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Adaptation Effects to Sleep Studies in Participants with and without Chronic Posttraumatic Stress Disorder

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Abstract

The "first night effect" (FNE) is the alteration of sleep architecture observed on the first night of polysomnographic (PSG) studies. It is unclear whether the FNE reflects adaptation to the equipment, sleeping environment, or both. Moreover, it is possible that certain patient populations, such as those with posttraumatic stress disorder (PTSD), demonstrate greater adaptation effects that are highly context dependent. We assessed FNE in participants with PTSD and healthy controls in a cross sectional study consisting of PSG testing at home and in the hospital. Contrary to our expectations, the PTSD group showed no adaptation effects in either setting. Only the control group assigned to the "hospital first" condition showed significant decreases in total sleep time on night one versus night two of the study. The results suggest that the FNE is related to adaptation to the combination of the hospital environment and the recording equipment.

Keywords

posttraumatic stress disorder; adaptation effects; first night effect; ambulatory polysomnography

Introduction

The "first night effect" (FNE) is a well-known phenomenon in polysomnographic (PSG) recordings characterized by decreased total sleep time, lower sleep efficiencies, reduction in REM sleep, and longer REM latencies on the first night of testing (Agnew, Webb, & Williams, 1966). First night data are often excluded in analyses of PSG recordings because they are considered to reflect a period of adaptation that is unrepresentative of usual sleep patterns.

Although the FNE has been widely studied in healthy subjects and clinical populations, few studies have systematically examined the causes of FNE. Some ambulatory PSG studies suggest that providing a comfortable sleeping environment or conducting home recording eliminates or reduces FNE (Coates et al., 1981; Edinger, Marsh, McCall, Erwin, & Lininger, 1997; Sharpley, Solomon & Cowen, 1988). Other home PSG studies of healthy participants (Le Bon et al., 2001), elderly individuals (Wauquier, van Sweden, Kerkhof, & Hamphuisen, 1991; Edinger, Marsh, McCall, Erwin, & Lininger, 1991) and patients with generalized anxiety disorder (Saletu et al., 1996) conclude that adaptation effects occur in certain subgroups regardless of setting. Others have postulated that adaptation to PSG recording

equipment plays a significant role in FNE. Lorenzo and Barbanoj studied the FNE in healthy volunteers during three nonconsecutive sets of laboratory recordings one month apart. They found FNE only in the “very first night” of the first series of recordings (Lorenzo & Barbanoj, 2002). These results suggest that familiarity with PSG equipment may eliminate FNE in subsequent PSG studies.

Individuals with posttraumatic stress disorder (PTSD) are an important test population for PSG studies that examine FNE. Most patients with PTSD report nightmares and insomnia, which are listed separately in the re-experiencing and hyperarousal clusters in the DSM-IV criteria for the disorder (First, Spitzer, Williams, & Gibbon, 1996). Subjective sleep disturbances are frequent among patients with PTSD both in treatment seeking (Roszell, McFall, & Malas, 1991) and epidemiologic samples (Neylan et al., 1998), while laboratory-based PSG studies have produced mixed results. A recent meta-analysis of 20 studies found that patients with PTSD had more stage 1 sleep, less slow wave sleep, and greater rapid-eye-movement (REM) density (REM activity/ minutes REM sleep) compared to people without PTSD (Kobayashi, Boarts, & Delahanty, 2007).

Given the high frequency of reported sleep disturbances and the hypothesized state of nighttime hypervigilance in subjects with PTSD, it has been proposed that FNE would be prominent in these subjects, particularly in an unfamiliar sleep environment. Two laboratory-based PSG studies comparing first night adaptation effects in PTSD subjects and controls have reported mixed findings (Ross et al., 1999; Woodward, Bliwise, Friedman, & Gusman, 1996b). Ross and colleagues found no differences in adaptation effects in a mixed sample of outpatient and residential treatment PTSD subjects compared to outpatient controls in a laboratory study (Ross et al., 1999). However, increased REM activity and density was observed in PTSD subjects on the first versus the second night. In contrast, Woodward (1996b) found that FNEs in PTSD subjects were dependent on whether the subjects were currently in a residential treatment program versus outpatient treatment. In this laboratory-based study, PTSD inpatients showed decreased FNEs compared to outpatient controls, whereas PTSD outpatients had enhanced FNE compared to outpatient controls (Woodward, Bliwise, Friedman, & Gusman, 1996b). These results suggest that adaptation effects observed in PTSD may reflect enhanced sensitivity to a novel sleeping environment.

As most previous PSG studies in PTSD have been conducted only in the sleep laboratory, it is difficult to discern whether FNEs observed in PTSD represent adaptation to recording equipment, novel sleeping environment, or both. A direct comparison of PSG testing in the two settings would clarify whether the recording context affects the results, allowing for more accurate study and enhanced understanding of PTSD- related sleep disruption.

To date, this is the first study to examine FNE in medically healthy medication-free subjects with PTSD and age-matched controls with two pairs of PSG studies conducted in both home and hospital settings. We hypothesized that both the PTSD group and the control group would have greater FNE in the hospital than at home, and that the PTSD group would have greater FNE compared to controls in night one versus night two of the study in both settings. Finally, we hypothesized that adaptation effects in both groups would be attenuated in the second pair of PSG studies.

Methods

Participants and Procedures

Medically healthy male and female subjects were recruited from internet and newspaper advertisements and from the San Francisco Veterans Affairs Medical Center (SFVAMC) PTSD Outpatient Program. Subjects were recruited as part of a larger study examining the

relationship between sleep architecture and hypothalamic-pituitary-adrenal axis activity in chronic PTSD. All subjects were given details of the study and asked to sign a written informed consent form if they wished to participate. The study protocol and consent form was approved by the Committee on Human Research at the University of California, San Francisco (UCSF).

Overall, 151 subjects were initially consented, 89 enrolled, 19 refused (due to scheduling conflicts), and 43 did not meet study criteria. The final data analysis included 60 subjects; 29 subjects had to be excluded due to equipment failure. Subjects were paid up to \$400 for completion of the study, which included 5 nights of polysomnographic testing.

The PTSD group with complete night 1 and 2 data in both settings use for these analyses consisted of 34 male and female subjects with current chronic PTSD as assessed by the Clinician Administered PTSD Scale (CAPS) (Blake et al., 1995). The CAPS measures frequency and intensity of PTSD-related symptoms, with possible scores ranging from 0 to 136. In a recent review of studies utilizing the CAPS, Weathers and colleagues (2001) proposed the following severity score ranges: 0–19 = Asymptomatic/few symptoms; 20–39 = Mild PTSD/subthreshold; 40–59 = Moderate PTSD/threshold; 60–79 = Severe PTSD symptomatology; > 80 = Extreme PTSD. The PTSD group was mixed with respect to trauma type and severity, and civilian and military trauma; however, all PTSD subjects had symptoms for at least one year in duration and none had been exposed to traumatic events within the past year. The control group was composed of 26 healthy participants, negative for lifetime PTSD or major depression. Subjects were age balanced within gender. In both groups, subjects were excluded if they met criteria for alcohol or substance abuse within the past two years, lifetime criteria for psychotic disorder, bipolar disorder, or obsessive compulsive disorder as assessed by the Structured Clinical Interview for DSM-IV (SCID-P) (Spitzer, Williams, Gibbon, & First, 1996). Medical exclusion criteria included any history of neurologic disease (traumatic brain injury, seizure, hemorrhage, stroke, or other brain injury), current systemic illness affecting central nervous system function, or use of any medication affecting the brain. All subjects were required to be free of any psychiatric medications for at least two months prior to participation. Subjects were asked to abstain from any alcohol the week before the study.

An oximeter (Respironics Cricket) was used to screen for obstructive sleep apnea (OSA). The cutoff criterion for apnea was 10 desaturation events per hour in bed, which has been shown to have a sensitivity of 98% and specificity of 48% in detecting OSA (Sériès, Marc, Cormier, & La Forge, 1993). Subjects who screened positive for OSA were excluded. All subjects were alcohol-free and allowed up to one caffeinated beverage each morning during the sleep recordings.

Subjects completed five nights of ambulatory polysomnography, two in participants' homes and three in a hospital research unit, separated by three nights of sleep in the participant's home without wearing the sleep equipment. The study was counterbalanced such that subjects were randomly assigned to begin the study either at home or in the hospital. Subjects received metyrapone prior to the third night of sleep in the hospital research unit in an investigation of hypothalamic-pituitary adrenal axis activity and sleep. (Metyrapone was not administered during the nights of the study reported here.) The results of the metyrapone study compared nights two and three in the hospital only and have been previously published (Neylan et al., 2003; Otte et al., 2005; Otte et al., 2007). The current study examines the two nights of home sleep recordings, never previously published, the hospital adaptation night, never previously published, versus the second night of sleep recordings in the hospital.

Measures

At baseline, subjects completed a set of subjective measures, including Symptom Check-List-90-Revised (SCL-90-R) (Derogatis, 1994), a standard self-report measure of general psychopathology, and the Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), a self-report measure that provides a subjective assessment of sleep quality, sleep latency, sleep duration, sleep maintenance, sleep disturbances (including nightmares), use of sedative-hypnotics, and daytime energy.

Subjects adhered to a stable sleep-wake schedule at their habitual times during the entire study period. Self-report ratings of subjective sleep quality were obtained for each night of sleep using a 100mm visual analog scale which ranged from “very bad” to “very good” sleep quality. Objective sleep quality was measured with ambulatory polysomnography, using an Oxford MR95 digital recorder. The parameters recorded included an electroencephalogram (EEG) at leads C3 and C4, left and right electrooculograms (EOG), submental electromyogram (EMG), and electrocardiogram (EKG) in accordance with standardized guidelines (Rechtschaffen & Kales, 1968). The EEG and EOG leads were referenced to linked mastoids. All sleep was imported into Pass Plus (Delta Software) analytic software and visually scored in 30-second epochs in accordance to Rechtschaffen and Kales (Rechtschaffen & Kales, 1968).

Sleep architecture was delineated as the percentage of time spent asleep in non-REM (NREM) stages one through four and stage REM. Sleep continuity was measured by calculating sleep maintenance defined as the ratio of total time spent asleep divided by the total recording period between sleep onset and offset. An awakening was defined by EEG arousals lasting 30 seconds or longer. REM periods were defined by at least three minutes of consecutive REM sleep with no less than 30 minutes of NREM sleep separating two REM periods. Polysomnographic measures included total sleep time in minutes, sleep maintenance (percent of actual sleep time between sleep onset and the final awakening), and total time awake after sleep onset in minutes (WASO). REM measures included REM percent (minutes of REM sleep/minutes of total sleep time), REM latency (minutes from sleep onset to first REM period), REM activity (number of rapid eye movements), and REM density (REM activity/ minutes REM sleep).

Statistical Analyses

Differences in demographic characteristics between PTSD subjects and controls were compared using t-tests for continuous variables and chi-square tests for dichotomous variables. Measures of sleep architecture and subjective sleep ratings on the four nights of PSG testing (hospital nights one and two and home nights one and two) were analyzed by a linear mixed model fitting PTSD status as a between-groups fixed effect and time as a within subjects repeated effect. We also included location (home versus hospital) and order (first versus second series of recordings) in the model. Group differences were then examined using t-tests for continuous variables. The relationships between polysomnographic variables and subjective sleep ratings were analyzed by two-tailed Pearson correlations. All values are expressed as mean and standard deviation. A nominal level of significance $\alpha = .05$ was accepted.

Results

Subject Characteristics

The mean age for the PTSD subjects was 42.2 (SD= 10.5) and control subjects was 39.8 (SD=11.3). The mean CAPS score for the PTSD subjects was 62.7 (SD= 18.2), reflecting moderate PTSD symptom severity, compared to .8 (SD= 1.5) in controls. Demographic

characteristics of the PTSD group and the control group are summarized in Table 1. Polysomnographic data from nights one and two in each location are presented in tables 2a and 2b. Main effects and significant interactions of group, night, location, and order on sleep architecture and subjective sleep ratings are summarized in Tables 3a and 3b. As noted above, the nights two and three polysomnography data acquired in the hospital has been reported previously in the male (Neylan et al., 2003) and female (Otte et al., 2007) subsamples.

Polysomnographic data

First night effects

In the overall sample, total sleep time was reduced on night one compared with night two as evidenced by a main effect of night ($F[1, 55.66] = 5.00, p < .029$). As expected, a night $\times$ location effect was seen with respect to total sleep time ($F[1, 55.54] = 13.92, p < .001$), reflecting larger first night effects in the hospital versus the home. Night $\times$ order effects were observed on total sleep time ($F[1, 55.54] = 5.81, p < .019$), demonstrating that the first night effect was more pronounced in the initial nights of the study. Note that the “order” variable refers to the first versus second series of recordings and does not reflect first night changes (night one versus night two within each series).

Group differences

We observed a group $\times$ location $\times$ order effect on total sleep time ($F[1, 55.82] = 7.10, p < .010$), indicating that differences in total sleep time between PTSD and control groups were modified both by location and order. The PTSD group demonstrated no first night changes in sleep duration or architecture in either location. The control group assigned to the “hospital first” condition showed adaptation changes in total sleep time its first night in the hospital ($F[1, 30.00] = 5.77, p < .023$), but not in the subsequent nights of the study at home. The control group assigned to the “home first” condition did not have any FNE, at home or later in the hospital (see Figure 1). Three-way group $\times$ location $\times$ order interactions were seen with respect to sleep maintenance ($F[1, 55.88] = 5.16, p < .027$) and REM density ($F[1, 56.11] = 4.83, p < .032$).

Several PSG findings emerged in the PTSD group whose first recordings took place at home. In that setting, the PTSD group assigned to the “home first” condition had reduced sleep maintenance relative to the “home first” control group ($F[1, 28.93] = 5.17, p < .031$). The “home first” PTSD group also demonstrated higher REM densities on its home nights than the “hospital first” PTSD group ($F[1, 32.21] = 7.60, p < .010$).

Location effects

In the overall sample, participants slept longer at home than in the hospital as evidenced by a main effect of location on total sleep time ($F[1, 56.06] = 5.18, p < .027$) and sleep maintenance ($F[1, 56.23] = 13.80, p < .001$). Three-way group $\times$ location $\times$ order interactions were seen with respect to total sleep time ($F[1, 55.82] = 7.10, p < .010$), sleep maintenance ($F[1, 55.88] = 5.16, p < .027$) and REM density ($F[1, 56.11] = 4.83, p < .032$).

Subjective sleep ratings

Mixed model analyses of subjective sleep ratings showed significant main effects for group ($F[1, 55.13] = 11.85, p < .001$), night ($F[1, 53.72] = 11.97, p < .001$), location ($F[1, 55.11] = 14.30, p < .000$) and order ($F[1, 55.11] = 7.01, p < .011$). Overall, mean subjective sleep ratings in the PTSD group were lower than those seen in the control group. Two-way interactions were seen between night and order ($F[1, 55.10] = 4.29, p = .043$) and between group and order ($F[1, 55.11] = 9.00, p < .004$).

Effect of comorbid depression and substance use disorders

About 29% of the PTSD group (n=10) had current major depressive disorder, and 65% had a past history of substance use disorder (SUD) (n=24). This contrasts with the control group, in which no subjects met criteria for depression, and about 12% (n=3) had a past history of SUD. We calculated the effect sizes of depression and SUD on the first night effect (total sleep time on night 1 versus night 2) in the PTSD and control groups. The effect sizes (Cohen's d) for the first night effect on total sleep time for the PTSD/ depression (+), PTSD/ depression (−), and control groups were .48, .47, and .49, respectively. Thus, comorbid depression did not affect total sleep time in our sample. A past history of SUD also did not appear to affect total sleep time in our study. The observed effect size in the PTSD/ SUD (+) group on total sleep time was .47, compared with .50 in the PTSD/ SUD (−) group, and .48 in the control/ SUD (−) group. In keeping with our results, the "hospital first" control group demonstrated the largest effect size (d= .87).

Discussion

This is the first study to examine the presence of FNE in PTSD outpatients and controls studied in both the hospital and home settings. We expected that the PTSD group would have an exaggerated adaptation response to the novel stressor of hospital PSG testing. Contrary to our prediction, we found that the PTSD group showed no first night changes in sleep architecture, at home or in the hospital. Only the control group assigned to the "hospital first" condition showed first-night changes in total sleep time in the hospital.

The lack of a significant FNE in our outpatient sample of PTSD subjects (e.g. no group $\times$ night effect) differs from the results of other studies examining FNE in PTSD. Ross and colleagues observed increases in REM density in the first REM period and mean REM activity in a mixed sample of inpatients and outpatients with PTSD compared to control subjects (Ross et al., 1999). Woodward and colleagues (1996b) noted more pronounced FNE among PTSD outpatients, and attenuated FNE among PTSD inpatients, compared with outpatient controls (Woodward, Bliwise, Friedman, & Gusman, 1996b). Some investigators have postulated that individuals with PTSD perceive the hospital as "safe" because of the presence of a sleep laboratory technician (Sheikh, Woodward, & Leskin, 2003). In our study, the PTSD group's lack of adaptation response in the hospital, compared with the marked FNE seen in the control group, may reflect the PTSD group's subjective experience of safety in a monitored environment.

It has been hypothesized that individuals with PTSD have "sleep state misperception," often reporting worse sleep than is objectively seen on PSG testing (Klein, Koren, Arnon, & Lavie, 2003; Lavie, 2001). One potential explanation for this discrepancy is that those with PTSD maintain a state of intact vigilance during lighter stages of sleep, which may not be detected on PSG. If PTSD subjects sustain a state of hypervigilance during sleep, they may not demonstrate the adaptation effects to PSG testing seen in controls. PTSD has also been described as a disorder of faulty adaptation of neurobiological systems after exposure to traumatic stressors (Germain, Buysse & Nofzinger, 2008). If the FNE represents a normal adaptation process to the conditions of PSG testing (Schmidt & Kaelbling, 1971), the lack of FNE in the PTSD may be understood as an inability to adapt to the "stressor" of PSG testing. The inability to adapt to the novel stressor of PSG testing has also been proposed in major depression, which has been associated with an attenuated FNE (Mendels & Hawkins, 1966; Toussaint et al., 1995).

In keeping with our hypothesis, neither group showed adaptation effects at home. Previous ambulatory PSG studies have demonstrated attenuated or absent FNE (Coates et al., 1981; Edinger et al., 1997; Sharpley, Solomon, & Cowen, 1988). Our findings indicate that the

practice of excluding first night data from analyses of home PSG studies may not be necessary in groups similar to those studied here.

The study's counterbalanced design allows us to evaluate whether first night effects are due to environment, recording equipment, or both. Although other studies investigating the FNE have directly compared PSG data from home and hospital recordings, ours is the first study to test for differences in sleep duration and architecture between the first and second series of recordings. In the overall sample, a night $\times$ order effect was observed, indicating that previous experience with PSG mitigates adaptation effects. In both groups, subjects demonstrated no re-adaptation FNE in second series of PSG testing in either setting, confirming our hypothesis that the FNE would be reduced in the second pair of PSG recordings. These results are consistent with those of Lorenzo and Barbanoj, who observed in an extended series of laboratory PSG sessions of healthy subjects that adaptation changes only occurred on the "very first night" of testing (Lorenzo & Barbanoj, 2002). In longitudinal sleep studies of groups similar to those studied here, only one adaptation night may be required, as participants habituate to laboratory PSG testing on the initial night of testing.

We observed that "home first" PTSD group had higher REM densities on its home nights than the "hospital first" PTSD group ($F [1, 32.21] = 7.60, p < .010$). It has been hypothesized that disruption of REM sleep mechanisms may underlie the insomnia and nightmares that characterize PTSD (Ross et al., 1999). Elevated REM density in the home PSG recording may reflect conditioned arousal in PTSD subjects when monitored during their habitual sleep environment. However, the finding of elevated REM densities in the "home first" PTSD group must be interpreted with caution, as this group demonstrated elevated REM densities in its second series of hospital-based recordings. It is possible that the group's high REM densities were due to sampling error rather than the setting.

Interestingly, participants with PTSD reported lower ratings of sleep quality than controls, even though there were no overall group differences in any measures of sleep architecture across the four nights. This discrepancy between objective and subjective measures of sleep disturbance is consistent with the findings of other investigators (Klein, Koren, Arnon, & Lavie, 2003; Lavie, 2001) and supports the hypothesis that sleep misperception rather than objective sleep alteration may underlie sleep disturbances in PTSD. Individuals with PTSD have been observed to have a range of negative cognitive biases in many symptom domains (Hembree & Foa, 2000), and thus may report more subjective distress with respect to sleep as well. Another potential explanation is that traditional PSG scoring only provides an overview of sleep architecture and that more subtle abnormalities, like hypervigilance, may be undetected with these measures. It is possible that as a consequence of traumatic exposure, PTSD subjects maintain alertness in light stages of sleep and perceive worse sleep quality than is demonstrated objectively. A third possibility is that our control subjects, who were specifically recruited into a sleep study, represent a biased sample. It is notable that the overall sleep maintenance of the control group was below than expected for a sample free from sleep disorders.

Our results must be interpreted in light of several limitations. As described above, it is possible that controls recruited into our multi-night sleep study may have mild insomnia, although there were no clinical or psychometric manifestations of sleep disorder. The control group exhibited surprisingly poor sleep maintenance and long wake times. Another potential explanation for the lack of group differences is that our medically-healthy, medication-free outpatient PTSD group may reflect a biased sample not reflective of the broad population of PTSD patients. It has been noted previously that PTSD patients who volunteer for sleep studies may have less sleep disruption than those who decline

(Woodward et al., 2007). The PTSD group's mean CAPS score indicates moderate symptom severity, but it is possible that ours is a less acutely disturbed sample than those typically seen in clinical settings.

We considered the possibility that comorbid depression and/or SUD may have affected our findings. As noted in the results section, current major depression or past history of SUD did not affect total sleep time in either the PTSD group or controls. Psychiatric outpatients (Mendels & Hawkins, 1966) and inpatients (Toussaint et al., 1995) with depression have been shown to have attenuated first night effects relative to healthy controls. No studies have investigated the presence of adaptation effects in individuals with substance use disorders. Further research with larger sample sizes would further elucidate the relationship between depression, SUD and adaptation effects.

Finally, PSG studies have demonstrated that adaptation varies significantly with age (Wauquier, van Sweden, Kerkhof, & Kamphuisen, 1991; Edinger, Marsh, McCall, Erwin, & Lininger, 1991), gender (Goel, Kim, & Lao, 2005), psychiatric comorbidities (Saletu et al., 1996), and medical conditions (Le Bon et al., 2003). Future investigations with different study populations may allow us to clarify the relationship between demographic factors, comorbid diagnoses and adaptation effects.

In summary, our finding that outpatients with chronic PTSD demonstrated no FNE supports the retention of first night data in sleep studies of individuals with PTSD. The absence of FNE in either group at home highlights the utility and cost-effectiveness of ambulatory sleep studies. The absence of a re-adaptation phenomenon in the second series of PSG recordings has important implications for longitudinal sleep studies, as it suggests that only the "very first" night of laboratory PSG data may need to be discarded. Finally, home-based PSG studies and treatment trials with interventions targeted at negative cognitive appraisals of sleep may be useful in defining and addressing the sleep disruption seen in PTSD.

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Figure 1.
Total Sleep Time in PTSD and Control Groups in Hospital and Home Settings in the First Series of Recordings

Table 1

Characteristics of Healthy Medication-Free PTSD Subjects and Age-Matched Controls

Measures	PTSD Group (Mean+/-SD)	Control Group (Mean+/-SD)	Contrasts
Age (years)	42.2 +/- 10.5	39.8 +/- 11.3	$t(60) = .84, p = .40$
Gender			
Male	61.8% ($n=21$)	50.0% ($n=13$)	$\chi^2(1) = .83, p = .36$
Female	38.2% ($n=13$)	50.0% ($n=13$)	
CAPS	62.7 +/- 18.2	.8 +/- 1.5	$t(60) = 17.3, p < .0001$
PSQI	11.2 +/- 4.4	4.1 +/- 2.0	$t(60) = 7.60, p < .0001$
SCL-90	1.26 +/- .69	.26 +/- .25	$t(60) = 7.00, p < .0001$
Depression:			
Yes	70.59% ($n=24$)	0	$\chi^2(1) = 9.18, p = .002$
No	29.41% ($n=10$)	100% ($n=26$)	
Past SUD:			
Yes	64.71% ($n=22$)	88.46% ($n=23$)	$\chi^2(1) = 17.14, p < .0001$
No	35.29% ($n=12$)	11.54% ($n=3$)	

Notes. CAPS= Clinician-Administered PTSD Scale; PSQI = Pittsburgh Sleep Quality Index (scores > 5 associated with significant insomnia); SCL-90 = Symptom Checklist-90; Depression= Current major depressive disorder; Past SUD= Past substance use disorder

Table 2

Night 1 versus Night 2 Sleep in Home and Hospital Recordings

PTSD Group (n=34)				
Measures	Home PSG Mean (SD)		Hospital PSG Mean (SD)	
	Night 1	Night 2	Night 1	Night 2
Total sleep time(min)	387.84(73.93)	370.94(91.57)	332.21(87.01)	367.87(61.02)
Sleep maintenance	.84(.13)	.85(.11)	.77(.18)	.83(.12)
Subjective rating	55.42(26.76)	59.61(27.38)	48.21(27.03)	56.09(26.94)
WASO (min)	77.54(69.56)	75.31(72.75)	98.38(81.15)	79.10(57.95)
REM density	4.08(2.27)	3.85(1.99)	4.04(1.74)	4.42(2.03)
REM percent	23.14(6.90)	20.39(7.55)	19.99(6.17)	21.79(6.63)
REM latency	95.17(64.13)	83.42(66.63)	93.34(45.30)	83.37(58.44)
Control Group(n=26)				
Measures	Home PSG Mean (SD)		Hospital PSG Mean (SD)	
	Night 1	Night 2	Night 1	Night 2
Total sleep time(min)	386.56(83.98)	395.27(68.49)	352.48(109.86)	404.04(83.06)
Sleep maintenance	.87 (.10)	.88(.10)	.81(.13)	.84(.11)
Subjective rating	73.12(24.80)	83.96(23.09)	54.60(28.42)	72.64(27.58)
WASO (min)	62.00(80.75)	51.50(35.66)	80.29(56.83)	80.12(53.91)
REM density	4.55(1.96)	4.30(1.98)	4.17(1.99)	3.88(2.08)
REM percent	21.56(5.41)	22.44(6.68)	20.17(6.87)	2.25(6.24)
REM latency	95.04(61.53)	99.32(47.63)	89.12(54.12)	97.15(57.35)

* Indicates first night effects (significant difference between nights 1 and 2), $p < .05$. WASO=Wake After Sleep Onset, REM percent= REM time (minutes)/ Total sleep time (minutes)

Table 3**Table 3a. Main effects of Group, Night, Location and Order on Sleep Parameters**

Measures	Group (<i>F,p</i>)	Night (<i>F,p</i>)	Location (<i>F,p</i>)	Order (<i>F,p</i>)
Total sleep time(min)	1.40, .242	5.00, .029*	5.18, .027*	.06, .812
Sleep maintenance	.98, .327	3.10, .084	13.80, .001*	.16, .691
Subjective rating	11.85, .001*	11.97, .001*	14.30, .001*	7.01, .011*
WASO (min)	.83, .367	1.20, .277	6.65, .013*	.11, .741
REM density	1.71, .196	.05, .828	.04, .852	1.15, .287
REM percent	.01, .915	.50, .484	1.56, .217	1.39, .244
REM latency	.121, .729	.272, .604	.006, .939	3.20, .081

Table 3b. Significant Interactions Between Group, Night, Location and Order on Sleep Parameters

Measures	Night × Loc (<i>F,p</i>)	Night × Ord (<i>F,p</i>)	Loc × Ord (<i>F,p</i>)	Grp × Ord (<i>F,p</i>)	Grp × Loc × Ord (<i>F,p</i>)
Total sleep time(min)	13.92, .000	5.81, .019			7.10, .010
Sleep maintenance					5.16, .027
WASO (min)			4.00, .050		4.51, .038
REM density					4.83, .032
Subjective rating		4.29, .043		9.00, .004	

* Indicates $p < .05$, WASO= Wake After Sleep Onset, REM percent= REM time (minutes)/ Total sleep time (minutes)

Grp= Group, Ord= Order, Loc= Location