | 1 (1.4) | | | |
| Accident | 1 (1.4) | – | 1 (1.4) | | | |
| Violent Crime (non-sexual) | 24 (33.8) | – | 24 (33.8) | | | |
| Sexual Trauma | 10 (14.1) | – | 10 (14.1) | | | |
| Combat-Trauma | 27 (38.0) | 27 (38.0) | – | | | |
| Sudden Unexpected Death of Someone Close | 2 (2.8) | – | 2 (2.8) | | | |
| Other Potentially Traumatic Event | 6 (8.5) | – | 6 (8.5) | | | |

SD, standard deviation; GED, general equivalency diploma.

**Table 2.** Intercorrelations among posttraumatic stress symptoms and sleep continuity and architecture measures (N = 71)

| Measures | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. CAP total scores | — | | | | | | | | | | | | | |
| 2. Cluster B symptoms | .93*** | — | | | | | | | | | | | | |
| 3. Cluster C symptoms | .96*** | .83*** | — | | | | | | | | | | | |
| 4. Cluster D symptoms | .96*** | .87*** | .86*** | — | | | | | | | | | | |
| 5. Total sleep time | -.41*** | -.44*** | -.38** | -.35** | — | | | | | | | | | |
| 6. Sleep efficiency percent | -.26* | -.30** | -.17 | -.25* | .52*** | — | | | | | | | | |
| 7. Wake after sleep onset | .24* | .25* | .18 | .27 | -.33** | -.54*** | — | | | | | | | |
| 8. Sleep latency | .01 | .03 | -.05 | -.01 | -.14 | -.70*** | -.17 | — | | | | | | |
| 9. Stage N1 sleep | .66*** | .60*** | .61*** | .68*** | -.45*** | -.31** | .40*** | -.08 | — | | | | | |
| 10. Stage N2 sleep | .50*** | .38** | .49*** | .49*** | -.14 | -.04 | .05 | -.08 | .54*** | — | | | | |
| 11. Stage N3 sleep | -.52*** | -.46*** | -.48*** | -.50*** | .44*** | .32** | -.31** | -.03 | -.63*** | -.36*** | — | | | |
| 12. REM sleep | -.27* | -.31* | -.23 | -.27* | .66*** | .41*** | -.32** | -.10 | -.33** | -.33** | .07 | — | | |
| 13. REM sleep latency | .28* | .17 | .24* | .30* | -.18 | -.04 | .26* | -.20 | .42*** | .42*** | -.25* | -.47*** | — | |
| 14. # of REM sleep periods | -.42*** | -.35** | -.42*** | -.38** | .53*** | .18 | -.08 | -.01 | -.41*** | -.41*** | .35** | .43*** | -.41*** | — |

All sleep variables are in minutes with the exception of sleep efficiency percent and REM sleep periods. CAPS, clinician-administered PTSD scale; REM, rapid eye-movement. Bold indicates significance at *p < .05. **p < .01. ***p < .001.

were correlated with Cluster B (reexperiencing) and Cluster D (hyperarousal) symptoms, such that an increase in WASO minutes and a decrease in TST minutes and SE% were correlated with greater reexperiencing and hyperarousal symptom severity. Sleep architecture variables (i.e. Stages N1, N2, N3) were correlated with Cluster B, C (avoidance) and D symptoms, such that an increase in Stages N1 and N2 sleep was associated with greater reexperiencing, avoidance, and hyperarousal symptom severity. Total time in REM sleep was negatively correlated with Cluster B and D symptoms. Findings also showed that REM sleep latency was positively correlated with Cluster C and D symptoms, and the number of REM sleep periods was negatively correlated with Cluster B, C, and D symptoms. These results suggest that fewer REM sleep periods, and consequently less time in REM sleep, were related to greater total PTSD severity.

## Sleep parameters predicting PTSD symptom clusters

### Reexperiencing symptoms
Regression models showed significant relationships between sleep and clinical variables. Sleep continuity variables significantly predicted Cluster B symptoms ($R^2$ = .26, F = 5.83, p < .001). Specifically, SE% (t = −2.16, p = .034) and SL (t = −2.14, p = .036) predicted Cluster B symptoms independently of the effects of TST (t = −.02, p = .984) and WASO (t = −1.90, p = .062). However, after adjusting for multiple comparisons, the relationships between Cluster B symptoms and sleep continuity variables were no longer statistically significant. Sleep architecture variables significantly predicted Cluster B symptoms, accounting for 39.5% of the variance in this relationship. Specifically, Stage N1 sleep (t = 3.06, p = .003) significantly predicted Cluster B symptoms independently of the effects of Stages N2 (t = .42, p = .674), N3 (t = −1.14, p = .260), and REM (t = −1.53, p = .132) sleep.

### Avoidance symptoms
Sleep continuity variables predicted Cluster C symptoms, accounting for 19% of the variance in this relationship. After adjusting for multiple comparisons, sleep architecture variables also significantly predicted Cluster C symptoms ($R^2$ = .42, F = 11.84, p < .001); specifically, Stage N1 sleep (t = 3.11, p = .003) predicted these symptoms independently of the effects of Stages N2 (t = 1.74, p = .086), N3 (t = −.63, p = .533) and REM (t = −.67, p = .506) sleep.

### Hyperarousal symptoms
An examination of the relationship between sleep variables and Cluster D symptoms showed that sleep staging and REM sleep variables (i.e. REM latency, number of REM periods) were significant predictors of Cluster D symptoms after adjustment for multiple comparisons (See Table 3). Specifically, Stage N1 sleep (t = 4.11, p < .001) predicted these symptoms independently of the effects of Stages N2 (t = 1.47, p = .147), N3 (t = −.57, p = .570), and REM (t = −.84, p = .407) sleep. Also, the number of REM sleep periods and REM sleep latency significantly predicted Cluster D symptoms, accounting for 16.8% of the variance in this relationship.

## Exploring group differences

Group differences (veterans vs civilians) for sleep and clinical variables are presented in Table 4. There were significant group

**Table 3.** Influence of polysomnographic sleep on Cluster D (hyperarousal) symptoms in veterans and civilians (N = 71)

| | B | SE B | $R^2$ | Adjusted $R^2$ | F |
|---|---|---|---|---|---|
| **Sleep Continuity, min** | | | .17 | .12 | 3.46 |
| Total sleep time | −.01 | .03 | | | |
| Sleep efficiency, % | −1.19 | .87 | | | |
| Wake after sleep onset | −.21 | .20 | | | |
| Sleep latency | −.23 | .17 | | | |
| **Sleep Architecture, min** | | | .49 | .46 | 16.06*** |
| Stage N1 | .61 | .15 | | | |
| Stage N2 | .03 | .02 | | | |
| Stage N3 | −.02 | .03 | | | |
| REM Sleep | −.02 | .03 | | | |
| **REM sleep measures, min** | | | .17† | .14 | 6.85** |
| REM sleep latency | .03 | .02 | | | |
| Number of REM sleep periods | −.86 | .34 | | | |

PSG, polysomnography; REM, rapid eye movement.
**$p$ < .005. ***$p$ < .001.
†Due to rounding.

**Table 4.** Means and standard deviations of clinical measures and sleep variables

| | Total sample (N = 71) | Veterans (n = 27) | Civilians (n = 44) | P, CI |
|---|---|---|---|---|
| PTSD total symptoms, mean (SD) | 39.56 (34.60) | 65.15 (36.94) | 23.86 (21.47) | **<.001 [25.46, 57.11]** |
| **PTSD total cluster symptom scores, mean (SD)** | | | | |
| Cluster B (reexperiencing) | 10.82 (9.95) | 16.85 (11.10) | 7.11 (7.05) | **<.001 [4.91, 14.56]** |
| Cluster C (avoidance) | 14.69 (14.59) | 25.41 (15.62) | 8.11 (9.09) | **<.001 [10.60, 23.99]** |
| Cluster D (Hyperarousal) | 14.05 (11.88) | 22.89 (11.71) | 8.64 (8.22) | **<.001 [9.06, 19.44]** |
| **PTSD hyperarousal symptoms, mean (SD)** | | | | |
| Irritability or outbursts of anger | 2.49 (2.81) | 4.41 (2.69) | 1.32 (2.19) | **<.001 [1.92, 4.26]** |
| Difficulty concentrating | 2.37 (2.78) | 4.30 (2.64) | 1.18 (2.15) | **<.001 [1.97, 4.26]** |
| Hypervigilance | 3.77 (3.15) | 5.33 (3.04) | 2.82 (2.85) | **.001 [1.09, 3.94]** |
| Exaggerated startle response | 1.77 (2.47) | 3.07 (2.95) | .98 (1.73) | **<.001 [1.00, 3.20]** |
| **PSG sleep continuity, min** | | | | |
| Total sleep time | 353.06 (65.33) | 322.52 (63.60) | 371.80 (59.65) | **.002 [−79.11, −19.45]** |
| Sleep efficiency, % | 84.99 (10.20) | 82.52 (11.62) | 86.50 (9.03) | .111 [−8.90,.93] |
| Wake after sleep onset | 28.99 (27.17) | 33.90 (33.68) | 25.98 (22.18) | .236 [−5.29, 21.13] |
| Sleep latency | 34.56 (41.28) | 33.52 (36.41) | 35.21 (44.40) | .869 [−21.96, 18.59] |
| **PSG sleep architecture, min** | | | | |
| Stage N1 | 7.42 (8.10) | 13.83 (10.06) | 3.49 (2.02) | **<.001 [7.24, 13.46]** |
| Stage N2 | 121.64 (54.29) | 183.51 (35.38) | 83.68 (12.79) | **<.001 [88.14, 111.50]** |
| Stage N3 | 80.45 (37.27) | 53.35 (31.24) | 97.08 (30.45) | **<.001 [−58.72, −28.73]** |
| REM Sleep | 75.96 (30.89) | 71.85 (35.14) | 78.48 (28.10) | .384 [−21.72, 8.46] |
| **PSG REM sleep measures, min** | | | | |
| REM sleep latency | 91.10 (54.93) | 116.65 (66.83) | 75.42 (39.38) | **.002 [16.13, 66.33]** |
| Number of REM sleep periods | 4.97 (3.41) | 2.56 (1.28) | 6.45 (3.47) | **<.001 [−5.29, −2.51]** |

PTSD, posttraumatic stress disorder; SD, standard deviation; PSG, polysomnography; REM, Rapid eye movement; CI, confidence intervals.
Bold indicates significant P-values and CI.

differences on CAPS total and all symptom cluster scores, with veterans being more symptomatic than civilians. Further, there were significant group differences on the individual hyperarousal symptoms, with veterans endorsing greater symptom severity on all items compared to civilians. There were group differences on sleep continuity, with civilians indicating greater TST compared to veterans. There were also group differences on sleep architecture, specifically, veterans had greater amounts of Stages N1 and N2 sleep compared to civilians, whereas civilians had greater amounts of Stage N3 sleep compared to veterans.

Given the existing literature suggesting sex differences on sleep and clinical variables [34–38], we explored sex differences on CAPS scores, and sleep continuity and architecture. We found that males were more symptomatic on CAPS total

scores ($t$ = −2.35, $p$ = .022), Cluster B ($t$ = −2.37, $p$ = 0.20), Cluster C ($t$ = −2.21, $p$ = .030), and Cluster D ($t$ = −2.12, $p$ = .038) symptoms compared to females. The only sex difference for sleep continuity measures was on TST, with females having greater TST compared to males ($t$ = 2.41, $p$ = .019). There were significant sex differences on sleep architecture, specifically, males had higher amounts of time spent in Stages N1 ($t$ = −3.83, $p$ < .001) and N2 ($t$ = −3.90, $p$ < .001) sleep, and lesser amounts of time spent in Stage N3 sleep ($t$ = 3.33, $p$ = .001) compared to females.

## Discussion

While there is evidence that PTSD symptoms vary widely and numerous possible presentations of the disorder exist [39], less

is known about distinct sleep profiles among trauma-exposed groups. Therefore, this study aimed to better understand how objectively measured sleep parameters may be associated with the unique PTSD symptom clusters. Overall, the findings are consistent with the extant literature and provide evidence that objective impairment in sleep maintenance is highly correlated with PTSD symptoms [40].

Specifically, greater time spent in Stage N1 sleep (which can be an indicator of poorer sleep quality; e.g. [41],) consistently and significantly predicted PTSD Clusters B (reexperiencing), Cluster C (avoidance), and Cluster D (hyperarousal) symptoms. Given the link between Stage N1 sleep to all PTSD Clusters, it may be a non-specific but reliable marker of PTSD. There are data to suggest that using spectral power analysis with symptom clusters may further refine this outcome [42].

In an extended investigation regarding the relationship between Cluster D symptoms and sleep architecture, we also found that REM sleep variables (i.e. REM sleep latency, number of REM sleep periods) significantly predicted Cluster D symptoms accounting for 16.8% of the variance in this relationship. Overall, these findings provide preliminary evidence showing that REM sleep processes may specifically contribute to maintaining PTSD hyperarousal symptoms in both veterans and civilians. Although these results were conserved with corrections, additional work is needed to understand the role of hyperarousal in PTSD. One consideration is that hyperarousal may be manifested by two distinct pathways via a daytime and a nocturnal pathway of hyperarousal. For example, there is ample evidence showing residual sleep symptoms during and following PTSD treatments [43–48], and that sleep disturbances in PTSD generally exacerbate the waking symptoms of the disorder [49, 50].

Given the opportunity to examine these relationships with unique samples of trauma-exposed adults, the group differences showed that combat-exposed veterans were more symptomatic and exhibited greater sleep disturbance compared to trauma-exposed civilians. Veteran populations are particularly more vulnerable to sleep disturbances compared to civilians due to irregular sleep/wake schedules in addition to other factors related to military service that may include extremes in temperature, physical exertion, stress associated with combat, and increased rates of physical and psychological injury [51–53]. Further, evidence suggests that active duty service members experience higher levels of traumatic stress, psychiatric comorbidity, and medical conditions (which can carryover to their transition as veterans) that may contribute to increased risk for insomnia compared to the general adult population [54, 55]. Additionally, males were more symptomatic on total CAPS scores and PTSD symptom clusters compared to females; and males had greater time spent in Stage N1 and N2 sleep, and lesser amounts of total sleep time and time spent in Stage N3 sleep compared to females. Collectively, these findings are partially consistent with the previous literature suggesting that men had a lower sleep efficiency percentage [56] and a higher percentage of Stage N1 sleep [57, 58], and lower Stage N3 sleep minutes compared to females [59]; and females had greater amounts of total sleep time than males [60].

While the current study has notable strengths in elucidating the relationship between objectively measured sleep and PTSD symptom clusters, the conclusions drawn from this study, and the generalizability of our findings are tempered by some limitations. First, the cross-sectional nature of this study limited the ability to make causal inferences among sleep and PTSD variables. Future studies should include longitudinal designs to elucidate the nature and significance of the relationships between sleep continuity/architecture and PTSD symptom clusters. Second, we did not account for comorbidities (e.g. depression, anxiety disorders), time since trauma, and type of trauma, which are other factors (in addition to PTSD) that may impact sleep architecture and contribute to sleep disturbance [61–63]. Relatedly, the veteran sample was more symptomatic overall compared to the civilian sample, which may be a consequence of the inclusion criteria of a current PTSD diagnosis that the civilian study did not require. Additionally, the PTSD symptom criteria have been updated since data were collected for this study. While there is evidence for overlap between the DSM-IV and DSM-5 criteria in both civilian and military samples [1, 64, 65], we cannot ascertain how the measured sleep parameters may influence the new negative alterations in cognition/mood PTSD cluster [3] or relate to the modified total symptom severity. Therefore, additional studies of mixed trauma-type and varying levels of PTSD symptomatology, including with updated PTSD diagnostic criteria, are needed. Fourth, while we explored sex differences on clinical and sleep variables, we are unable to distinguish the effects of sex from veteran status, given that the veteran sample was primarily male. It should also be noted that use of medication in the veteran sample was allowed, and mild OSA was an exclusion criterion. Finally, we did not include an objective measure of hyperarousal. This inclusion in future studies would more definitively explain the nature and role of hyperarousal in the relationship between sleep and PTSD and could inform future treatments.

The present study investigated differential relations between PTSD symptom clusters and objective sleep continuity and architecture. Our findings extend previous literature by demonstrating that objective impairment in sleep maintenance was a significant predictor of PTSD intrusion symptoms. Additionally, our findings showed that Stage N1 sleep, and, to a lesser degree, REM sleep variables played significant roles in PTSD symptom clusters in both civilians and veterans. Future research is needed to understand other factors (e.g. type of trauma) related to PTSD and objective sleep impairment, as well as identify and investigate hyperarousal pathways implicated in PTSD in order to enhance treatment outcomes.

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## Disclosure Statement

## Data Availability

The data underlying this article are not available to be shared due to ethical and institutional restrictions.

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