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Individuals with and without military-related PTSD differ in subjective sleepiness and alertness but not objective sleepiness

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ABSTRACT

Posttraumatic stress disorder-related sleep disturbances may increase daytime sleepiness and compromise performance in individuals with posttraumatic stress disorder. We investigated nighttime sleep predictors of sleepiness in Veterans with and without posttraumatic stress disorder. Thirty-seven post-9/11 Veterans with posttraumatic stress disorder and 47 without posttraumatic stress disorder (Control) completed a 48-h lab stay. Nighttime quantitative EEG and sleep architecture parameters were collected with polysomnography. Data from daytime sleepiness batteries assessing subjective sleepiness (global vigor questionnaire), objective sleepiness (Multiple Sleep Latency Tests) and alertness (psychomotor vigilance task) were included in analyses. Independent samples t-tests and linear regressions were performed to identify group differences in sleepiness and nighttime sleep predictors of sleepiness in the overall sample and within each group. Participants with posttraumatic stress disorder had higher subjective sleepiness ($t = 4.20$; $p < .001$) and lower alertness (psychomotor vigilance task reaction time ($t = -3.70$; $p < .001$) and lapses: $t = -2.13$; $p = .04$) than the control group. Objective daytime sleepiness did not differ between groups ($t = -0.79$, $p = .43$). In the whole sample, higher rapid eye movement delta power predicted lower alertness quantified by psychomotor vigilance task reaction time ($\beta = 0.372$, $p = .013$) and lapses ($\beta = 0.388$, $p = .013$). More fragmented sleep predicted higher objective sleepiness in the posttraumatic stress disorder group ($\beta = -.467$, $p = .005$) but no other nighttime sleep measures influenced the relationship between group and sleepiness. Objective measures of sleep and sleepiness were not associated with the increased subjective sleepiness and reduced alertness of the posttraumatic stress disorder group.

1. Introduction

Posttraumatic stress disorder (PTSD), a signature wound of recent military operations, is characterized by disturbed sleep (Germain et al., 2008; Grieger et al., 2006). The prevalence of sleep disturbances in military-related PTSD is higher than that seen in civilians with PTSD, which may be due to the high prevalence of disturbed sleep in military personnel overall (Good et al., 2020). Individuals with PTSD can have reduced total sleep time, reduced slow wave (deep) sleep and increased time awake after sleep onset (WASO), as outlined in a recent meta-analysis (Zhang et al., 2019). Altered rapid-eye movement sleep is also widely, albeit inconsistently reported in individuals with PTSD (Cowdin et al., 2014; Kobayashi et al., 2007; Mellman et al., 1997; Zhang et al., 2019). The heterogeneity of PTSD may contribute to inconsistency in objectively-measured sleep outcomes; for example, WASO was reduced in individuals with non-combat PTSD but not in individuals with combat-related PTSD (Zhang et al., 2019). Conversely, subjective sleep complaints are consistently reported. Individuals with PTSD report reduced total sleep time, worse sleep quality, difficulty falling asleep and increased awakenings (Hurwitz et al., 1998; Klein et al., 2002; Zhang et al., 2017).

Nighttime sleep disturbances in individuals with PTSD may increase

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daytime sleepiness, contributing to compromised vigilance and cognitive function (Dretsch et al., 2012; Scott et al., 2015). While individuals with PTSD consistently report increased daytime sleepiness (Germain et al., 2004; Hurwitz et al., 1998), studies objectively measuring daytime sleepiness report conflicting results. Zhang and colleagues (2017) reported shorter sleep latency (SL; 10.8 ± 3.6 vs. 14.1 ± 2.9 min) on the Multiple Sleep Latency Test (MSLT), the gold standard objective measure of daytime sleepiness, in individuals with PTSD. Other studies of daytime sleepiness in civilians (Breslau et al., 2004) and Vietnam War Veterans (Hurwitz et al., 1998), however report no differences in MSLT SL between individuals with and without PTSD. Interestingly, individuals with PTSD did report more subjective sleepiness despite demonstrating normal levels of objective daytime sleepiness (MSLT SL > 8 min) (Breslau et al., 2004; Hurwitz et al., 1998; Roehrs and Roth, 1992). Consistent with studies of healthy sleepers (Franzen et al., 2008; Van Dongen et al., 2004), subjective and objective measures of daytime sleepiness may measure different dimensions underlying sleepiness in PTSD, but their relationship needs to be further defined. Furthermore, investigating the relationship between functional measures of alertness and measures of daytime sleepiness may provide insight into the functional implications of increased daytime sleepiness in PTSD, which, in turn, can impact fitness for duty decision-making.

The psychomotor vigilance task (PVT) assesses vigilance and alertness, providing a functional measure of sleepiness (Dinges et al., 1997). Under laboratory conditions, shortened sleep duration results in increased subjective sleepiness, objective sleepiness, and PVT performance decrements (Carskadon and Dement, 1982; Dinges et al., 1997; Van Dongen et al., 2004). While Dinges and colleagues (1997) report that subjective, objective and functional measures of sleepiness respond to sleep restriction in a similar fashion, other groups were unable to confirm those relationships suggesting instead that the measures reflect different aspects of sleepiness (Buysse et al., 2008; Franzen et al., 2008; Van Dongen et al., 2004).

Identifying the relationship between different daytime sleepiness measures within individuals with PTSD compared to individuals without PTSD, along with understanding how nighttime sleep impacts sleepiness can provide valuable insight into mechanisms underlying PTSD symptomology. Furthermore, understanding the relationship between nighttime sleep, daytime sleepiness and performance measures in individuals with PTSD can provide insight into implications for fitness for duty. In turn, this effort can identify targets for mitigation strategies and inform the development of effective sleep- and/or sleepiness-focused countermeasures.

This study sought to investigate the relationship between nighttime sleep and both objective and subjective measures of daytime sleepiness within a population of post 9/11 active duty, reserve and Veteran-status military personnel with and without service-related PTSD. We first aimed to investigate differences in daytime sleepiness between groups using subjective, objective and functional measures. Our second aim was to investigate whether nighttime sleep parameters predicted daytime sleepiness within each group. To this end, group differences in nighttime sleep parameters and relationships between nighttime sleep and daytime sleepiness in the overall sample were assessed. It was hypothesized that participants with PTSD would present with higher subjective and objective sleepiness and lower alertness, and that increased sleepiness would relate to nighttime sleep disturbances.

2. Methods

2.1. Participants

Eighty-five post 9/11 Veterans and Service Members, 48 controls and 37 with PTSD, completed screening and experimental procedures. Experimental procedures were completed between September 2015 and June 2018. All participants were between the ages of 18–50 and were deployed at least once in support of the Global War on Terror. Participants were informed that all data collected would be kept strictly confidential and no information would enter their medical or military record. Participants received financial compensation for their participation in the study. This study was approved by the University of Pittsburgh Institutional Review Board and by the Office of Research Protections of the US Army Medical Research and Development Command at Ft. Detrick, MD.

After signing informed consent, participants completed screening procedures to determine eligibility and group assignment. All participants completed self-report questionnaires, clinician-administered interviews, a health review and drug screen, and an in-home sleep apnea study or in-laboratory sleep PSG screen. Self-report measures included the Insomnia Severity Index (ISI) (Bastien et al., 2001), Epworth Sleepiness Scale (ESS) (Johns, 1991), The Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989) and The Pittsburgh Sleep Quality Index Addendum for PTSD (PSQI-A) (Germain et al., 2005). The Structured Clinical Interview for DSM-IV (SCID) (Association, 2000) was administered to assess mood, psychotic, and substance use disorders. The Structured Clinical Interview for Sleep Disorders (SCI-SLD) and the Clinician Administered PTSD Scale (CAPS) were also administered and used to assess for the presence of DSM-IV sleep disorders and PTSD, respectively. The CAPS is considered the gold standard for diagnosing both past and current PTSD (Weathers et al., 2001). PTSD diagnosis was determined based on the 1–2 scoring rule; participants were assigned to the PTSD group if they met criteria for PTSD and to the control group if they did not meet criteria (Weathers et al., 2001).

Participants were excluded if they had any of the following: 1) extremely severe PTSD (CAPS score >80 with marked functional impairments in activities of daily living); 2) history of psychotic or bipolar disorder; 3) current history (within 3 months) of alcohol or substance dependence; 4) current diagnosis of untreated and/or severe depression; 5) using medications that affect sleep or wakefulness within the past 2 weeks (6 weeks for fluoxetine); 6) consumed ≥4 cups of caffeine per 24 h; 7) consumed ≥2 alcoholic drinks per day/≥14 alcoholic drinks per week; 8) current post-concussive symptoms and/or rehabilitation treatment for traumatic brain injury; 9) significant or unstable acute or chronic medical conditions; 10) pregnant and/or breastfeeding; 11) sleep disorders such as delayed sleep phase syndrome, narcolepsy, restless leg syndrome, periodic leg movement disorder, obstructive sleep apnea, or current night shift work, or 12) positive drug screen. Participants excluded due to severe PTSD with functional impairments were referred to treatment.

2.2. Procedures

Prior to beginning experimental procedures, participants were asked to limit caffeine intake to 2 cups per day for the 2 weeks leading up to the study to minimize potential effects of caffeine withdrawal. Due to the effects of caffeine on sleep and sleepiness, it was not allowed during the study. In the 3–7 days leading up to the lab stay, participants also completed sleep monitoring with a sleep diary and activity monitor to ensure stable sleep-wake patterns, which were reviewed upon arrival to the laboratory.

Experimental procedures included a 48-h lab stay which began with a familiarization night where participants completed trials of study tasks, and were monitored for overnight PSG recording using high-density EEG (hdEEG). Lights out was at 23:00 and lights on was at 07:00, providing participants with an 8-h sleep opportunity each night of the study to ensure all participants received adequate sleep and were not sleep restricted during the study. Participants stayed in bed with lights out for the entirety of the 8-h period and did not have access to their phone or other media during this time. Shortly after awakening, participants were prepared for a standard AASM PSG montage which was used to assess daytime sleepiness. During the daytime, participants completed the daytime sleepiness battery every 2 h from 09:00 until 19:00. The battery included a subjective sleepiness questionnaire

assessing global vigor, a functional measure of alertness using the 10-min PVT (PC-PVT 1.0 (Khitrov et al., 2014)) and an objective measure of sleepiness (MSLT), which were completed in that order (Fig. 1). Each battery lasted approximately 40 min and participants were allowed to engage in nonstrenuous activities (i.e. watching TV, reading) between batteries. Half of the daytime sleepiness batteries also included a working memory n-back task which was completed after the MSLT. N-back data has been described elsewhere (LaGoy et al., In Press). Participants were monitored by trained research staff to ensure they stayed awake and maintained an even-keeled environment throughout their stay in the lab.

2.3. Sleep PSG and spectral EEG measures

PSG was conducted on both study nights. Night 2 will be used for analysis for the present study to minimize any potential first night effects. PSG was collected using a 64-channel hdEEG net (Electrical Geodesics Inc, Eugene, OR) with 2 additional electrodes placed bilaterally on the submentalis to record electromyography (EMG). Analyses were conducted using sleep staging and quantitative EEG (qEEG; described in following section) measures from the night 2 data. Sleep stages were scored in 30-sec epochs according to AASM criteria (Iber et al., 2007). Data were processed according to standard protocols (Cohen et al., 2013). Artifact rejection occurred through an automated algorithm and was confirmed through automated and manual inspection by certified sleep technicians. Muscle artifact rejection occurred through a separate automated algorithm which identifies high frequency signals (26.25–32.00 Hz) using a 3-min moving window threshold (Brunner et al., 1996). Artifacts were rejected in 5-s epochs. Macro-architectural sleep measures of interest included total sleep time (TST), sleep onset latency (SL), wake after sleep onset (WASO), number of arousals and sleep efficiency (SE). Power spectral analysis was performed according to established laboratory guidelines (Cohen et al., 2013). Spectral EEG measures of interest included spectral power within the delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), sigma (12–16 Hz) and beta (16–32 Hz) frequency bands averaged across the scalp. Nighttime sleep data is presented to investigate the relationship between group, nighttime sleep and daytime sleepiness and is not meant to reflect an exhaustive investigation of group differences in nighttime sleep.

2.4. Daytime sleepiness measures

Subjective sleepiness was assessed using the Mood-VAS 20, a self-report questionnaire of mood, sleepiness, and alertness (Monk, 1989). The Global Vigor subscale of the Mood-VAS 20 asks participants to rate their levels of alertness, effort, weariness, and sleepiness. Increased ratings of global vigor reflect reduced daytime sleepiness and increased alertness. Participants completed six Mood Questionnaires per day in the study, for a total of twelve trials. The mean of the first 5 questionnaires of day 2 was used for analysis (Fig. 1). The last trial was excluded as it was influenced by the participant's anticipation of completing the study. Analysis focused on day 2 measures to exclude potential first night effects.

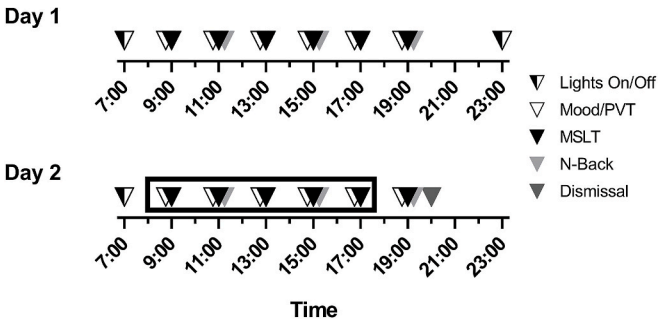

Fig. 1. Timeline depicting administration daytime testing. Trials used for data analysis are enclosed by the box on day 2. PVT = Psychomotor vigilance task; MSLT = Multiple sleep latency test.

The MSLT provides an objective measure of daytime sleepiness or sleep propensity (Littner et al., 2005). Study participants completed 6 MSLT trials each day according to established guidelines (Littner et al., 2005) and the first 5 trials on day 2 were used for analysis to account for potential first night effects on day 1 results and for potential effects of anticipating study completion on the last trial (Fig. 1). Each trial consisted of a 20-min opportunity for sleep in which participants were instructed to try to fall asleep (Littner et al., 2005). Sleep onset was defined as 3 consecutive epochs of stage 1 sleep or one epoch of any other sleep stage and was determined by live monitoring of PSG recordings. We defined MSLT SL as the time to the first epoch of consolidated sleep or the first epoch of any stage besides stage 1 (George et al., 1997; Insana et al., 2011; Reynolds et al., 1991). The test ended after the participants fell asleep or after 20 min if they were not able to fall asleep. The MSLT PSG montage consisted of AASM standard montage: 6 EEG (F3, F4, C3, C4, O1, O2), 2 EOG and 2 EMG electrodes referenced to 2 mastoid electrodes. Daytime PSG was conducted using Grass Telefactor M15 bipolar Neurodata amplifiers and Stellate-Harmonie collection software. All MSLTs were scored by trained sleep technicians in 30-sec epochs using standard sleep stage scoring criteria (Iber et al., 2007). The primary measure of interest was average SL across the 5 naps. Participants were also classified as having pathological (SL $<$ 5 min), borderline pathological (SL 5–8 min), or normal (SL $>$ 8 min) daytime sleepiness (Roehrs and Roth, 1992). The numbers of participants who entered various sleep stages during individual naps and had sleep onset REM periods (SOREMP) were also presented for completeness.

The PVT, a sustained-attention reaction time (RT) task was used as the functional measure of alertness. A 10-min computer-based version (PC-PVT 1.0) of the task was used (Khitrov et al., 2014). Participants focused on a fixation cross centered on a black background and were instructed to respond as quickly as they could once red numbers appeared on the screen. The numbers counted up from 0, providing participants with immediate feedback about their reaction time. Stimuli were presented at variable intervals from 2 to 10 s following standard practice. Participants completed six PVT trials per day in the study. Data analysis was limited to the first 5 PVT trials on day 2 for the same reason as above (Fig. 1). PVT outcome measures included median RT, lapses (RT $>$ 500 ms), fastest 10% RT and slowest 10% RT. Median RT and lapses were used to characterize alertness as these measures are sensitive to sleep loss (Dinges et al., 1997; Khitrov et al., 2014; Van Dongen et al., 2004). Fastest and slowest 10% RT are presented for completeness. The fastest 10% RT provides a measure of optimum functional capabilities while the slowest 10% RT provides a measure of lapse severity (Dinges et al., 1997; Drummond et al., 2005).

2.5. Data analysis

Shapiro-Wilk tests were used to assess for normality. Sleep efficiency was transformed through a reciprocal natural log transformation. Nighttime SL, WASO, number of arousals and spectral parameters were log-transformed. Independent samples t-tests and chi-square tests were performed to assess group differences on continuous (questionnaires, daytime sleepiness measures, nighttime sleep parameters) and categorical (demographics, MSLT groups and sleep stages) variables respectively. Linear regressions were performed to identify nighttime sleep parameters that predicted daytime sleepiness and that would be potential moderators. Separate regressions were performed for sleep efficiency, number of arousals, non-REM (NREM) and REM parameters. Sleep efficiency was selected as a measure of whole night sleep quality as it incorporates WASO and TST. NREM parameters included percent time spent in N2 and N3 and power within the delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), sigma (12–16 Hz) and beta (16–32 Hz) frequency bands. REM parameters included percent time spent in REM and power

within each frequency band. The influence of significant nighttime predictors of daytime sleepiness was further assessed within each group to understand how nighttime sleep parameters may relate differently to daytime sleepiness of control and PTSD participants. Separate linear regressions of nighttime sleep parameters on daytime sleepiness were run within each group after adjusting for ISI and PSQI-A scores. Models were adjusted for ISI and PSQI-A to investigate differences in daytime sleepiness that may be attributable to PTSD symptoms not related to sleep complaints which are captured in the ISI and PSQI-A. All statistical analyses were performed in SPSS, version 25 (Armonk, NY) with $\alpha = 0.05$ set a priori. Bonferroni corrections were used to adjust for multiple comparisons across the three regressions, therefore an adjusted p value of .017 was used for these comparisons.

3. Results

Forty-eight participants in the control group and 37 participants in the PTSD group completed all study procedures. One participant from the control group was missing data from the global vigor questionnaires and was excluded from data analysis, therefore the final sample included 47 control participants.

The PTSD group (18.9%) had a significantly higher proportion of females than the control group (2.1%). Overall, the sample was largely Caucasian (71.3%) and African American (8.5%). Groups did not differ in age ($t_{83} = 1.059$, $p = .293$) or racial composition ($\chi^2 = 5.186$, $p = .394$). The control group had an average age of 32.6 ± 5.7 years and the PTSD group had an average age of 31.3 ± 5.0 years. Groups did not differ on AHI but differed on baseline PTSD and sleep measures collected during screening. Differences in lifetime and past month CAPS score confirm successful group allocation and participant recruitment. One participant in the PTSD group had a CAPS score greater than 80 but did not present with functional impairments of daily activities and thus was included in the study. As expected, the PTSD group presented with increased ISI, PSQI, PSQI-A and ESS scores compared to the control group reflecting increased prevalence of disturbed sleep in the PTSD group (Table 1).

3.1. Subjective sleep measures

For subjective sleepiness, the control group (84.9 ± 9.7) reported greater vigor ($t_{55.197} = 4.201$, $p < .001$), indicating less sleepiness, compared to the PTSD group (72.2 ± 16.3).

3.2. Objective sleep and functional measures

Overall, overnight PSG and spectral measures were not significantly different between the groups. SE was slightly lower in the PTSD than in the control group but remained above 85% (Table 2). The PTSD group experienced a greater number of arousals than the control group but WASO did not differ between groups. No group differences were observed in the log-transformed spectral power measures during NREM or REM sleep (Table 3). Further, the average MSLT SL did not differ between groups ($t_{83} = -0.794$, $p = .430$); the control group had a mean SL of 6.9 ± 4.7 min and the PTSD group had a mean SL of 7.6 ± 4.3 min on the MSLTs. The proportion of participants from each group who were classified as having pathological, borderline pathological and normal sleepiness did not differ (Table 4). Groups also did not differ in the proportion of participants who had SOREMPs (Table 4). Data from individual naps are presented in the supplementary materials (Supplementary Tables 1–2).

For alertness, the two groups differed significantly on PVT median RT ($t = -3.703$, $p < .001$, $g = 0.84$) and lapses ($t = -2.126$, $p = .038$, $g = 0.49$). The PTSD group had a higher (worse) median RT and more lapses than the control (Fig. 2). The PTSD group also had significantly worse performance across the remaining PVT measures: longer RT on the slowest 10% ($t = -3.615$, $p < .001$, $g = 0.60$) and fastest 10% of trials ($t = -3.702$, $p < .001$, $g = 0.79$).

3.3. Relationships between nighttime sleep and daytime sleepiness

For the whole sample, a higher number of arousals predicted decreased vigor (β = −.217, std error = 15.157, $t = -2.002$, $p = .049$), increased (slower) median RT (β = 0.222, std error = 44.333, $t = 2.049$, $p = .044$) and increased lapses (β = 0.221, std error = 5.361, $t = 2.041$, $p = .045$)., although none of these relationships remained significant after correction for multiple comparisons. For the whole sample, increased REM beta activity predicted decreased global vigor, although this relationship was no longer significant after correction for multiple comparisons (β = −.356, std error = 11.191, $t = -2.334$, $p = .022$). None of the sleep measures predicted MSLT SL. Increased REM delta activity predicted increased (slower) median RT (β = .372, std error = 49.896, $t = 2.531$, $p = .013$) and increased lapses (β = 0.388, std error = 7.192, $t = 2.633$, $p = .010$) on the PVT. No other nighttime parameters predicted any measure of daytime sleepiness (Supplementary Tables 3–6).

Number of arousals, REM beta and REM delta were included as predictors in separate regression models. After adjusting for ISI and PSQI-A, neither REM beta nor REM delta were significant predictors of any daytime sleepiness measures in either the control or PTSD group, but number of arousals predicted MSLT SL in the PTSD group (Table 5). Increased arousals predicted decreased (shorter) MLST SL.

4. Discussion

In the current study, participants with PTSD reported more sleep complaints than participants without PTSD on the ISI, PSQI, PSQI-A and ESS, and reported lower vigor during the laboratory stay, consistent with prior studies (Germain et al., 2004; Straus et al., 2015). Increased subjective sleep complaints and sleepiness were corroborated by a functional measure of alertness, but not by objective PSG and qEEG sleep measures or objective sleepiness measures. Both groups were highly efficient sleepers (SE > 85%), but the PTSD group had slightly lower SE and more nighttime arousals than the control group. In addition, nighttime arousals predicted greater objective sleepiness in the PTSD but not the control group. No other objective architectural or

Table 1
Baseline PTSD and sleep questionnaire results for overall sample and for each group (mean ± standard deviation).

	Overall		Control (n = 47)		PTSD (n = 37)		t	p
	Mean	SD	Mean	SD	Mean	SD		
CAPS – Lifetime	48.9	33.7	27.0	23.5	77.5	21.0	−10.289	<0.001[a]
CAPS – Past Month	27.4	25.1	8.5	7.9	51.9	17.3	−14.192	<0.001[a]
AHI	2.5	2.7	2.3	2.7	2.6	2.8	−0.512	0.610
ISI	8.1	6.4	3.9	4.0	13.5	4.5	−10.397	<0.001[a]
PSQI	6.3	3.7	4.1	2.6	9.2	2.7	−8.831	<0.001[a]
PSQI-A	2.2	2.8	1.0	1.8	3.8	3.0	−4.880	<0.001[a]
ESS	6.1	3.9	4.9	3.0	7.7	4.5	−3.289	0.002[a]

[a] Statistically significant ($p < .05$); AHI = Apnea-hypopnea Index; CAPS = Clinician Administered PTSD Scale; ESS = Epworth Sleepiness Scale; ISI = Insomnia Severity Index; PSQI = Pittsburgh Sleep Quality Index; PSQI-A = PSQI Addendum for PTSD.

Table 2
Sleep architecture for control and PTSD groups (mean ± standard deviation).

	Control		PTSD		*t*	*p*	*g*
	Mean	SD	Mean	SD			
Total Sleep Time	473.36	6.15	469.57	10.65	1.749	0.084	0.45
Sleep Onset Latency**	7.36	6.07	10.54	9.35	−1.993	0.050	0.41
WASO**	41.82	31.60	49.91	28.64	−1.519	0.133	0.27
Arousals**	22.74	5.06	25.61	6.12	−2.244	0.081	0.50
Sleep Efficiency**	89.8	6.7	87.4	5.9	−2.177	0.032[a]	0.37
Stage 1 (%)	8.6	4.8	9.4	3.7	−0.843	0.402	0.18
Stage 2 (%)	53.0	6.6	54.7	7.4	−1.109	0.264	0.25
SWS (%)	14.1	7.2	11.2	6.0	1.923	0.058	0.42
REM (%)	24.3	6.2	24.6	5.6	−0.141	0.888	0.05

[a] Statistically significant ($p < .05$); **Statistics presented for analysis on transformed data; SWS = Slow wave sleep; REM = Rapid eye movement sleep; WASO = Wake after sleep onset; Hedge's g used due to unequal sample sizes.

Table 3
Log-transformed spectral activity in control and PTSD groups during NREM and REM sleep (mean ± standard deviation).

		Control		PTSD		*t*	*p*	*g*
		Mean	SD	Mean	SD			
NREM	Delta	1.87	0.17	1.88	0.13	−0.207	0.836	0.05
	Theta	1.18	0.17	1.17	0.17	0.372	0.711	0.09
	Alpha	0.81	0.21	0.80	0.23	0.069	0.945	0.02
	Sigma	0.54	0.18	0.56	0.19	−0.471	0.639	0.10
	Beta	0.29	0.13	0.33	0.14	−1.458	0.149	0.32
REM	Delta	1.26	0.14	1.29	0.15	−0.906	0.367	0.19
	Theta	0.95	0.15	0.92	0.16	0.719	0.474	0.16
	Alpha	0.60	0.17	0.60	0.21	0.083	0.934	0.02
	Sigma	0.23	0.17	0.21	0.18	0.344	0.731	0.08
	Beta	0.37	0.18	0.40	0.21	−0.945	0.347	0.23

NREM = Non-rapid eye movement sleep; REM = rapid eye movement sleep; Delta = 0.5–4 Hz; Theta = 4–8 Hz; Alpha = 8–12 Hz; Sigma = 12–16 Hz; Beta = 16–32 Hz: Hedge's g used due to unequal sample sizes.

Table 4
Counts (percent of each group) of participants from each group that had MSLT naps containing each sleep stage.

	Control	PTSD	Chi-squared	*p*
	Count (%)	Count (%)		
Stage 1	47 (100.0)	37 (100.0)	–	–
Stage 2	29 (67.1)	22 (59.5)	0.044	>0.999
SWS	0 (0.0)	0 (0.0)	–	–
REM	4 (8.5)	4 (10.8)	0.127	0.727
Multiple naps with REM	2 (4.3)	1 (2.7)	0.145	>0.999

SWS = Slow wave sleep; REM = rapid eye movement sleep; PTSD = post-traumatic stress disorder.

spectral sleep measures influenced relationships between group and daytime sleepiness measures, although REM spectral sleep measures predicted aspects of daytime sleepiness in the overall sample.

Objective sleep disturbances in individuals with PTSD have been inconsistently reported (Kobayashi et al., 2007). The absence of objective PSG and qEEG differences, however, does not rule out the possibility of functionally-relevant disruptions within the sleep neuroarchitecture of individuals with PTSD. Disturbed functioning in sleep-related regions such as the locus coeruleus, dorsal raphe nuclei and other deep brain structures, is observed in neuroimaging studies but may not be captured by PSG surface level recordings (Ebdlahad et al., 2013; Germain et al., 2008, 2013). Still, this disturbed functioning may influence daytime sleepiness. The two groups did not differ on objective sleepiness, but the PTSD group reported higher levels of subjective sleepiness than the control group, which was reflected in a functional measure of alertness. In prior studies of healthy sleepers, subjective, objective and functional measures of sleepiness reflected different aspects of sleepiness, consistent with the control group from the current study (Franzen et al., 2008; Van Dongen et al., 2004). While different measures of sleepiness seem to reflect distinct constructs in healthy controls, greater overlap may exist across these sleepiness measures in individuals with PTSD.

Increased subjective sleepiness and decreased alertness may have broader implications for operational readiness. Longer (slower) median RT and more lapses in the PTSD group reflect increased difficulty sustaining attention and decreased alertness. Further, the poorer performance observed across other PVT measures (i.e., slowest 10% RT and fastest 10% RT) indicate inferior overall performance on the PVT compared to the control group. As the operational relevance of PVT decrements in individuals with PTSD has yet to be established, the extent to which this finding extends to other domains of performance that are also critical to ensure safety and readiness in Service members remains to be investigated. Still, the decrements in alertness observed in the current study suggest interventions that improve alertness and subjective sleepiness in individuals with PTSD are needed.

Of note, both groups showed SL shorter than 8 min on the MSLT, indicating non-negligible levels of sleepiness in the sample (Littner et al., 2005). Pervasive sleep disturbances, related to work demands and military sleep schedules, which characterize active-duty and Veteran military populations, likely contributed to high objective sleepiness across both study groups (Luxton et al., 2011; Seelig et al., 2010; Troxel et al., 2015). In particular, both groups demonstrated high WASO and nighttime awakenings which may have contributed to high objective sleepiness. Approximately 47% of the control group and 38% of the PTSD group had average SL values that demonstrated pathological levels of daytime sleepiness. Further, SL, in both groups, was shorter than previously reported by Zhang and colleagues, who found an average MSLT SL of 10.8 ± 3.6 min in participants with earthquake-related PTSD (Zhang et al., 2017). While deployment- and military-related factors, common to both groups, may have contributed to the high objective sleepiness observed in both groups, higher subjective sleepiness and lower alertness in the PTSD group despite these common factors reflect alterations in daytime function that are specific to PTSD. Future studies

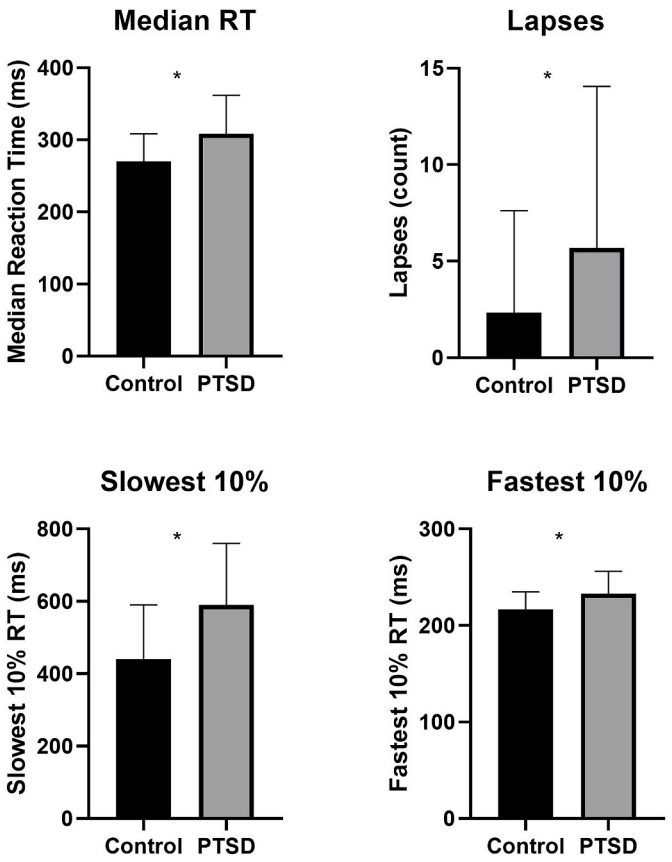

Fig. 2. Differences in alertness assessed using the psychomotor vigilance task (PVT) between the control and PTSD groups. The control group had faster (lower) median RT (A), lower lapses (B), faster fastest 10% RT (C) and faster slowest 10% RT (D) than the PTSD group. Standard deviation error bars displayed. *p < .05

with civilian, combat-naive control groups are needed to confirm this finding and to better understand the role of combat-related factors on objective sleepiness. Further, examining objective sleepiness using the Maintenance of Wakefulness test, a test similar to the MSLT that instead instructs participants to try to stay awake, would provide additional insight into the severity of daytime sleepiness and the public health implications of this daytime sleepiness in individuals with PTSD.

More fragmented sleep, marked by more nighttime arousals, predicted greater objective sleepiness (shorter MSLT SL) in Veterans with PTSD but did not influence sleepiness in Veterans without PTSD, despite both groups having fragmented sleep. Treatment interventions to reduce sleep fragmentation in individuals with PTSD may therefore reduce objective sleepiness in these individuals. No other sleep architectural or qEEG measures of nighttime sleep influenced the relationship between group and daytime sleepiness, although REM sleep parameters did predict different measures of daytime sleepiness in the whole sample. Increased REM beta predicted decreased global vigor, but this relationship was no longer significant after Bonferroni correction. Additionally, increased REM delta predicted higher (slower) PVT median RT and more PVT lapses. Neither REM beta nor delta moderated the relationship between group and daytime sleepiness. REM sleep contributes to memory, emotion processing and sensorimotor development (Baird et al., 2018; Germain et al., 2013). REM delta and beta may relate to sensory inhibition and cortical arousal during REM sleep (Bernardi et al., 2019). As such, arousal during REM sleep may influence alertness during the following day.

A few limitations must be acknowledged. Despite being one of the largest single laboratory studies of post-9/11 veterans with PSG, a larger sample for increased statistical power may be needed to identify significant differences (Kobayashi et al., 2007). In addition, all participants were free of comorbid sleep, psychiatric, or medical conditions, and participants were predominately men which may limit the generalizability. Similarly, caffeine intake was limited prior to the study, and prohibited during the laboratory stay. The extent to which caffeine may have a differential impact on sleepiness and performance among individuals with and without PTSD is unknown. Also, the instruction of "try to fall asleep" during the MSLT may have contributed to greater sleep onset anxiety in participants with insomnia, causing them to have longer MSLT SL (Roehrs and Roth, 1992). Given the low MSLT SL across both study groups, it is unclear to what extent these instructions may have impacted SL, but we cannot discount that there may have been an effect. Further, napping was not examined in the current study. While no participants in the current study were frequent nappers, future studies should examine napping in individuals with PTSD to further assess daytime sleepiness in these individuals and to examine the effectiveness of napping as an intervention strategy. Finally, the assessment of functional performance in this work was limited to the PVT. More complex assessments of cognitive and physical performance may be required to unmask related sleep determinants.

In summary, participants with PTSD reported greater feelings of daytime sleepiness than participants without PTSD, which was reflected in functional decrements, although no group differences were found on objective sleep measures. A high proportion of our sample of combat-exposed Veterans had high objective sleepiness regardless of PTSD status. In participants with PTSD, nighttime arousals predicted more objective sleepiness, identifying sleep fragmentation as a potential intervention target. Further work is needed to identify the common combat-related factors across study groups that contributed to high objective sleepiness and to identify the PTSD-specific factors that contributed to high subjective sleepiness and low alertness in the PTSD group.

Author statement

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Table 5
Regression of number of arousals, REM beta and REM delta on daytime sleepiness within control and PTSD groups.

		Arousals			REM Beta			REM Delta		
		B	Std. Error	*p*	β	Std. Error	*p*	β	Std. Error	*p*
Control	Mood	-.064	16.208	.683	-.170	8.272	.272	-.095	11.161	.549
	MSLT SL	-.026	7.512	.568	.068	3.871	.646	-.024	5.183	.875
	PVT Median RT	-.139	41.883	.347	.030	25.441	.804	.054	33.943	.663
	PVT Lapses	-.291	3.059	.057	-.071	3.275	.524	.027	4.393	.816
PTSD	Mood	-.091	23.617	.561	-.127	11.742	.410	-.090	16.968	.576
	MSLT SL	-.467	6.213	.005	.061	3.573	.732	.115	5.113	.536
	PVT Median RT	.279	76.529	.110	.263	42.698	.131	.297	60.966	.101
	PVT Lapses	.325	11.161	.065	.240	6.887	.178	.267	9.856	.149

Models adjusted for ISI and PSQI-A scores within each group; REM = Rapid eye movement sleep; PTSD = Posttraumatic stress disorder; MSLT SL = Multiple Sleep Latency Test Sleep Latency; PVT = Psychomotor vigilance task; RT = Reaction time.

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Declaration of competing interest

Dr. Germain owns equity and serves as CEO for Noctem, LLC. The work presented here does not conflict with these interests. The other authors have no conflicts of interest to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2021.07.017.

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