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A Prospective Evaluation of PTSD Symptoms following CPAP Treatment for Sleep-disordered Breathing in Veterans

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A PROSPECTIVE EVALUATION OF PTSD SYMPTOMS FOLLOWING CPAP TREATMENT FOR SLEEP-DISORDERED BREATHING IN VETERANS

by

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Dedication

For my family:

Jim, Lou Ann, & Colleen Monahan

Without your love and support this wouldn't have been possible.

Thank you.

Abstract

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Previous research has observed elevated rates of OSA observed in individuals with PTSD compared to the general population. Retrospective studies suggest that successful treatment of OSA in individuals with PTSD is related to reductions in nightmares and overall PTSD symptom severity. The purpose of the current study was to extend this research by prospectively examining PTSD sympomatology in a sample of Veterans initiating treatment for OSA. Participants were 47 Veterans presenting to a VAMC Neurology Sleep Clinic for overnight polysomnography. Veterans were eligible if they were: (a) diagnosed with OSA; (b) received continuous positive airway pressure (CPAP) treatment; and (c) had a minimum score of 25 on the baseline administration of the PCL. The majority of the sample were male (n = 42; 89.4%) and Caucasian (n = 23; 48.9%) or African American (n = 22; 46.8%), with a mean age of 53.5 years. Veterans completed self-report questionnaires across two pre-treatment and two post-treatment (two weeks and four weeks from treatment initiation) time points. A 2 (treatment compliance status) x 4 (time) mixed model repeated measures analysis was conducted on PTSD symptom severity as measured by the PCL administered at each time point. A statistically significant compliance status x time interaction emerged, (F(3, 102.15) = 5.66. p = .001) such that CPAP-compliant Veterans reported a statistically significant reduction in PTSD symptoms from pre to post-treatment, whereas CPAP non-compliant Veterans did not. These findings suggest that successful treatment of a physical sleep disorder like OSA is associated with a subsequent reduction of posttraumatic distress.

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The diagnosis of posttraumatic stress disorder (PTSD) denotes psychological symptoms across three broad domains: re-experiencing, avoidance/numbing, and hyperarousal (DSM-IV-TR; APA, 2000). Although not unique to PTSD, sleep problems are a salient aspect of this diagnosis and are characterized by persistent nightmares as well as onset and/or maintenance insomnia (Ross, Ball, Sullivan, & Caroff, 1989). These sleep-related symptoms are conceptualized as distinct from each other, whereby the occurrence of nightmares ("recurrent distressing dreams") is designated as a re-experiencing symptom and sleep initiation/maintenance difficulties reflect hyperarousal (APA, 2000). Individuals with PTSD frequently report sleep initiation difficulties, frequent mid-sleep awakenings, awakenings accompanied by panic or startle, and nightmares (Harvey, Jones, Schimdt, 2003; Mellman, Kulick-Bell, Ashlock, & Nolan, 1995).

Although sleep disturbance in the context of PTSD may occur as a symptom of the disorder, it may also occur as an independent phenomenon, in some cases pre-dating other PTSD symptoms. And the relationship between PTSD and dysfunctional sleep processes appears to be reciprocal. It is well documented that individuals with PTSD are at elevated risk for sleep disturbance (see reviews: Germain, 2009; Harvey et al., 2003; Maher, Rego, & Asnis, 2006; Pillar, Malhotra, & Lavie, 2000) with the severity of disturbed sleep directly proportional to the level of PTSD symptom severity (Germain, Buysse, Shear, Fayyad, & Austin, 2004; Krakow et al., 2001). A large majority (70-91%) of individuals with PTSD report some sleep-related dysfunction (Maher et al., 2006). Disturbed sleep following a traumatic experience has been found to predict future PTSD

onset (Koren, Arnon, Lavie, & Klein, 2002) and is associated with more severe PTSD symptomatology overall (Krakow et al., 2004; Lavie, 2006; Maher et al., 2006). It appears then that sleep dysfunction can manifest as both an antecedent to and a consequence of PTSD; however, the majority of extant research has focused on sleep disturbance as a result of PTSD.

Despite extensive research on sleep disturbance and PTSD, one factor that has received little attention in the literature to date is the co-occurrence of sleep-disorder breathing (SDB) in individuals with PTSD. The SDB label has been used to refer to a group of organic sleep disorders which cause a repetitive disruption to the respiratory process during sleep. Relative to the general population, individuals with PTSD seem to be at higher risk for SDB (Bixler et al., 2001; Maher et al., 2006). Unfortunately, few studies that have examined sleep-related difficulties in individuals with PTSD have concurrently examined SDB as a possible antecedent (Pillar et al., 2000). Interestingly, retrospective studies suggest that successful treatment of SBD in individuals with PTSD is associated with an improvement in nightmares and overall PTSD symptoms (Krakow et al., 2000; Youakim, Doghramji, Schutte, 1998); however no studies have examined prospectively whether changes in PTSD symptomatology occur following successful treatment of SDB.

Disturbed Sleep and PTSD

As previously noted, higher rates of self-reported sleep disturbance have been well-documented among individuals with PTSD compared to those without. Sleep problems have been observed in a number of studies of individuals diagnosed with PTSD including combat veterans (Inman, Silver, & Doghramji, 1990; Neylan et al., 1998),

sexual assault survivors (Foa, Riggs, & Gershuny, 1995), motor vehicle accident (MVA) survivors (Harvey & Bryant, 1998; Koren, Arnon, Lavie, & Ehud, 2002), and survivors of natural disaster (Mellman et al., 1995; Sharan, Chaudhary, Kavathekar, & Saxena, 1996). Although many studies have employed self-report methods to assess sleep disturbance, the gold standard for measuring sleep processes and diagnosing sleep disorders is polysomnography (PSG). PSG uses psychophysiological instruments to measure brain activity during sleep. PSG provides objective data regarding sleep episode characteristics such as total sleep time, number of awakenings, and time spent in the various stages. Although self-report studies have overwhelmingly suggested that PTSD is associated with various sleep-related disturbances, studies that have used PSG to examine sleep processes in PTSD have been less consistent in their findings (Breslau et al., 2004; Klein, Koren, Arnon, & Lavie, 2002). In fact, the majority of studies using PSG have not found a statistically significant difference in the rates of sleep disturbances for individuals with PTSD compared to those without (Breslau et al., 2004; Fuller, Waters, & Scott, 1994; Klein, Koren, Arnon, & Lavie; 2002; Lavie, Katz, Pillar, & Zinger, 1998; Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002; Mellman, David, Kulick-Bell, Hebding, & Nolan, 1995; Neylan et al., 2003; & Otte et al., 2007).

Despite the apparent lack of congruence among studies using objective vs. subjective assessment, sleep dysfunction has long been considered a hallmark feature of PTSD pathology (Ross et al., 1989). As previously noted, the relationship between disturbed sleep and PTSD appears to be reciprocal. Previous research has suggested that disturbed sleep in the wake of trauma is a risk factor for future PTSD development and more severe PTSD symptomatology. Along these lines, Foa and colleagues (1995) found

the presence of both sleep disturbance and nightmares following trauma predicted PTSD in a sample of 158 female assault victims. The presence of trauma-related nightmares following life-threatening injury was also associated with PTSD symptoms severity in a sample of 60 patients hospitalized in a regional trauma unit (Mellman, David, Bustamante, Torres, & Fins, 2001).

Although the implications of these findings are limited to some extent by cross-sectional design, other prospective research has similarly demonstrated that disturbed sleep is predictive of PTSD onset and PTSD symptom severity. Koren and colleagues (2002) assessed 102 MVA survivors longitudinally for one year following their MVA. Their findings revealed that a majority of the survivors experienced sleep difficulties immediately following the accident; however, the severity of sleep difficulty was predictive of later PTSD diagnosis (Koren et al., 2002). A comparable finding was reported in a separate sample of MVA survivors such that both disturbed sleep and the presence of nightmares one month following trauma predicted PTSD development six months later (Harvey & Bryant, 1998). A more recent prospective study demonstrated that sleep disturbance immediately prior to trauma exposure also increased risk for the future development of PTSD (Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2010). In this study, a sample of traumatically injured hospital patients (N = 1033) were assessed at the time of injury for current psychiatric disorders, including sleep dysfunction. Results found that pre-trauma disturbed sleep predicted future onset of PTSD after controlling for age, gender, mechanism of traumatic injury, severity of injury, and prior psychiatric disorder.

Overall, the research suggests that disturbed sleep is common among individuals with PTSD and may contribute to the maintenance of PTSD symptoms. Given the apparent reciprocal nature of this relationship, an important goal for researchers is to better understand the relation between disturbed sleep and PTSD. One factor that has been largely excluded from this research base is the co-occurrence of sleep-disordered breathing (SDB) in individuals with PTSD. Elevated rates of SDB have been observed among individuals with PTSD (Krakow et al., 2000; Krakow et al., 2001); however, many studies that have examined sleep-related difficulties in individuals with PTSD have excluded individuals on the basis of a sleep-disordered breathing diagnosis. The exclusion of SDB as a source of sleep disruption is important to recognize as individuals with PTSD likely experience both psychogenic and organically manifested sleep disturbance. Unfortunately, large proportions of individuals with a sleep-breathing disorder are unaware of the underlying condition (Punjabi, 2008) and attribute ongoing sleep difficulties to other factors. SDB may be an important factor contributing to the high rates sleep disturbance reported, but not objectively observed in individuals with PTSD, as well as a potent risk factor increasing a trauma survivors' vulnerability to develop clinically significant PTSD symptoms.

Sleep Disordered Breathing

Sleep-disordered breathing refers to a repetitive disruption of the respiratory process during sleep. This disruption is generally attributed to an obstruction of the airway and each interruption, referred to as an apnea, restricts airflow resulting in one or more consecutively missed breaths. Although not all SDB is associated with subsequent negative consequences, the International Classification of Diseases (ICD-10) designates

that clinically significant SDB is defined as five or more episodes of apnea per hour of sleep (Flemons, 2002). When this clinically significant sleep-disordered breathing is accompanied by snoring as well as daytime sleepiness or fatigue the individual is at risk for sleep apnea syndrome. The most prevalent form of sleep apnea is obstructive sleep apnea (OSA). OSA is characterized by a complete or partial obstruction to the upper respiratory tract during sleep that occurs despite adequate respiratory effort. Common risk factors for OSA include increasing age, obesity, large neck circumference, craniofacial abnormalities, and genetic predisposition (Levitzky, 2008; Punjabi, 2008). For some individuals suffering from OSA, the sleep architecture is significantly altered, including almost complete deprivation to rapid eye movement (REM) stage sleep as well as stage 3 and stage 4 (deep) sleep due to repeated mid-sleep awakenings. Consequently, individuals with OSA experience daytime sleepiness and fatigue; however, they may be unaware of the cause.

Obstructive sleep apnea is associated with a range of negative outcomes. In addition to fragmented sleep architecture and daytime fatigue, individuals suffering from OSA are at risk for impaired memory, cognitive dysfunction, and significant medical conditions including hypertension, obesity, and stroke (Phillipson, 1993; Wright, Johns, Watt, Melville, & Sheldon, 1997). Given the health risks associated with OSA, early identification and treatment is essential. The most effective form of treatment for OSA is continuous positive airway pressure (CPAP). This technology delivers pressurized air to a mask that covers the nose which is attached to the face by head straps. The continuous positive pressure prevents the airway from collapsing during inhalation. The therapeutic effect of treatment with CPAP is immediate and has been demonstrated with average

nightly usage between 3.4-4.5 hours (Wright et al., 1997). In addition to the immediate reversal of apneic episodes and increased density of REM stage sleep, treatment with CPAP is associated with more distal benefits including a reduction in daytime somnolence, improved mood, and increased alertness (Engelman et al., 1999; Jenkinson, Davies, Mullins, & Stradling, 1999; Means et al., 2003; Schwartz, Kohler, & Karatinos, 2005).

Epidemiological research has estimated that more than 4% of adults suffer from sleep apnea syndrome (Young et al., 1993). The majority of this published literature, however, has examined the prevalence rates in age selected samples given the presence of sleep apnea has been observed more frequently in older adult men (Ancoli-Israel, Kripke, Mason, & Kaplan, 1985; Bixler, Vgontzas, Have, Tyson, & Kales, 1998). When considering obstructive sleep apnea more specifically, few studies have investigated its prevalence in the general population. One reason for this lack of research is that a formal diagnosis of OSA can only be established through an overnight polysomnography assessment. Punjabi (2008) conducted a literature review of available population-based studies on the prevalence of OSA and estimated the prevalence of OSA is approximately 3 to 7% for adult men and 2 to 5% for adult women. One study that used overnight PSG to examine a general population sample of men (n = 741) aged 20-100 found OSA occurred in 3.3% of their sample with the highest prevalence (5%) occurring for ages 45-64 (Bixler et al. 1998).

Apneic events associated with OSA can occur during any stage of sleep but more commonly occur during REM stage sleep. REM stage sleep is characterized by an increase in EEG activity which includes low amplitude-high frequency brain waves,

sporadic rapid eye movement, and muscle paralysis (Carskadon & Dement, 2000). During REM stage sleep the muscles of the upper airway become hypotonic which can lead to a partial or complete obstruction that disrupts the flow of air (Strollo & Rogers, 1996; Youakim et al., 1998). The propensity of apneic episodes to occur during REM sleep in OSA results in REM stage sleep fragmentation and an overall decrease in the percentage of REM stage sleep (Strollo & Rogers, 1996).

Sleep Disordered Breathing and PTSD

Cross-sectional data suggest a strong association between sleep-disordered breathing and PTSD (Krakow et al., 2000; Krakow et al., 2001; Orr et al., 2010; Youakim et al., 1998). For example, an examination of 156 sexual assault survivors with PTSD found approximately 55% reported symptoms consistent with a positive screen for SDB (Krakow et al., 2000); however, in that study, the investigators did not confirm SDB through polysomnographic assessment. A similar rate (54%) was reported for a sample of patients with war-related PTSD who did undergo overnight polysomnography (Dagan, Lavie, Bleich, 1991). In this study 13 of the 24 patients exhibited a pattern of respiratory disturbance characteristic of SBD. A second study that utilized overnight polysomnography to detect SDB (Krakow et al., 2004) found a slightly lower prevalence (50%) rate in a sample of 78 fire evacuees seeking treatment for PTSD. A more recent study analyzed the records of 80 soldiers (89.6% men; mean age = 37.7 years) who recently returned from combat deployment and were diagnosed with PTSD (Orr et al., 2010). Of the 80 soldiers, 58 underwent overnight polysomnography and 61% were subsequently diagnosed with obstructive sleep apnea. Finally, in an uncontrolled study examining crime victims (N = 44) with nightmares and PTSD, Krakow and colleagues

(2001) found approximately 50% of the sample met diagnostic criteria for OSA. Taken together, these data support the notion that SDB and PTSD frequently co-occur, and that individuals with PTSD are at higher risk for SDB and OSA. Unfortunately, these studies are limited by small samples and varying methodologies. Further research is needed to provide a more accurate prevalence rate of these disorders among individuals with PTSD.

Potential pathways linking SDB to PTSD. The relationship between SDB or OSA and PTSD has not been directly examined; however, similarities across several lines of research have led to hypotheses about mechanisms potentially underlying this relationship. The first of these hypotheses concerns REM stage sleep dysfunction as a mechanism explaining co-morbid OSA and PTSD. This hypothesis posits that disturbed sleep among individuals with PTSD involves altered REM sleep accrual or dysfunctional REM sleep processes (Ross et al., 1989). Given the strong association between apneic episodes and REM stage sleep dysfunction, the presence of OSA may be particularly relevant to the development or exacerbation of PTSD symptoms. Dysfunctional REM sleep mechanisms have been implicated as a possible pathway linking sleep dysfunction to PTSD, although this theory has not been subjected to empirical scrutiny (Ross et al., 1994). Some evidence suggests individuals with PTSD experience lower percentages of REM stage sleep than do individuals without PTSD (Glaubman, Mikulincer, Porat, & Wasserman, 1990; Habukawa et al., 2003). Other studies have observed greater REM density and more rapid REM onset latency in individuals with PTSD (Mellman et al., 1993; Ross et al., 1989), which may result from previous episodes of REM sleep dysfunction. Regardless, dysfunctional REM stage sleep has been observed in individuals with OSA and may be a risk factor for PTSD symptoms given that awakenings from

REM stage sleep have been associated with a higher rate of reported nightmares and associated physiological arousal (Mellman et al., 1995; Ross et al., 1989; Ross et al., 1994).

Another potential pathway linking OSA to PTSD pertains to the possibility that similar maladaptive health behaviors are prevalent among individuals with OSA and individuals with PTSD. In particular, research has demonstrated strong associations between obesity and both PTSD and OSA. As previously mentioned, obesity has been associated with increased risk for OSA (Levitzky, 2008; Young et al., 1993), and it has also been linked to PTSD. For example, a nationally representative survey of approximately 13,000 individuals examined the association of obesity across mental disorders and found that PTSD was most strongly associated with obesity (Scott, McGee, Wells, Oakley-Browne, 2008). This association appears particularly pronounced among veterans. A strong association between PTSD and obesity was observed in a large sample of female veterans (n = 1259) such that the veterans who had screened positive for PTSD concurrently demonstrated higher body mass index (BMI) than veterans not screening positive (Dobie et al., 2004). Parallel findings were observed in a sample of male veterans with PTSD such that the rate of obesity in veterans (46.5%) exceeded the national average by 50% (Vieweg et al., 2006). Given the high rates of obesity found in samples of individuals with PTSD, and its associated risk for OSA; individuals with PTSD may be at greater risk to develop OSA due to poor health behaviors which lead to obesity.

Despite the lack of a more comprehensive understanding between OSA and psychiatric conditions like PTSD, the strong association observed between the two

conditions has led to an increase in empirical inquiry. More recent research in this area has examined whether or not successful treatment of OSA could result in amelioration of PTSD symptoms more generally. If such a relationship was observed between successful treatment of OSA and reduction in PTSD symptoms, those data might support the notion that OSA contributes to the persistence of PTSD. No published investigation has prospectively examined changes in PTSD symptoms following treatment for sleep-disordered breathing, but several studies have examined this relationship retrospectively. For example, Krakow and colleagues (2000) examined existing hospital records of 23 patients treated with CPAP for sleep-disordered breathing that also presented with co-morbid nightmares or PTSD. Post-treatment follow-up of these individuals revealed that those patients that had maintained CPAP treatment reported a 93% improvement in sleep and daytime functioning compared to a 33% improvement for patients who had not complied with treatment. In addition, CPAP compliant patients reported an 85% improvement in nightmares and 75% improvement in PTSD symptoms compared with a 10% and 45% worsening in symptoms respectively for the non-treatment compliant individuals.

Similar results were reported in a single case evaluation of a 42-year male Vietnam veteran with co-morbid PTSD and OSA (Youakim et al., 1998). In this instance, the veteran was treated with CPAP for OSA and experienced immediate improvements in sleep quality and quantity and an overall decrease daytime sleepiness. At 4-month follow-up the veteran indicated considerable improvement in PTSD symptoms including a decrease in nightmare frequency (from nightly to once a month) and intensity,

diminished startle response, fewer re-experiencing symptoms. In addition the veteran reported decreased frequency of re-experiencing symptoms.

Although not specific to PTSD, several studies were identified that examined changes in depressive symptoms following CPAP treatment for obstructive sleep apnea (Borak, Cieslicki, Koziej, Matuszewski, & Zielinkski; 1996; Engleman et al., 1999; Engleman, Cheshire, Deary, & Douglas, 1993; Means et al., 2003; Schwartz et al., 2005). For example, in a hospital sample (N = 50) newly diagnosed with OSA, the initiation of CPAP treatment was associated with a reduction in self-reported depressive symptoms 4-6 weeks following treatment initiation (Schwartz et al., 2005). Similar findings were reported for a sample (n = 21) of patients diagnosed with OSA (Engleman et al. 1996) in which participants that "complied well" (n = 14) with CPAP treatment (i.e., > 4.5hrs./night) reported a significant reduction in depression score compared to individuals (n = 7) utilizing their CPAP less. A third study identified examined 39 hospital outpatients diagnosed with OSA following clinical evaluation with overnight polysomnography. Results showed a statistically significant decrease in depressive symptoms for the overall sample 3-month post treatment initiation. Of note, CPAP compliance in this investigation was not found to be a significant predictor of change in depressive symptoms; however, the authors noted the analyses approached statistical significance. In the only study to implement a randomized placebo-controlled design, Engelman and colleagues (1999) prospectively monitored depression symptoms in 34 patients presenting with mild OSA. Following random assignment to either CPAP or placebo CPAP, they found patients utilizing CPAP reported significantly reduced depression symptoms. Finally, one study was identified that failed to find a significant

change in self-reported depressive symptoms following CPAP treatment for OSA (Borak et al., 1996). This study assessed depressive symptoms in 20 men diagnosed with severe OSA prior to CPAP treatment and again 3-months and 12-months following CPAP initiation. Unfortunately, this study does not provide information on CPAP utilization across the follow-up period, so it is unclear if the lack of change in depressive symptoms was associated with CPAP compliance/noncompliance, severity of OSA, or a combination of both factors. Given the lack of research examining the association between PTSD and OSA, in addition to the symptom overlap for PTSD and depression diagnoses, these studies provide more general support for the idea that successful treatment OSA may be associated with improvements in concurrent psychiatric functioning.

Although it is difficult to draw conclusions from this small literature base, this research highlights the possibility of improvement in psychological symptoms following successful treatment of co-morbid sleep disorders. To further investigate this potential relationship, the current study prospectively monitored PTSD symptoms in veteran sample initiating CPAP treatment for obstructive sleep apnea.

Hypotheses

The purpose of this study was to prospectively examine the associations between CPAP treatment for obstructive sleep apnea and change in posttraumatic stress symptoms in a sample of veterans. This study monitored change in posttraumatic stress symptoms, subjectively rated indices of sleep quality and sleep difficulties, and trauma related nightmares following OSA treatment implementation. The study design is quasi-experimental in nature, specifically a single group, pretest-posttest design that included

two pre-treatment observations and two post-treatment observations. A nonequivalent dependent variable was included, which allowed for the novel examination of CPAP treatment on PTSD symptoms and other aspects of sleep functioning in the absence of a true experimental design. In particular, the aims and corresponding hypotheses of the study were as follows:

1. To examine whether participants who complied with CPAP treatment for OSA realized a significant change in their posttraumatic stress symptoms compared to pre-treatment severity.

   Hypothesis 1: We predicted that participants who were compliant (reporting an average of at least 4 hours/night over the post-treatment monitoring period) with CPAP treatment would report a statistically significant reduction in PTSD symptoms during post-treatment follow-up compared to pretreatment symptom severity.

2. To examine whether participants who complied with CPAP treatment for OSA realized a significant improvement across indices of sleep quality and sleep difficulties

   Hypothesis 2a. We predicted that participants who were compliant (reporting an average of at least 4 hours/night over the post-treatment monitoring period) with CPAP treatment would report a statistically significant increase in overall sleep quality during post-treatment follow-up compared to pretreatment symptom severity.

   Hypothesis 2b. We predicted that participants who were compliant (reporting an average of at least 4 hours/night over the post-treatment

monitoring period) with CPAP treatment would report a statistically significant decrease in PTSD-related sleep dysfunction during post-treatment follow-up compared to pretreatment symptom severity.

3. To examine whether participants who complied with CPAP treatment for OSA realized a significant improvement in nightmare-related distress compared to pre-treatment report.

   Hypothesis 3. We predicted that participants who were compliant (reporting an average of at least 4 hours/night over the post-treatment monitoring period) with CPAP treatment would report a statistically significant decrease in nightmare-related distress during post-treatment follow-up compared to pretreatment symptom severity.

4. To examine whether participants who complied with CPAP treatment for OSA realized a significant improvement in daytime sleepiness compared to pre-treatment report.

   Hypothesis 4. We predicted that participants who were compliant (reporting an average of at least 4 hours/night over the post-treatment monitoring period) with CPAP treatment would report a statistically significant decrease in daytime sleepiness during post-treatment follow-up compared to pretreatment symptom severity.

Method

Participants

Of the 489 veterans initially invited to participate, 70 were enrolled and completed the baseline assessment. Of the 70 that completed the baseline assessment, 51

participants were eligible for further participation (see Procedures for more detailed information); however, only 47 completed at least one post-treatment follow-up assessment. The final sample consisted of 47 veterans, who presented to the Neurology Sleep Clinic at the Memphis Veteran Affairs Medical Center (VAMC) and at a minimum completed the baseline assessment and one post-treatment assessment. The majority of the sample was male (*n* = 42; 89.4%) and ages ranged from 31 to 69 years old (*M* = 53.53; *SD* = 10.27). A majority of the sample described themselves as either White/Caucasian (*n* = 23; 48.9%) or Black/African American (*n* = 22; 46.8%) with one individual (2.1%) selecting multiple racial descriptors and one (2.1%) declining to specify. Just over half the sample (55.3%; n = 26) reported being involved in a relationship with 24 participants currently married and two participants in a relationship with a member of the opposite sex. Of the remaining participants, seven (14.9%) reported they were currently single, 13 (27.7%) reported being divorced, and one participant (2.1%) was widowed. CPRS medical record review found 31.9% (n = 15) of participants had PTSD listed as an active diagnosis. Baseline sample characteristics are presented in Table 1.

Table 1

Baseline Sample Demographics

	Total Sample	CPAP-compliant	CPAP Non-compliant	*t*-statistic (*df*)	χ^2
N	47	25	22		
Age - *M* (SD)	53.53 (10.27)	53.84 (11.19)	53.18 (9.38)	*t* (45) = -.22	
Gender – (%)					
Male	42 (89.4)	21 (84.0)	21 (95.5)		.00
Female	5 (10.6)	4 (16.0)	1 (4.5)		1.80
Race/Ethnicity - (%)					
White or Caucasian	23 (48.9)	14 (56.0)	9 (40.9)		1.07
Black or African American	22 (46.8)	10 (40.0)	12 (54.5)		.99
Other	2 (4.3)	1 (4.0)	1 (4.5)		.01
Relationship Status - (%)					
Single	7 (14.9)	3 (12.0)	4 (18.2)		.35

*$p \leq .05$. **$p \leq .01$.

(table continues)

Table 1 (continued)

Baseline Sample Demographics

	Total sample	CPAP-compliant	CPAP Non-compliant	*t*-statistic (*df*)	χ^2
Not married – in relationship (opposite sex)	2 (4.3)	1 (4.0)	1 (4.5)		.01
Married	24 (51.1)	13 (52.0)	11 (50.0)		.02
Divorced	13 (27.7)	8 (32.0)	5 (22.7)		.50
Widowed	1 (2.1)	0 (0.0)	1 (4.5)		1.16
BMI - *M* (SD)	33.58 (5.31)	33.91 (4.74)	33.20 (6.01)	*t* (44) = -.45	
AHI - *M* (SD)	40.12 (28.36)	38.51 (29.17)	42.14 (27.95)	*t* (43) = .42	
Active Medical Diagnoses - *M* (SD)	7.84 (4.19)	8.00 (4.40)	7.65 (4.02)	*t* (43) = -.28	
CPRS Diagnosed PTSD – (%)	15 (31.9)	9 (36.0)	6 (27.3)		.18

*$p \leq .05$. **$p \leq .01$.

Measures

Eligible veterans who consented to participate completed a battery of questionnaires that assessed various aspects of mental and physical health functioning including traumatic event exposure, PTSD symptom severity, aspects of sleep and sleep-related dysfunction, nightmares, and daytime functioning. Participants also reported on their coping styles, physical health, and personality characteristics. A participant's level of CPAP compliance was assessed using a self-report daily sleep diary that included specific assessment of nightly CPAP usage. Participants' medical records were also reviewed to extract body mass index (BMI; as calculated in the Neurology Sleep Clinic polysomnographic report), active comorbid medical or psychiatric diagnoses, and 3-month CPAP SmartCard data (as an objective index of CPAP compliance after approximately 3-months of use). For a complete list of measures administered at each time point refer to Table 2.

Table 2

Timeline of Measurement Administration

	Baseline Screening	Pre-treatment – 1 Prior to CPAP*	Post-treatment – 1 2-weeks post CPAP	Post-treatment – 2 1-month post CPAP
Demographics	X			
The Life Events Checklist	X			
PTSD Checklist – Stressor Specific	X	X	X	X
Pittsburgh Sleep Quality Index	X	X	X	X
Epworth Sleepiness Scale	X	X	X	X
Nightmare Distress Questionnaire	X	X	X	X
Daily Sleep Diary (Compliance)			X	X
Short Form Health Survey 12	X	X	X	X
Brief COPE	X			
Big Five Inventory	X	X	X	X

Note. * Pre-treatment 1 was completed as close to receipt of CPAP treatment, but prior to treatment initiation.

Measures of Trauma Exposure and Post-traumatic Stress Disorder.

The Life Events Checklist (LEC; Gray, Litz, Hsu, & Lombardo, 2004). The LEC is a 17-item self-report measure designed to screen for lifetime exposure to potentially traumatic events. For each item, the participant is instructed to indicate whether the event happened to them personally, if they witnessed the event, if they learned about the event, if they are unsure the item applies, or the item does not apply to them. For the purposes of this project, a brief follow-up assessment was conducted to obtain more information about the most salient traumatic experience identified to verify the trauma met Criterion A trauma exposure as specified in DSM-IV-TR (APA, 2000).

PTSD Checklist-Stressor - Specific Version (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993). The PCL is a 17-item measure that directly corresponds to the DSM-IV-TR (APA, 2000) criteria for PTSD. The PCL utilizes a 5-point Likert scale ranging from 1 ("*not at all*") to 5 ("*extremely"*) and instructs participants to respond to questions regarding the level of distress that was experienced related to each PTSD symptom over the past month. The stem question "*The event you experienced was:"* is specific to the PCL-S version and was pre-populated for all assessment time points based on the worst traumatic event description provided during the preliminary (baseline) trauma assessment. A full PTSD symptom severity score can be derived by totaling the responses from all 17-items (range: 17-85) and specific PTSD symptom subscale scores can be derived for the cluster B ("re-experiencing"), cluster C ("avoidance/numbing"), and cluster D ("hypeararousal") as defined by DSM-IV-TR (APA, 2000). The PCL has demonstrated strong psychometric properties in other studies of PTSD (Weathers et al., 1993; Yeager, Magruder, Knapp, Nicholas, & Frueh, 2007)

and is well-validated in veteran samples (Grieger, Kolkow, Spira, & Morse, 2007; Keen, Kutter, Niles, Krinsley, 2008; Pietrzak, Johnson, Goldstein, Malley, & Southwick, 2009). Previous research has found the PCL demonstrated sound test-retest correlation coefficients of .88 after one week and .68 after two weeks (Ruggiero, Del Ben, Scotti, & Rabalais, 2003). For the purposes of this study a screening cut-off score of ≥ 25 was used to identify participants who are experiencing at least mild PTSD symptoms. This is the suggested cut-off score when screening for PTSD in a primary care sample of veterans (Prins, Kimerling, Yeager, & Magruder, 2010). For the purposes of this study, the PCL-S instructions were modified for the follow-up assessments, instructing participants to respond to questions in regard to their experience over the previous two weeks.

Measures of Sleep, Sleep Disturbance, Nightmares, and Daytime Functioning

Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI is a 19-item self report measure designed to assess sleep quality and includes indices of sleep duration, sleep latency, and the frequency and severity of sleep difficulties. The PSQI utilizes a 4-point Likert-type scale ranging from 0 ("*not during the past month*") to 3 ("*three or more times a week*"). The PSQI instructs participants to respond to questions retrospectively about their experience in the past month and to reply to the questions in a manner that is consistent with the majority of days in the previous month. The PSQI can be used to derive seven component scores (sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction) which are summed together to yield a Global Sleep Quality Index score (range: 0-21) with higher scores indicating more sleep

difficulties and lower sleep quality. Qualitatively, a Global Sleep Quality Index of 5 or greater indicates a "poor" sleeper (Buysse et al., 1998). The PSQI has demonstrated good test–retest reliability, validity, and internal homogeneity (Buysse et al., 1989). Using a cut-score of 5, the PSQI has demonstrated both high sensitivity (89.6%) and specificity (86.5%; kappa = 0.75) in distinguishing good from poor sleepers (Buysse et al., 1989). For this study, the PSQI instructions were modified for the follow-up assessments, instructing participants to respond to with reference to the previous two weeks.

Pittsburgh Sleep Quality Index Addendum for PTSD (PSQI-A; Germain, Hall, Krakow, Shear, Buysse, 2005). The PSQI-A consists of seven self-report items that assess the frequency of seven disruptive nocturnal behaviors (DNB) associated with PTSD. These seven items assess the frequency of hot flashes, general nervousness, memories or nightmares of trauma, anxiety or panic unrelated to trauma, nightmares unrelated to trauma, night terrors during sleep, and episodes of acting out dreams. The PSQI-A addendum is added to the PSQI's original 19-item measure. The global PSQI-A score of 4 produced a sensitivity of 94% and a specificity of 82% for discriminating individuals with PTSD from those without PTSD. For the purposes of this study, the PSQI-A instructions were modified for the follow-up assessments, instructing participants to respond to questions in regard to their experience over the previous two weeks.

Epworth Sleepiness Scale (ESS; Johns, 1991). The ESS is an eight item self report questionnaire designed to provide a subjective estimation of daytime sleepiness. The ESS utilizes a 4-point Likert-type scale ranging from 0 ("*would never doze*") to 3 ("*high chance of dozing"*) and instructs participants to rate their usual level of sleepiness

across eight common situations of daily living (e.g., "*sitting and reading", "watching TV", "as a passenger in a car for an hour without a break"*). A total ESS score (range: 0-24) is calculated by summing the total of the eight items to derive an average sleep propensity score. A score greater than 10 is typically considered outside normal limits thus indicative of significant sleepiness (Johns, 1991). The ESS has demonstrated sound test-retest reliability in both medically healthy normal sleepers and patients treated for obstructive sleep apnea (Johns, 1992). The ESS has also been found to be sensitive to sleep-disordered breathing severity demonstrating a linear relationship with sleep apnea (Johns, 1993).

Nightmare Distress Questionnaire (NDQ; Belicki, 1992). The NDQ is a 13-item self report measure designed to provide a measure of nightmare intensity. The scale employs a Likert-type scale format to assess the frequency of nightmare related emotional distress. The first 10 items are reversed scored and all 13 items provide a summed total score ranging from 13-55, higher scores indicating higher levels of nightmare related distress. Across four samples Belicki (1992) reported reliability estimates ranging from .83 to .88. For the purposes of this investigation the time period assessed was modified from "*past year*" to "*past two weeks.*"

Health Measure

Short Form Health Survey 12 (SF-12; Ware, Kosinski, & Keller, 1996). The SF-12 is a 12-item self-report measure used to assess overall health functioning. The SF-12 can be used to derive two subscales: the mental health component summary and the physical health component summary. Two week test-retest correlations of .89 and .76 we found for the Physical component summary and mental health component summary

respectively (Ware et al., 1996). The SF-12 has been used in a wide range of populations and is considered a valid instrument.

Coping

Brief COPE (Carver, 1997). The Brief COPE is a 28-item self-report measure used to examine an individual's strategies of coping in response to stressful situations. The measure is divided into 14 subscales which include (self-distraction, active coping, denial, substance use, emotional support, instrumental support, behavioral disengagement, venting, positive reframing, planning, humor, acceptance, religion, and self-blame. The Brief COPE has been utilized in a range of populations including veterans with mental health concerns (Moore, Varra, Michael, & Simpson, 2010). Carver (1997) reported the internal reliabilities of the Brief COPE subscales to range from .50 (venting) to .90 (substance use). The Brief COPE was administered to participants during the baseline assessment.

Treatment Compliance

Daily Sleep Diary. The Daily Sleep Diary is a commonly used sleep measurement technique. The measure included in this study was modified specifically for this project to retrospectively assess both the characteristics of sleep and CPAP treatment compliance. For each post-treatment assessment, the participant was instructed to complete a daily log of their sleep habits including 1) time they got into bed the previous evening; 2) the time they got out of bed in the morning; 3) length of time they used their CPAP machine; 4) a subjective rating of overall sleep quality; and 5) any sleep medication use.

Side Effects Questionnaire (SEQ; Kribbs et al., 1993). The SEQ is a 22-item self-report measure designed to assess the presence and severity of common side effects

associated with CPAP use. Each side effect is rated on a scale from 0 ("*not a problem*") to 3 ("*a serious problem*"). The SEQ was administered at the final follow-up assessment (post-treatment time point 2). The side effects scale was added to the active study protocol due to elevated rates of CPAP non-compliance. Thererfore only a subset of participants (n = 24) were adminstered this measure.

Personality

The Big Five Inventory (BFI; John, Naumann, & Soto, 2008). The BFI is a 44-item self-report inventory designed to assess the Big Five personality dimensions of extraversion, agreeableness, conscientiousness, neuroticism, and openness. The measure asks participants to rate how much they agree with a series of short statements utilizing a 5-point Likert-type scale ranging from 1 ("*Disagree Strongly"*) to 5 ("*Agree Strongly"*). For the purposes of this study only the conscientiousness personality trait was measured. The purpose of the conscientiousness scale is to serve as a non-equivalent dependent variable that is not expected to change as a function of the CPAP treatment.

Procedure

Recruitment. Potential study participants were identified through the computerized personalized record system (CPRS) as they were scheduled for an overnight sleep study in the Neurology Sleep Clinic at the Memphis Veterans Affairs Medical Center. Once identified, potential participants were screened through the information available in their CPRS medical record and Neurology Sleep Clinic initial consult. A participant was eligible for recruitment if they met the following criteria: 1) at least 18 years of age; 2) no previous history of treatment with CPAP for sleep breathing disorders; 3) no previously diagnosed Axis-I psychotic disorder; and 4) no indication of

medical diagnosis (e.g., terminally ill) that could compromise their participation. Participants meeting these initial eligibility requirements (N = 489) were sent a letter explaining the purpose of the study and how the veteran may initiate participation in the study or how to opt out of the project. Approximately 10 days after sending the contact letter, if the participant had not initiated contact, the researcher followed up with potential participants by telephone to ensure receipt of the recruitment letter and answer any questions. Interested participants were scheduled for a baseline assessment prior to their overnight sleep study. Following the recruitment letter, participants were also recruited in-person at the Memphis VAMC. Potential participants were approached by research staff in the waiting room of the Neurology Sleep Clinic the evening of their overnight sleep study. Veterans were provided information about the research project and if interested, completed the baseline assessment prior to the start of their overnight sleep study.

During the baseline assessment, the researcher explained project procedures, potential risks and benefits of participation, aspects of confidentiality, and provided the rationale for the HIPAA release. All study participants provided informed consent and HIPPA authorization if they chose to proceed with participation. In order to maintain confidentiality eligible veterans who consented to participate were assigned an alphanumeric participant identification number produced by a random number generator.

Assessment. Assessment for current study consisted of three phases: a baseline screening assessment, a pretreatment follow-up phase, and a post-treatment follow-up phase. A total of 70 participants were recruited for the baseline assessment and each participant completed a battery of self-report questionnaires (for a complete list of

measures administered at each time point see Table 2). Participants were not compensated for completing the baseline assessment.

The second phase of the study consisted of a pre-treatment assessment which occurred following the overnight sleep study, but prior to the receipt of the CPAP machine. A participant was deemed eligible for the pretreatment follow-up phase if: 1) they scored ≥ 25 on the baseline administration of the PCL-S and 2) were diagnosed with OSA from their overnight PSG examination. Eligible and interested participants were assessed prior to the receipt of their CPAP machine with the assessment measures reflecting the veterans' functioning and symptom experience over the previous two week period. A total of 51 participants were eligible for the pretreatment phase; however, only 27 (52.9%) participants completed this assessment phase. Of the remaining 24 participants (47.1%), 18 were given their CPAP machine immediately following their overnight sleep study (thus were unable to complete the pretreatment follow-up phase), four participants were unable to be contacted for further participation, and two participants did not complete the pretreatment assessment prior to CPAP treatment initiation. Participants were compensated $10 for completing the pretreatment assessment.

The third phase of the study consisted of two post-treatment follow-up assessments. A veteran was deemed eligible for the post-treatment follow-up phase if they met the following criteria: 1) completed a night the overnight sleep study; 2) were diagnosed with OSA; and 3) received a CPAP machine. Participants meeting these criteria were assessed two weeks following initiation of CPAP treatment (post-treatment follow-up 1) and again four weeks following initiation of CPAP treatment (post-

treatment follow-up 2) Forty-five participants (95.7%) completed the 2 week post-treatment follow-up and 44 participants (93.6%) completed the 4 week post-treatment follow-up. Participants were compensated $5 for completion of each post-treatment follow-up assessment. Figure 1 presents a flow chart of participant recruitment and completion across the three phases of assessment. All study procedures were approved through the university's institutional review board (IRB) as well as the Memphis VAMC (IRB) and Research and Development committee.

Data Analysis Plan

The following analyses evaluated the associations between CPAP compliance for obstructive sleep apnea and change in PTSD symptoms, subjectively rated sleep difficulties, daytime sleepiness, and nightmare distress after CPAP treatment initiation. The final study sample (N = 47) consisted of veterans who, at a minimum, completed the baseline assessment, had a PCL $\geq$ 25 at baseline, and completed at least one of the post-treatment assessments.

Prior to conducting the analyses we examined the distributional properties of all continuous variables. Using a criterion of 3.29 SD units from the mean (Tabachnick & Fidell, 2005), there were no outliers on any variable in the dataset. We examined whether the variables were skewed or kurtotic and found all variables demonstrated acceptable distributional properties (all skewness and kurtosis values $\leq$ 1.2) without transformation.

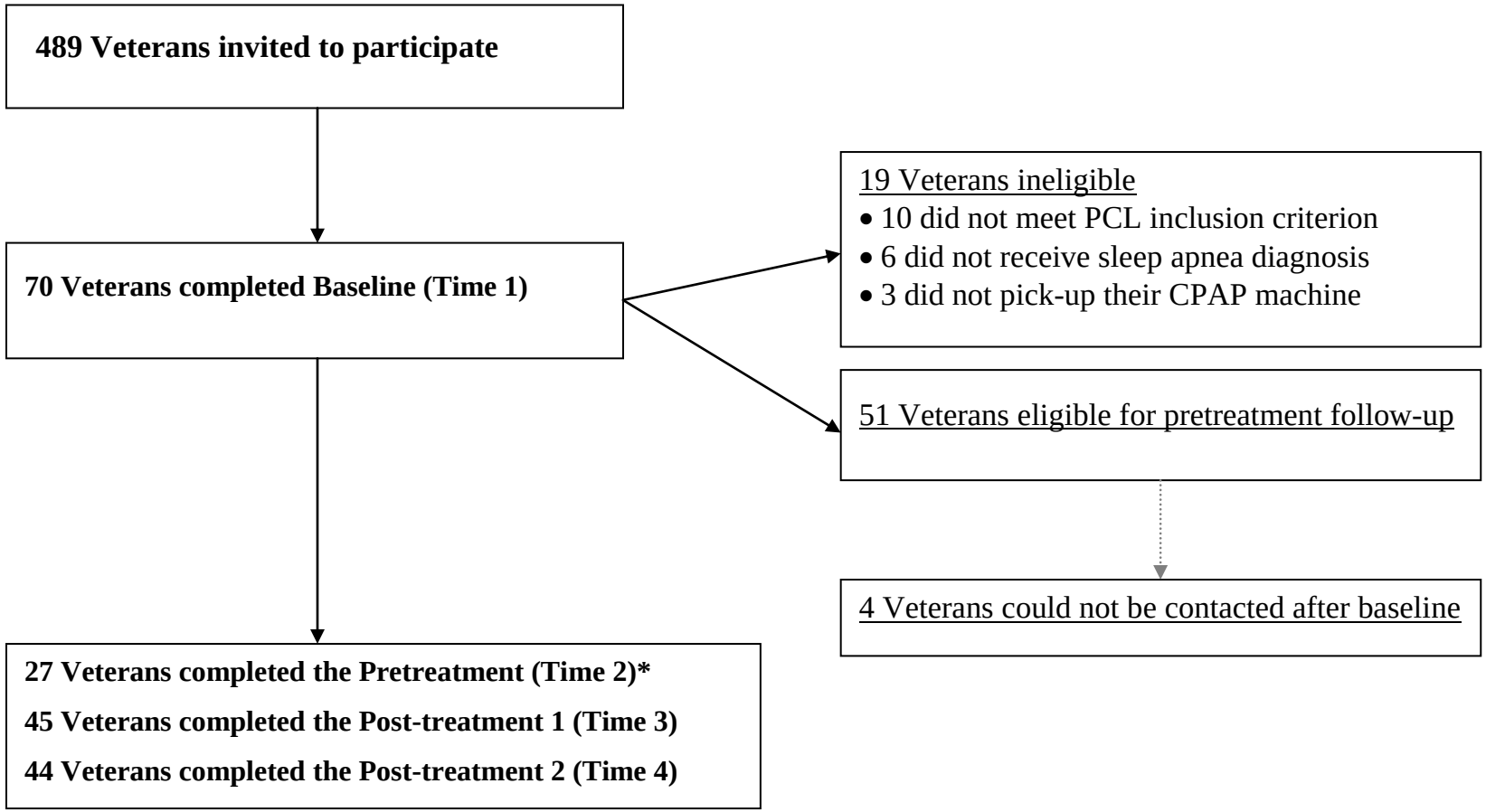

Figure 1. Flow chart of participant recruitment

Note. Of the 47 veterans eligible for pretreatment assessment, 18 received CPAP immediately following the overnight polysomnography study, thus were unable to complete the pretreatment assessment.

Baseline descriptive characteristics of the overall sample will be presented first, including the demographic information (gender, age, ethnicity, relationship status) as well as the means and standard deviations for the primary outcome variables (PTSD symptom severity, overall sleep quality, PTSD-related sleep disruption, nightmare distress, and daytime sleepiness). Baseline and pre-treatment descriptive characteristics were compared between participants who were CPAP-compliant and those who were non-compliant. We conducted T-tests and chi square analyses to assess any significant differences between these groups prior to CPAP treatment initiation. Using independent samples t-test, we compared the types of coping strategies used by individuals who were CPAP complaints compared to strategies utilized by participants who were not CPAP-compliant. Finally, we compared participants who were CPAP-compliant and non-compliant on the rate of endorsement of different adverse side effects associated with CPAP treatment.

The primary study analyses examined whether or not there was a statistically significant difference between CPAP-compliant and non-compliant participants on self-reported PTSD symptom severity and indices of sleep disturbance at the follow-up time points. Prior to testing the mixed models, relations between sample characteristics (compliance status, BMI, and demographic variables) and the primary outcome variables (PCL-S total score, PSQI Global Severity Index, PSQI-PTSD Addendum score, NDS total score, and ESS total score) were explored using Pearson correlation statistics. Next, a series of mixed-model repeated measures analyses were conducted to compare CPAP-compliant and non-compliant participants on each of the primary outcome variables (listed above) across the four time points. Mixed-effect models (also

known hierarchical linear models or multilevel models) provide a flexible framework for repeated measures analyses. Compared to traditional repeated measures analysis of variance (ANOVA), mixed-effect models utilize all available data for each participant to better accommodate for missing data (Gueorguieva & Krystal, 2004). For each model tested, one of the primary outcome variables served as the dependent variable. Demographic variables (i.e., gender, BMI, age, ethnicity, and relationship status) were included as covariates in a model if they were found to be correlated at the $p < .10$ level with group status (treatment compliant or non-compliant) or the respective primary outcome variable of the model.

Planned post-hoc comparisons were conducted for any statistically significant model effect or significant interaction. For all follow-up pairwise comparisons, precautions were taken to adjust for the possibility of detecting a false positive (Type-I error) significance level. We used the Bonferonni-Holm adjustment (Holm, 1979), to adjust the acceptable α-level based on the number of independent analyses conducted on our dataset (6). The Bonferroni-Holm procedure is adapted from the traditional Bonferroni correction, which modifies the acceptable alpha (α) level based on the number of comparisons being conducted. The traditional Bonferroni correction calculates adjusted α as: α/k (where k = number of comparisons) to be applied to all comparisons. This approach has been criticized as overly conservative and has the tendency to reduce power significantly (Holm, 1979). Given the small sample size of the current dataset, the modified Bonferroni-Holm adjustment was selected for its ability to increase power while still correcting for the possibility of familywise Type-I error. The Bonferroni-Holm adjustment calculates α based on the following procedure:

1. Order the p-values from lowest to highest [$p_{(1)}$, $p_{(2)}$, … $p_{(k)}$] for all comparisons
2. Compare $p_{(1)}$ to α/k (where k = number of comparisons), if $p_{(1)} > α/k$, then reject null hypothesis 1.
3. Next, compare $p_{(2)}$ to α/(k-1), if $p_{(2)} > α/k-1$, then reject null hypothesis 2.
4. Evaluate all comparisons to confirm or reject the null hypothesis.

The main study hypotheses had a maximum of six comparisons based on the four time points evaluated. Using the standard $p > .05$ statistical significance level as the starting α, Table 3 presents the adjusted *p*-values that will be applied to each comparison using the Bonferroni-Holm adjustment.

As previously mentioned, this study included a non-equivalent dependent variable as an additional measure of control in order to reduce threats to internal validity that are associated with quasi-experimental designs (Coryn & Hobson, 2011). A non-equivalent dependent variable serves as a control variable that is similar to the primary dependent variables and is theoretically affected in the same way by threats inherent to a single group pretest-posttest design, but different enough from the primary variables that it will not be affected by treatment. Using the mixed-model repeated measures analyses described above, we examined whether participants who were compliant with CPAP treatment experienced change self-rated level of conscientiousness (the non-equivalent dependent variable) compared to pretreatment self-rating.

Table 3

Adjusted α-level Based on Bonferroni-Holm Adjustment for Pairwise Comparisons

Ordered comparison (c)	Bonferroni-Holm equation $\alpha = .05/(k - c + 1)$	Adjusted α-level
1	$\alpha = .05/(k - 1 + 1)$	.008
2	$\alpha = .05/(k - 2 + 1)$	.01
3	$\alpha = .05/(k - 3 + 1)$	.013
4	$\alpha = .05/(k - 4 + 1)$	.02
5	$\alpha = .05/(k - 5 + 1)$	.025
6	$\alpha = .05/(k - 6 + 1)$	.05

Note. k = total number of comparisons (6). c = Ordered comparison based on *p*-value from least to greatest.

Results

Sample Characteristics

Prior to completing the PCL-S, all participants (N = 47) identified at least one potentially traumatic event using the Life Events Checklist. The most commonly reported "worst" events were categorized as "combat or exposure to war-zone" (48.9%) and "sudden, violent death" (10.6%). Other frequently endorsed "worst" events included "life threatening illness or injury" (6.4%), "transportation accident" (6.4%), "physical assault" (6.4%), and "exposure to toxic substances" (4.3%). At the baseline assessment the overall sample had a mean PCL-S score of 56.83 (*SD* = 15.33). For individuals that completed the pretreatment assessment (*n* = 28), the mean PCL-S score was 54.36 (*SD* = 14.09).

Baseline and pretreatment ratings were examined for measures of overall sleep quality, PTSD-related sleep disturbance, nightmare distress, and daytime sleepiness. As a whole, participants in the sample were considered poor sleepers (based on prior research, which has suggested a cut score of 5 as the threshold for poor sleep), with a mean Global Severity Index score of 14.30 (*SD* = 3.54) at baseline and 14.17 (*SD* = 3.12) at pretreatment. Participants reported moderate levels of PTSD-related sleep dysfunction, with means of 9.00 (*SD* = 5.25) and 9.64 (*SD* = 5.24) at baseline and pretreatment respectively. The sample mean for nightmare-related distress was not in the elevated range prior to CPAP treatment initiation, with mean severity scores of 31.39 (*SD* = 9.88) at baseline and 29.54 (*SD* = 9.00) at pretreatment. The sample also an elevated level of daytime sleepiness with mean severity scores of 10.52 (*SD* = 6.04) at baseline and 12.40 (*SD* = 5.93) at pretreatment Table 4 presents baseline and pretreatment descriptive information for the primary outcome variables (PCL-S total scores, PSQI Global scores,

PSQI-PTSD Addendum scores, NDS total scores, and ESS total scores) separately for CPAP-compliant participants and non-compliant participants. There were no statistically significant baseline or pretreatment differences between CPAP-compliant and non-compliant participants on any of the primary outcome variables.

In addition to the self-report questionnaires administered by study personnel, participant's medical records were reviewed to obtain two additional physical health indicators, body mass index (BMI) and comorbid health conditions (total number of clinician-diagnosed physical health conditions). Participant's BMI was recorded as part of their overnight sleep study at the Neurology Sleep Clinic. The Centers for Disease Control and Prevention (2011) classifies BMIs of <18.5 as "underweight," 18.5 to 24.9 as "normal," 25.0 – 29.9 as "overweight," and > 30.0 as "obese." Review of medical records revealed the sample mean fell in the "obese" range at 33.59 (*SD* = 5.31). Twelve participants (25.5%) were classified as "overweight" and 35 participants (74.5%) were considered "obese." There was not a statistically significant difference in BMI for CPAP-compliant and non-compliant participants. With respect to comorbid health conditions, the mean number of active diagnoses (excluding mental health conditions) for the full sample was 7.84 (*SD* = 4.19). There was not a statistically significant difference in the number of active physical health conditions for CPAP-compliant and non-compliant participants and this health index was not significantly correlated with any of the primary outcome variables examined.

Table 4

Baseline and Pre-treatment Descriptives across Time on PTSD Symptoms, Indices of Sleep, and Nightmare Distress compared by CPAP Compliance Status

	CPAP-compliant (*n* = 25)	CPAP Non-compliant (*n* = 22)	
	M (SD)	*M* (SD)	*t*-statistic
PCL-S Total Score (PCL-S)			
Baseline PCL-S	56.13 (15.64)	57.59 (15.31)	*t* (44) = .32
Pretreatment 1 PCL-S	54.13 (14.99)	54.62 (13.57)	*t* (26) = .09
PSQI Global Severity Index (GSI)			
Baseline GSI	13.76 (3. 81)	14.95 (3.15)	*t* (44) = 1.14
Pretreatment 1 GSI	14.25 (3.26)	14.08 (3.07)	*t* (27) = -.15
PSQI-PTSD Addendum (PSQI-PTSD)			
Baseline PSQI-PTSD	8.43 (5.10)	9.81 (5.50)	*t* (37) = .80
Pretreatment 1 PSQI-PTSD	9.43 (5.76)	10.00 (4.54)	*t* (20) = .24

*$p \leq .05$. **$p \leq .01$.

(table continues)

Table 4 (continued)

Baseline and Pre-treatment Descriptives across Time on PTSD Symptoms, Indices of Sleep, and Nightmare Distress compared by CPAP Compliance Status

	CPAP-compliant (n = 25)	CPAP Non-compliant (n = 22)	
	M (SD)	*M* (SD)	*t*-statistic
Nightmare Distress Scale (NDS)			
Baseline NDS	29.73 (9.17)	33.32 (10.56)	t (39) = 1.17
Pretreatment 1 NDS	28.15 (9.13)	30.92 (9.01)	t (24) =.78
Epworth Sleepiness Scale (ESS)			
Baseline EES	9.25 (6.16)	12.05 (5.67)	t (42) = 1.56
Pretreatment 1 EES	12.91 (6.20)	11.76 (5.78)	t (27) = -.51

*$p \leq .05$. **$p \leq .01$.

The apnea-hypopnea index (AHI) is an objective measure of OSA severity that calculates the quantity of respiratory pauses (≥ 10 seconds in duration) during sleep per hour. According to recommendations from the American Academy of Sleep Medicine (Ruehland et al., 2009), AHI values between 5-15/hr are categorized a "mild OSA," values between 15-30/hr as "moderate OSA," and values >30/hr as "severe OSA." Results from the Neurology Sleep Clinic's sleep study report indicated 55.3% of the current sample was rated as having severe OSA, 14.9% as moderate, and 25.5% as mild. There was not a statistically significant difference in OSA severity between CPAP-compliant and non-compliant participants and AHI was not significantly correlated with any of the primary outcome variables examined.

CPAP compliance. Subjectively reported CPAP utilization resulted in 27 participants being classified as CPAP-compliant, averaging 6.19 (*SD* = 1.50) hours per night. The CPAP non-compliant group (*n* = 20) reported an average nightly usage of 1.11 (1.30) hours. A measure of objective CPAP compliance (retrieved from each veteran's CPAP "SmartCard," an electronic device embedded in the CPAP equipment) was retrieved through chart review following the 3-month follow-up visit with the Neurology Sleep Clinic. Of the 47 total participants, SmartCard data was available for 23 participants. Although a direct comparison of CPAP utilization was not possible due to the different follow-up periods for the current study and the Neurology Sleep Clinic (1-month and 3-months respectively), the downloaded SmartCard data yielded a total days of use index. We compared the self-reported total day CPAP usage to the SmartCard total day usage and found SmartCard usage (total days used 3-months) was less than self-reported CPAP usage (1-month) for two participants. For these participants, the

SmartCard indicated a total of 13 days of usage for one and 9 days of usage for the other. We recalculated average nightly CPAP usage based on SmartCard indicated total days used and average self-reported nightly use. For both participants, CPAP usage fell below the cut-off (≥ 4) for treatment compliance, thus were reclassified as CPAP non-compliant. The final sample included 25 participants considered CPAP-compliant and 22 participants considered non-compliant.

Cross-sectional Comparisons by CPAP Compliance Status

Coping Strategies. Using independent samples *t*-tests, we compared the coping strategies utilized by participants who were CPAP-compliant compared to participants who were CPAP non-compliant. Of the 14 strategies measured by the Brief Cope (Carver, 1997), differences emerged across two scales. Participants who were not CPAP-compliant reported using both "instrumental support" and "venting" significantly more compared with participants who were CPAP-compliant (Table 5). No other statistically significant group differences emerged for coping strategy utilization.

Side Effects of CPAP. The side effects associated with CPAP treatment were measured at the four week follow-up time point. Using independent samples *t*-tests we compared the side effects of CPAP endorsed by participants who were CPAP-compliant compared with those who were non-compliant. Out of 21 side effects measured, results found non-compliant participants reported significantly more problems associated with CPAP mask irritation and CPAP-related sleep disturbance including more disturbed sleep, more restlessness, and poorer sleep (Table 6). No statistically significant group differences emerged for the 17 other side effects measured.

Table 5

Comparison of Coping Strategies by CPAP Compliance Status

	CPAP-compliant (n = 14)	CPAP Non-compliant (n = 13)	
	M (SD)	*M* (SD)	*t*-statistic
Coping Strategy			
Self-distraction	5.29 (1.49)	5.15 (2.08)	t (25) = -.19
Active coping	4.79 (1.76)	5.77 (1.74)	t (25) = 1.46
Denial	5.00 (2.18)	4.31 (1.84)	t (25) = -.89
Substance use	3.54 (2.30)	3.54 (1.98)	t (25) = -00
Use of emotional support	3.57 (2.03)	5.08 (1.98)	t (25) = 1.95†
Use of instrumental support	3.21 (1.48)	5.69 (1.98)	t (25) = 3.76**
Behavioral disengagement	3.29 (1.48)	4.15 (2.11)	t (25) = 1.26
Venting	4.21 (1.48)	5.77 (2.01)	t (25) = 2.31*
Positive reframing	4.71 (1.68)	5.15 (1.68)	t (25) = .68
Planning	5.07 (1.86)	5.23 (1.48)	t (25) = .25

$^{\dagger}p < .10$. *$p \leq .05$. **$p \leq .01$.

(table continues)

Table 5 (continued)

Comparison of Coping Strategies by CPAP Compliance Status

	CPAP-compliant (n = 14)	CPAP Non-compliant (n = 13)	
	M (SD)	*M* (SD)	*t*-statistic
Humor	3.57 (1.87)	3.77 (2.49)	t (25) = .24
Acceptance	5.29 (1.98)	6.38 (1.50)	t (25) = 1.62
Religion	5.64 (1.95)	6.15 (2.27)	t (25) = .63
Self-blame	4.57 (2.28)	5.38 (1.61)	t (25) = 1.06

$^{\dagger}p < .10$. $^{*}p \leq .05$. $^{**}p \leq .01$.

Table 6

Comparison of CPAP Side Effects by CPAP Compliance Status

	CPAP-compliant (n = 15)	CPAP Non-compliant (n = 9)	
Side Effects	*M* (SD)	*M* (SD)	*t*-statistic
CPAP is too inconvenient to use	1.00 (1.13)	1.56 (.88)	t (22) = 1.26
The CPAP mark irritates my face	.87 (.99)	1.75 (.71)	t (21) = 2.23*
I can't sleep because of the noise CPAP makes	.31 (.63)	.89 (1.05)	t (20) = 1.62
My eyes are irritated by CPAP	.67 (.90)	.78 (1.09)	t (22) = .27
The CPAP mask makes me claustrophobic	.53 (.92)	1.11 (.93)	t (22) = 1.49
My ears are irritated by the CPAP machine[a]	.13 (.35)	.56 (.88)	t (22) = 1.67
I have chest pains when I use CPAP	.13 (.35)	.11 (.33)	t (22) = -.15
I am embarrassed to use my CPAP	.33 (.62)	.44 (1.01)	t (22) = .34
My nose bleeds when I use CPAP	.27 (.46)	.22 (67)	t (22) = -.19
I have difficulty operating the CPAP machine	.13 (.35)	.11 (.33)	t (22) = -.15
The CPAP makes the bridge of my nose hurt	1.20 (1.21)	.78 (1.09)	t (22) = -.86

$^{\dagger}p < .10$. $*p \leq .05$. $**p \leq .01$.

(table continues)

Table 6 (continued)

Comparison of CPAP Side Effects by CPAP Compliance Status

	CPAP-compliant (n = 15)	CPAP Non-compliant (n = 9)	
Side Effects	*M* (SD)	*M* (SD)	*t*-statistic
I have trouble putting on the CPAP mask	.20 (.56).	.22 (.44)	t (22) = .10
CPAP disturbs my sleep	.47 (.83)	1.89 (.93)	t (22) = 3.88**
CPAP gives me headaches	.20 (.56)	.11 (33)	t (22) = -.43
CPAP results in less intimacy with my bed partner	.47 (.74)	.25 (.46)	t (21) = -.75
CPAP makes my nose stuffy or dry[a]	.47 (.64)	1.11 (1.27)	t (22) = 1.66
I toss and turn more with CPAP	.58 (.99)	1.67 (1.12)	t (22) = 2.34*
I sleep poorly with CPAP	.80 (1.15)	1.78 (.97)	t (22) = 2.14*
My bed partner sleeps worse when I use CPAP[a]	.29 (61)	.63 (1.19)	t (22) =.89
I sleep worse when I use CPAP	.71 (1.14)	1.57 (1.27)	t (22) = 1.57

Note. [a] Levene's Test for Equality of Variances = $p < .05$, thus equal variances not assumed.

$^{\dagger}p < .10$. *$p \leq .05$. **$p \leq .01$.

Mixed-model Repeated Measures Analyses

Prior to testing the mixed models, relations between compliance status and demographic variables and the primary outcome variables (PCL-S total score, PSQI Global Severity Index total, PSQI-PTSD Addendum score, NDS total score, and ESS total score) were explored using Pearson correlation statistics. Factors correlated at the $p < .10$ level with compliance status or the primary outcome variables were included as covariates in their respective models. Correlation analyses are presented in Table 7. For the purposes of the current analyses, relationship status was recoded into a dichotomous variable that reflected being in a relationship or not. This was necessary given that some categories of relationship status had too few participants to allow for post-hoc comparisons if found to be significant. The recoded relationship status variable resulted in 26 participants being classified as involved in a relationship and 21 not currently involved.

Table 7

Correlation Matrix for Demographic Variables and Primary Outcome Variables

Variables	1	2	3	4	5	6
Sample Characteristics						
1. Age	___					
2. Gender	.05	___				
3. Ethnicity	.02	.15	___			
4. Relationship status	.11	-.38**	-.17	___		
5. Body mass index[a]	-.19	.19	-.03	.06	___	
6. Treatment compliance status	.03	.19	-.14	.10	.07[a]	___
Outcome Variables						
PCL-S total– Baseline[a]	-.11	-.06	.03	.11	-.30*[b]	-.05
PCL-S total – Pre-treatment[l]	-.15	-.00	-.13	.32	-.09	.01
PCL-S total – Post-treatment 1[b]	.08	.01	.10	.05	-.29[c]	-.28
PCL-S total – Post-treatment 2[c]	.05	.03	.12	.04	-.19	-.21
PSQI global – Baseline[a]	-.05	.01	-.02	-.03	-.07[b]	-.17
PSQI global – Pre-treatment[l]	-.19	-.16	-.07	-.10	.09	.03
PSQI global – Post-treatment 1[c]	-.10	.06	.12	-.01	-.04[d]	-42**
PSQI global – Post-treatment 2[e]	-.02	-.04	-.10	.16	-.08	-.20
PSQI-PTSD – Baseline[h]	-.06	-.16	.16	.17	-.27[i]	-.13
PSQI-PTSD – Pre-treatment[n]	-.31	-.30	.11	.32	-.20	-.09
PSQI-PTSD – Post-treatment 1[j]	.01	-.05	.32[†]	-.12	-.05[k]	-.15

[†]$p < .10$. *$p \leq .05$. **$p \leq .01$.

(table continues)

Table 7 (continued)

Correlation Matrix for Demographic Variables and Primary Outcome Variables

Variables	1	2	3	4	5	6
PSQI-PTSD – Post-treatment 2[j]	-.03	-.19	.29[†]	.12	-.23	-.17
NDS total – Baseline[a]	-.01	-.12	.11	-.20	-.37*[b]	-.18
NDS total – Pre-treatment[l]	-.26	-.20	-.08	-.43*	-.20	-.16
NDS total – Post-treatment 1[b]	-.17	.09	.16	-.09	-.34*[c]	-.14
NDS total – Post-treatment 2[c]	-.06	.02	.13	-.20	-.29[†]	-.15
ESS total – Baseline[c]	-.19	-.04	.07	.03	.06	-.23
ESS total – Pretreatment[l]	-.23	.18	-.04	.23	.16	.04
ESS total – Post-treatment 1[d]	-.15	-.04	-.06	.07	.01[e]	-.16
ESS total – Post-treatment 2[e]	-.09	.13	.21	.05	.04	-.18
Conscientiousness – Baseline[d]	.03	-.14	.11	.07	-.14[e]	.22
Conscientiousness – Pretreatment[o]	.36[†]	.09	.10	.09	-.32	.20
Conscientiousness - Post-treatment 1[d]	-.01	-.02	.13	.29[†]	-.06	.25
Conscientiousness Post-treatment 2[d]	-.02	.00	-.05	.18	-.14	.24

Note. PCL-S total = PTSD Checklist total score; PSQI global = Pittsburgh Sleep Quality Index Global Severity Index; PSQI-PTSD = Pittsburgh Sleep Quality Index PTSD Addendum total score; NDS total = Nightmare Distress Scale total score; ESS total = Epworth Sleepiness Scale total score.

[a]$n = 46$. [b]$n = 45$. [c]$n = 44$. [d]$n = 43$. [e]$n = 42$. [f]$n = 41$. [g]$n = 40$. [h]$n = 39$. [i]$n = 38$. [j]$n = 37$. [k]$n = 36$. [l]$n = 27$. [m]$n = 26$. [n]$n = 20$ [o] $n = 28$.

[†]$p < .10$. *$p \leq .05$. **$p \leq .01$.

Hypothesis 1. A 2 (group) x 4 (time) mixed-model repeated measures analysis was conducted to compare participants who were CPAP-compliant to those who were non-compliant on PTSD symptom severity. BMI was included as a covariate in this model given the significant negative correlation with baseline PCL-S total score. The results of this analysis did not show any statistically significant main effects (time, CPAP compliance, or BMI); however, analyses did find a statistically significant compliance status x time interaction ($F(3, 102.15) = 5.66$. $p = .001$). Follow-up Bonferonni-Holm corrected pairwise comparisons (Table 8) revealed that CPAP-compliant participants reported a statistically significant linear reduction in PTSD symptoms from pre to post-treatment, whereas non-compliant participants did not report a statistically significant reduction from pre to post-treatment (Figure 2). For CPAP-compliant participants, the estimated marginal means (*EMM*) for PCL score at the baseline (*EMM* = 56.14) and pretreatment (*EMM* = 55.65) time points were significantly higher than the post-treatment 1 (*EMM* = 46.73) and post-treatment 2 (*EMM* = 47.03) time points. Non-compliant participants reported a statistically significant reduction in PTSD symptoms from baseline (*EMM* = 57.59) to pretreatment (*EMM* = 52.07), indicating a significant reduction in PTSD symptoms *prior to* the treatment implementation. Further examination of this decrease in PTSD symptoms from baseline to pretreatment revealed 5 out of the 13 non-compliant participants that completed pretreatment follow-up reported ≥ 9 point decreases in symptoms from baseline to pretreatment. Of note, there was not a statistically significant difference from baseline to pretreatment estimated marginal means for the CPAP-compliant group. The CPAP-compliant and non-compliant groups did not differ significantly from one another at pretreatment. Table 8 presents the means

and standard deviations for each group across the four time points and Bonferonni-Holm corrected pairwise comparisons are presented in Table 9.

A series of exploratory 2 (group) x 4 (time) mixed-model repeated measures analyses were conducted to compare participants who were CPAP-compliant to those who were non-compliant across the three DSM-IV-TR PTSD symptom domains (reexperiencing, avoidance/numbing, and hyperarousal). These analyses failed to find statistically significant main effects or significant interactions for reexperiencing (Figure 3) and avoidance/numbing (Figure 4) symptom domains. A trend level compliance status x time interaction emerged for the hyperarousal symptom domain ($F(3, 105.11) = 2.40$. $p = .072$). Despite this trend level finding, follow-up Bonferonni-Holm corrected pairwise comparisons did not find a significant change in hyperarousal symptoms for either group (CPAP-compliant or non-compliant) from pre-to-post treatment (Figure 5). Of note, when we compared the estimated marginal means for CPAP-compliant and non-compliant participants at each time point, we found a statistically significant difference at the pretreatment time point such that non-compliant participants reported significantly more hyperarousal symptoms compared to CPAP-compliant participants (Figure 5).

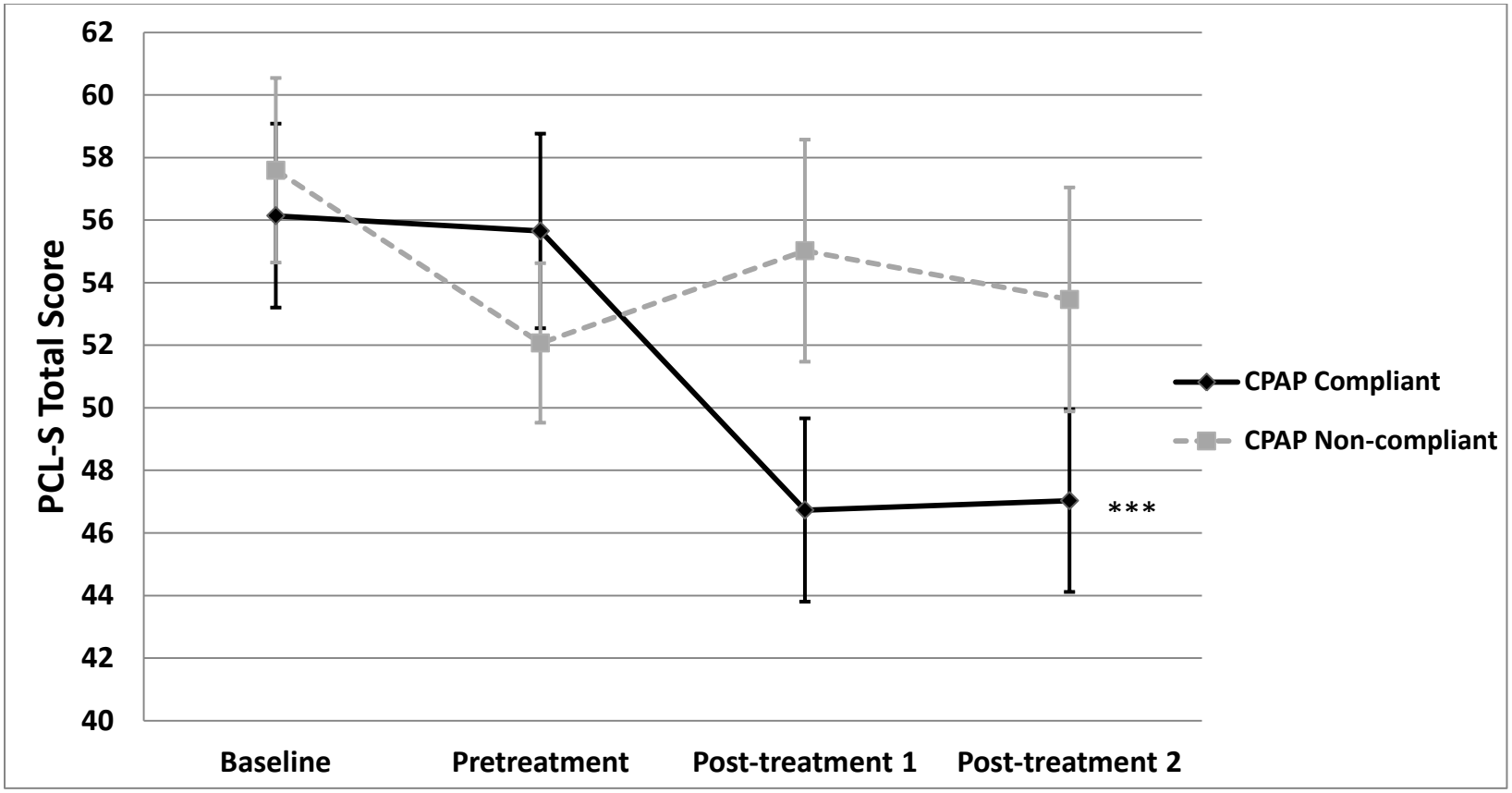

Figure 2. Change over time in PTSD symptoms for CPAP-compliant and non-compliant participants.

Note. Sample sizes for the CPAP-compliant group were: baseline, *n* = 24, pre-treatment, *n* = 14, post-treatment 1, n = 24, and post-treatment 2, *n* = 25. Sample sizes for the CPAP non-compliant group were: baseline, *n* = 22, pre-treatment, *n* = 13, post-treatment 1, n = 21, and post-treatment 2, *n* = 19.

*$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

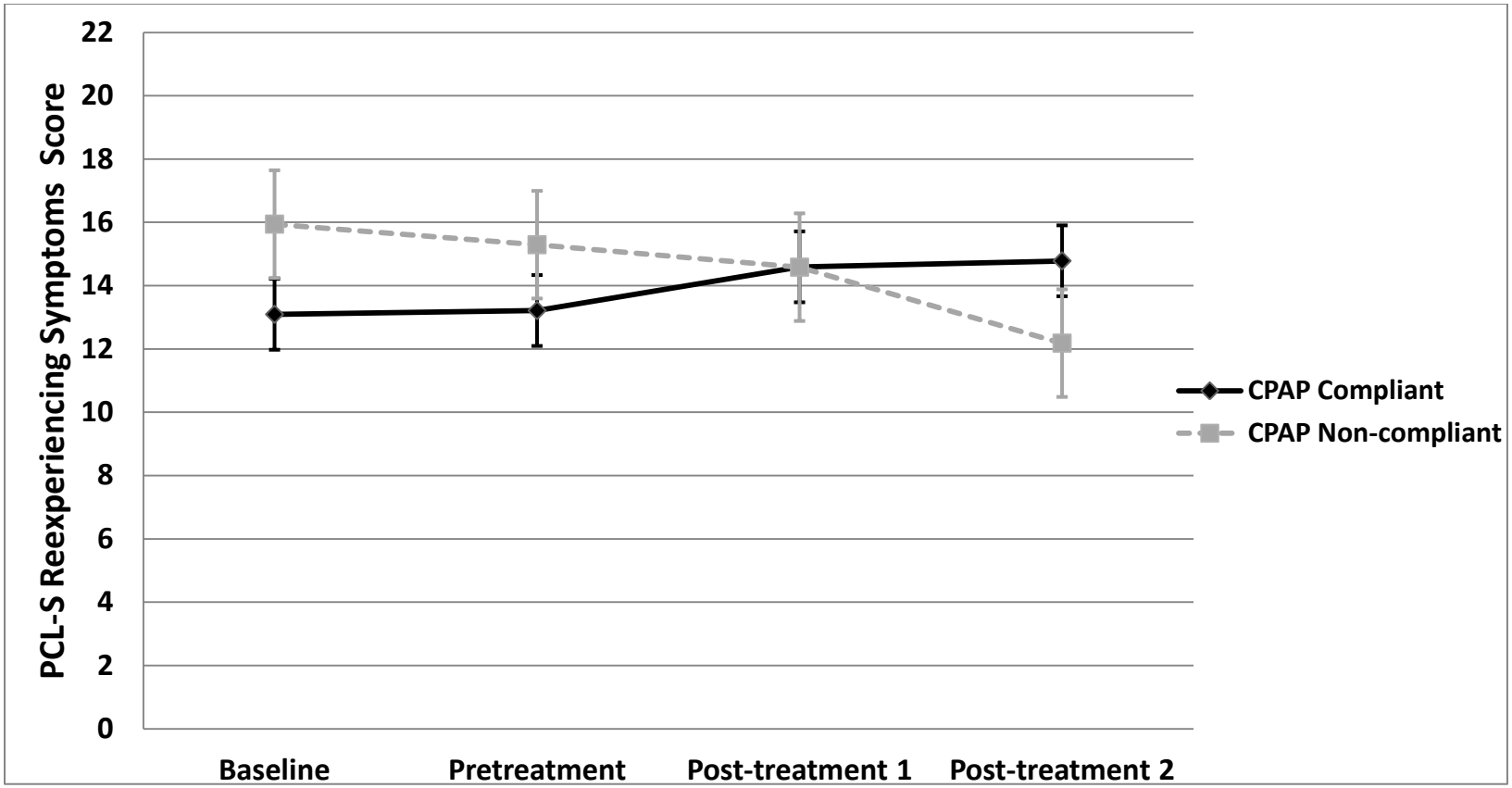

Figure 3. Change over time in re-experiencing symptoms for CPAP-compliant and non-compliant participants.

Note. Sample sizes for the CPAP-compliant group were: baseline, $n = 24$, pre-treatment, $n = 14$, post-treatment 1, n = 24, and post-treatment 2, $n = 25$. Sample sizes for the CPAP non-compliant group were: baseline, $n = 22$, pre-treatment, $n = 13$, post-treatment 1, n = 21, and post-treatment 2, $n = 19$.

*$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

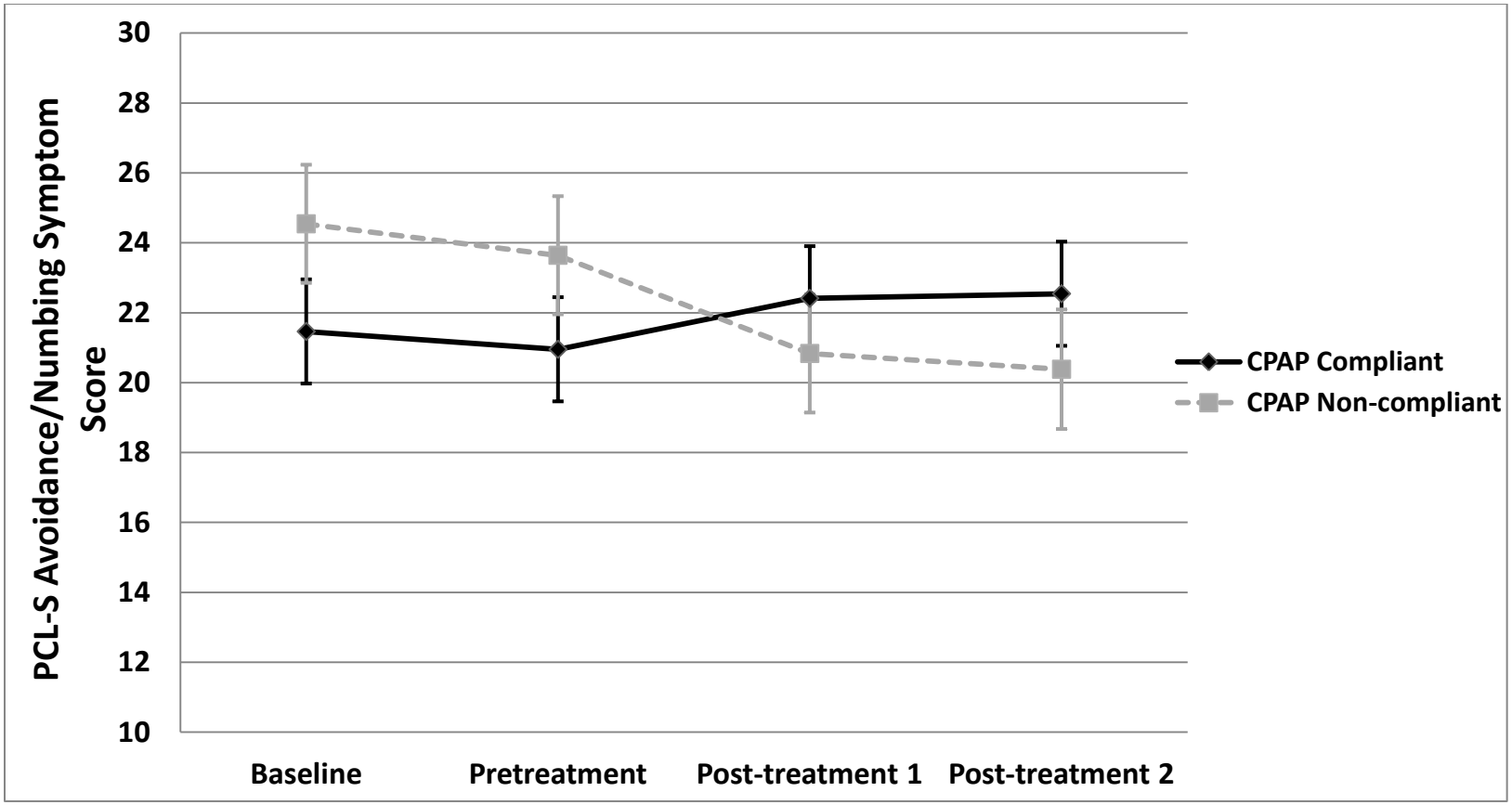

Figure 4. Change over time in avoidance/numbing symptoms for CPAP-compliant and non-compliant participants.

Note. Sample sizes for the CPAP-compliant group were: baseline, *n* = 24, pre-treatment, *n* = 14, post-treatment 1, n = 24, and post-treatment 2, *n* = 25. Sample sizes for the CPAP non-compliant group were: baseline, *n* = 22, pre-treatment, *n* = 13, post-treatment 1, n = 21, and post-treatment 2, *n* = 19.

*$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

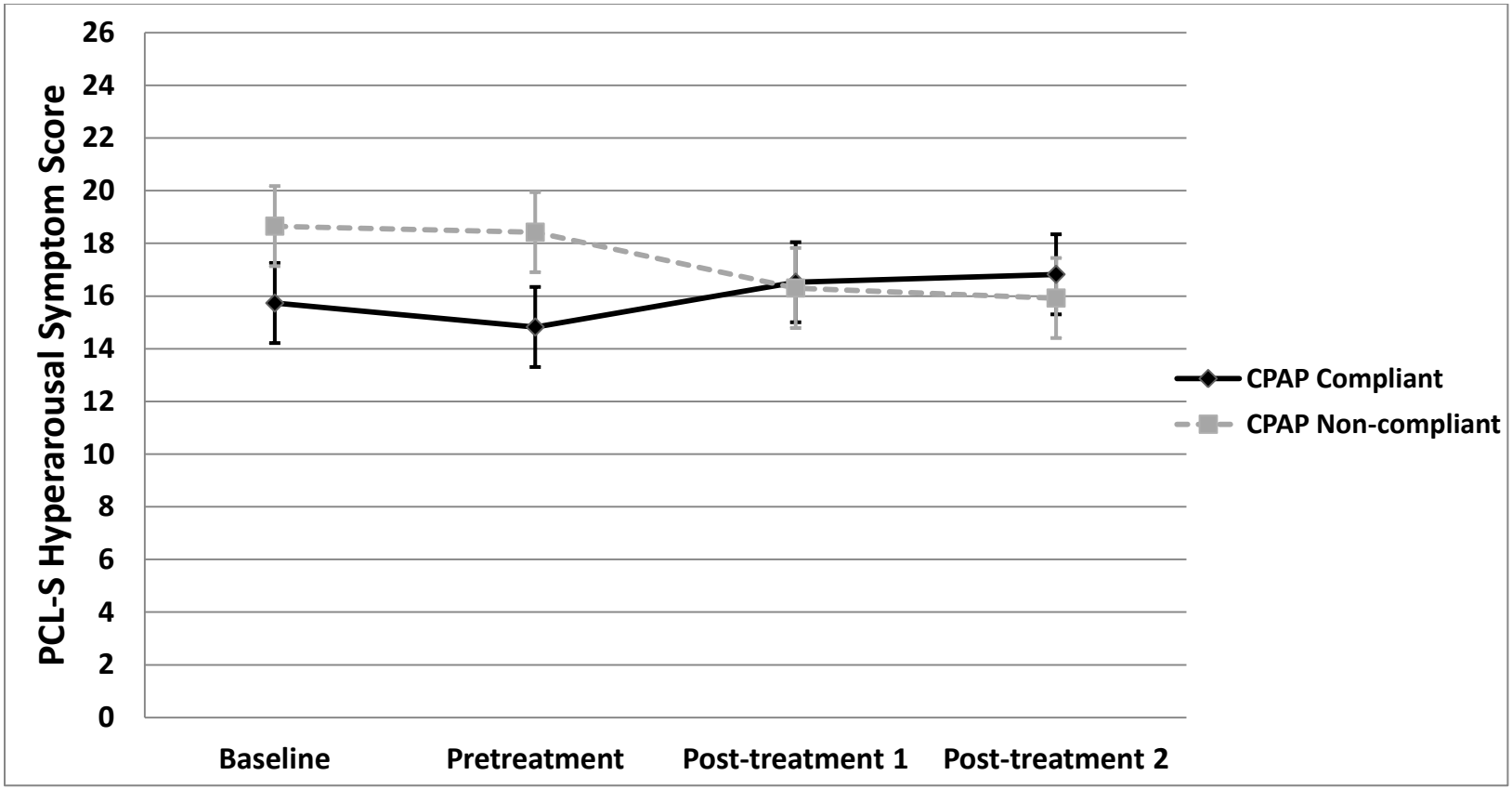

Figure 5. Change over time in hyperarousal symptoms for CPAP-compliant and non-compliant participants.

Note. Sample sizes for the CPAP-compliant group were: baseline, *n* = 24, pre-treatment, *n* = 14, post-treatment 1, n = 24, and post-treatment 2, *n* = 25. Sample sizes for the CPAP non-compliant group were: baseline, *n* = 22, pre-treatment, *n* = 13, post-treatment 1, n = 21, and post-treatment 2, *n* = 19.

*$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

Two additional exploratory 2 (group) x 4 (time) mixed-model repeated measures analyses were tested that avoidance and numbing symptom domains separately. Analyses revealed a significant compliance status X time interaction for avoidance symptoms (F(3, 107.47) = 2.96. p = .036). Follow-up Bonferonni-Holm corrected pairwise comparisons revealed that participants who were not compliant with CPAP treatment reported a statistically significant reduction in avoidance symptoms from the baseline (*EMM* = 7.93) and pretreatment (*EMM* = 7.80) time points to post-treatment 1 (*EMM* = 6.06) time point and between baseline and post-treatment 2 (*EMM* = 5.92) time points. CPAP-compliant participants did not report a statistically significant change from pre-to-post treatment. When we compared the estimated marginal means for CPAP-compliant and non-compliant participants at each time point, we found a statistically significant difference at the baseline and pretreatment time points such that non-compliant participants reported significantly more avoidance symptoms compared to CPAP-compliant participants. No statistically significant main effects or interactions emerged for the model testing numbing symptoms.

Hypothesis 2a. A 2 (group) x 4 (time) mixed-model repeated measures analysis was conducted to compare CPAP-compliant and non-compliant participants on overall sleep quality as measured by the PSQI Global Severity Index. Analyses revealed a statistically significant compliance status x time interaction (F(3, 101.59) = 5.65. p = .001). Follow-up Bonferonni-Holm corrected pairwise comparisons revealed that both CPAP-compliant and non-compliant participants reported a statistically significant improvement overall sleep quality from pre to post-treatment (Figure 6). For CPAP-compliant participants, the global severity index score at the baseline (*EMM* = 13.76)

and pretreatment (*EMM* = 13.81) time points were significantly higher than the post-treatment 1 (*EMM* = 9.60) and post-treatment 2 (*EMM* = 10.56) time points. There was not a statistically significant difference between baseline and pretreatment estimated means for this group. For CPAP non-compliant veterans, the baseline time point (*EMM* = 15.07) was significantly different from the post-treatment 2 (*EMM* = 12.13) time point; however, there was not a statistically significant difference between the baseline and post-treatment 1 (*EMM* = 13.15) time point or the pretreatment time point (*EMM* = 13.86) and either of the post-treatment time points (Table 9). Analyses also revealed a statistically significant main effect for time ($F(3, 101.59) = 13.12$, $p < .001$) such that the full sample showed improvement in overall sleep quality across time. Means and standard deviations for each group are presented in Table 8.

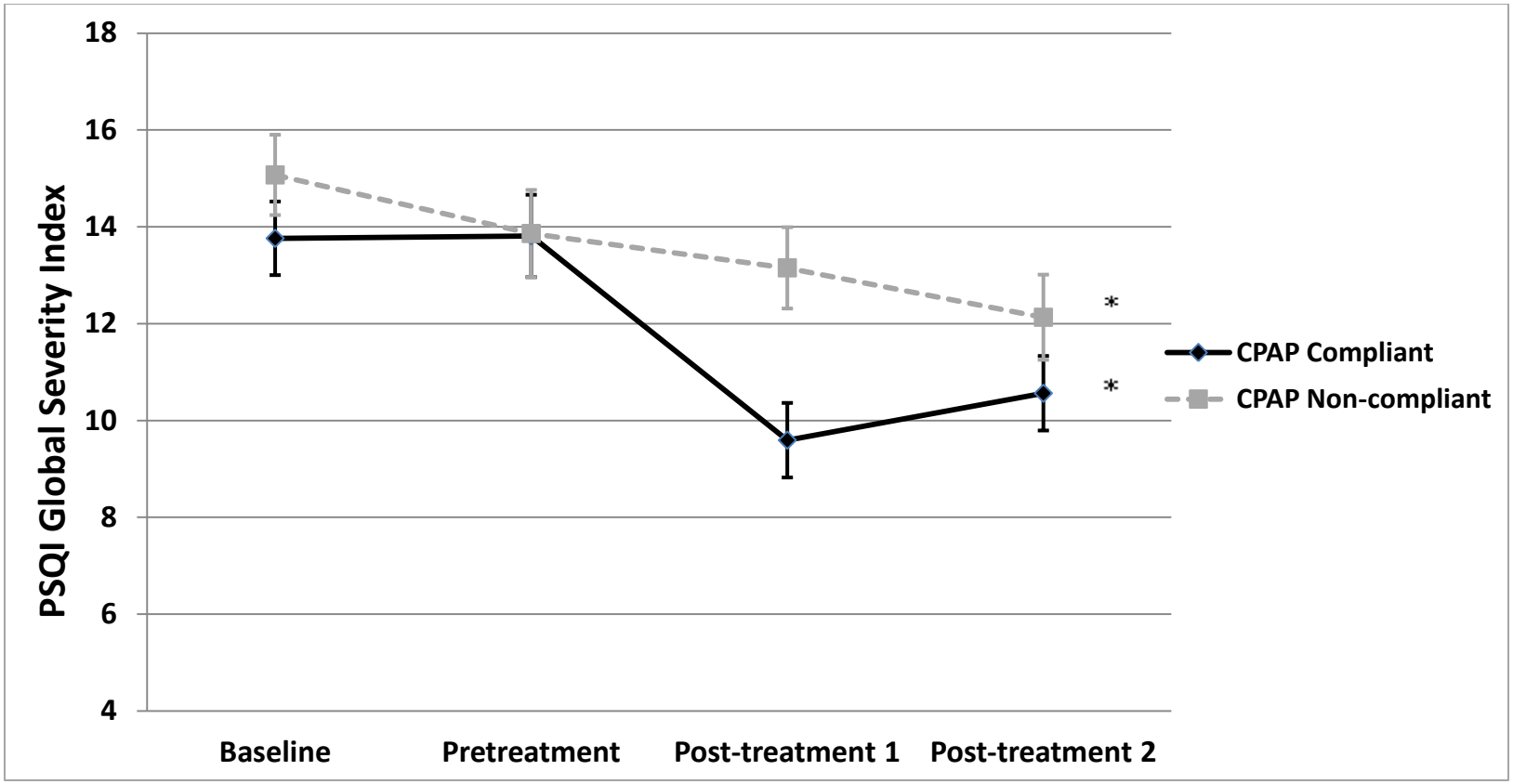

Figure 6. Change over time in overall sleep quality for CPAP-compliant and non-compliant participants.

Note. Sample sizes for the CPAP-compliant group were: baseline, *n* = 24, pre-treatment, *n* = 14, post-treatment 1, n = 24, and post-treatment 2, *n* = 25. Sample sizes for the CPAP non-compliant group were: baseline, *n* = 22, pre-treatment, *n* = 13, post-treatment 1, n = 21, and post-treatment 2, *n* = 19.

*$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

Hypothesis 2b. A separate 2 (group) x 4 (time) mixed-model repeated measures analysis was conducted on PTSD-related sleep dysfunction as measured by the PSQI-PTSD Addendum. Ethnicity was included as a covariate in this model given the trend level positive association with post-treatment 1 PSQI-PTSD total score (r = .32; p < .10) and post-treatment 2 PSQI-PTSD total score (r = .29; p < .10). Analyses revealed statistically significant main effects for time (F(3, 83.31) = 3.91, p = .012) such that the full sample reported a mean reduction in PTSD-related sleep disruption from pre-to-post treatment follow-up. Follow-up Bonferonni-Holm corrected pairwise comparisons (Table 9) showed a statistically significant reduction in reported PTSD-related sleep disruption from the baseline (*EMM* = 9.00) and pretreatment (*EMM* = 8.58) time points to the post-treatment 1 (*EMM* = 6.93) time point. There was not a statistically significant difference between any other time points. No statistically significant main effects emerged for CPAP compliance status or ethnicity.

Hypothesis 3. A 2 (group) x 4 (time) mixed-model repeated measures analysis was conducted to compare CPAP-compliant and non-compliant participants on nightmare distress as measured by the Nightmare Distress Scale. Both BMI and relationship status were included as a covariates in this model given significant associations with NDS total score. Mixed model analyses revealed a statistically significant main effect for time (F(3, 105.08) = 3.91, p = .011) such that the overall sample reported a linear increase in nightmare-related distress across time (see Figure 7). Follow-up Bonferonni-Holm corrected pairwise comparisons revealed a statistically significant increase in reported nightmare-related distress from the baseline (*EMM* = 28.03) time point to the post-treatment 1 (*EMM* = 30.38), and post-treatment 2 (*EMM* = 30.54) time points. However,

post-hoc comparison also revealed a statistically significant increase in nightmare distress from the baseline to pretreatment (*EMM* = 29.81) time point and no statistically significant difference between the pretreatment time point and either post-treatment follow-up time point (Table 9).

Hypothesis 4. A 2 (group) x 4 (time) mixed-model repeated measures analysis was conducted to compare participants who were CPAP-compliant to those who were non-compliant on daytime sleepiness as measured by the Epworth Sleepiness Scale (Figure 8). Analyses failed to find any statistically significant main effects; however, there was a statistically significant compliance status x time interaction ($F(3, 100.87 = 3.60$, $p = .016$). Follow-up Bonferonni-Holm corrected pairwise comparisons revealed a statistically significant increase in reported daytime sleepiness from baseline (*EMM* = 8.85) to the pretreatment follow-up (*EMM* = 11.53) for the CPAP-compliant group. This difference was not observed for CPAP non-compliant participants. There were no other statistically significant differences on the pairwise comparison analyses for either group (Table 9).

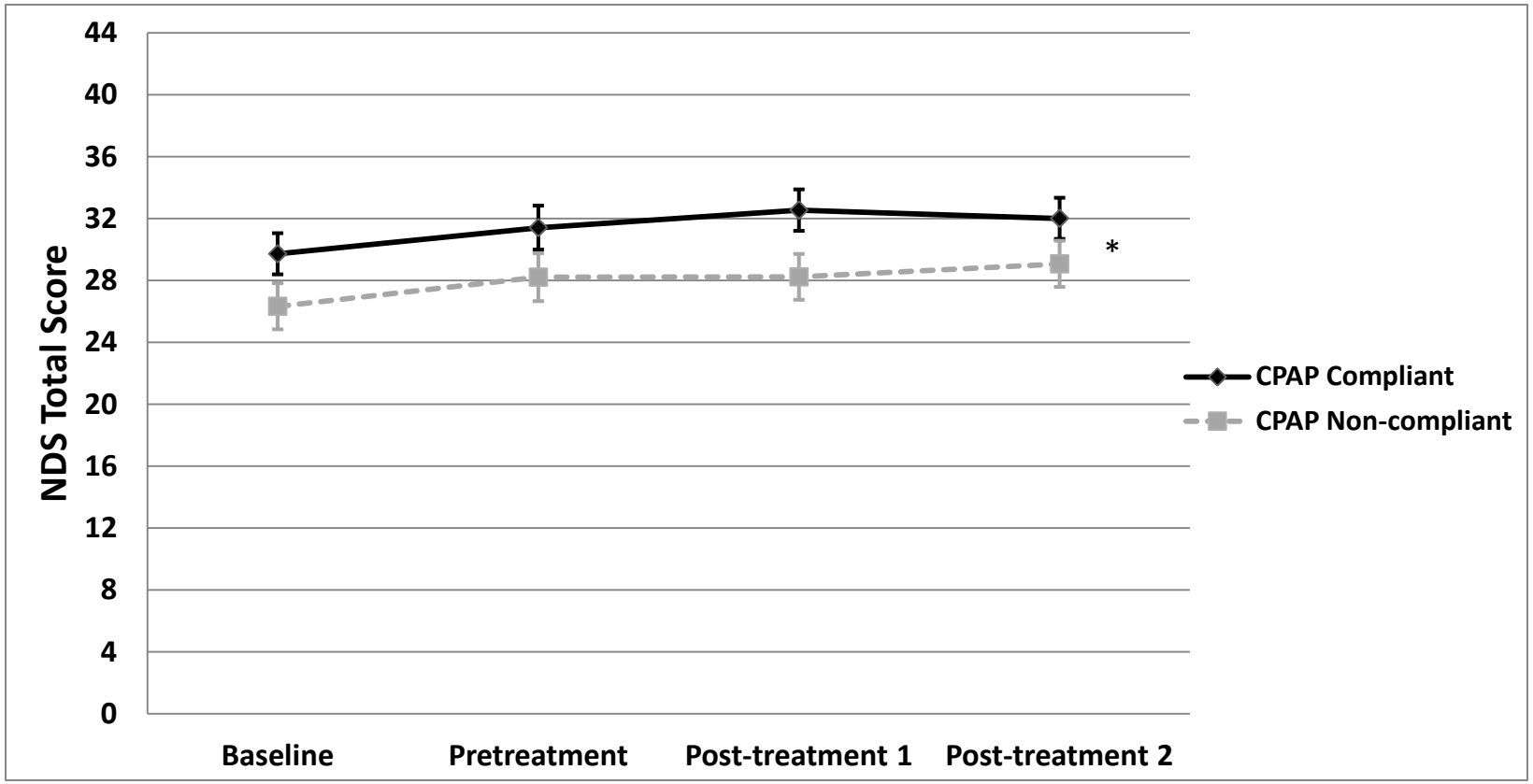

Figure 7. Change in nightmare distress over time for CPAP-compliant and non-compliant participants.

Note. Sample sizes for the CPAP-compliant group were: baseline, *n* = 24, pre-treatment, *n* = 14, post-treatment 1, n = 24, and post-treatment 2, *n* = 25. Sample sizes for the CPAP non-compliant group were: baseline, *n* = 22, pre-treatment, *n* = 13, post-treatment 1, n = 21, and post-treatment 2, *n* = 19.

*$p \leq .05$; **$p \leq .01$; ***$p \leq .001$.

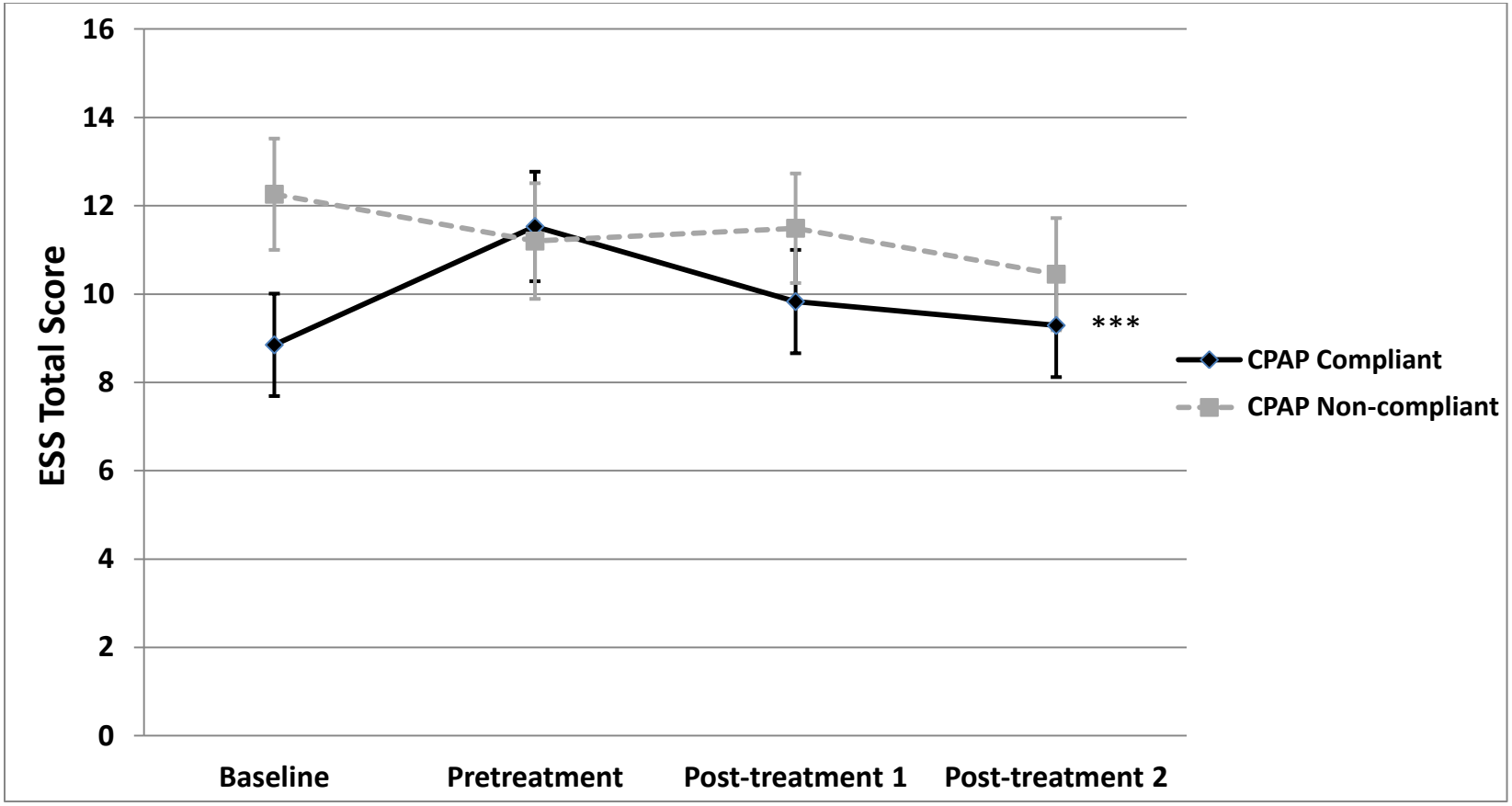

Figure 8. Change over time in daytime sleepiness for CPAP-compliant and non-compliant participants.

Note. Sample sizes for the CPAP-compliant group were: baseline, $n = 24$, pre-treatment, $n = 14$, post-treatment 1, n = 23, and post-treatment 2, $n = 24$. Sample sizes for the CPAP non-compliant group were: baseline, $n = 20$, pre-treatment, $n = 13$, post-treatment 1, n = 20, and post-treatment 2, $n = 18$.

*$p \leq .05$; **$p \leq .01$; ***$p \leq .001$

Non-equivalent Dependent Variable. A 2 (group) x 4 (time) mixed-model repeated measures analysis was conducted to compare participants who were CPAP-compliant to those who were non-compliant on the non-equivalent dependent variable conscientiousness as measured by the Big Five Inventory conscientiousness subscale. Age and relationship status were included as covariates in the model given the trend level positive associations with conscientiousness (Table 5). Analyses revealed a statistically significant main effect for compliance status ($F(1, 44.32) = 4.31$, $p = .044$); however, follow-up independent samples t-tests did not find a statistically significant difference between CPAP-compliant and non-compliant participants on self-reported conscientiousness at any of the four time points. No other statistically significant main effects or interactions emerged from the model. These findings suggest there was not a significant change in self-reported conscientiousness over time for either the CPAP-compliant or non-compliant participants.

Table 8

Mean Scores across Time for the Primary Outcome Variables by Group Status

Outcome variable	T1	T2	T3	T4	Within-subjects effect size (*d*)			
Group status	*M* (SD)	*M* (SD)	*M* (SD)	*M* (SD)	d_1	d_2	d_3	d_4
PCL-S total score								
CPAP-compliant	56.13 (15.65)	54.62 (13.57)	46.83 (15.73)	47.16 (15.40)	.96	.96	.70	.68
CPAP non-compliant	57.59 (15.31)	54.62 (13.57)	55.33 (14.42)	53.53 (13.79)	.40	.21	-.08	.15
PSQI Global Severity Index								
CPAP-compliant	13.76 (3.81)	14.25 (3.26)	9.63 (3.72)	10.56 (4.52)	1.17	.75	1.52	.92
CPAP non-compliant	14.95 (3.15)	14.08 (3.07)	13.20 (4.27)	12.29 (3.82)	.70	1.06	.51	.75
PSQI-PTSD Addendum								
CPAP-compliant	8.43 (5.11)	9.43 (5.76)	6.59 (5.35)	6.73 (5.24)	.40	.38	.81	.82
CPAP non-compliant	9.81 (5.50)	10.00 (5.54)	8.20 (5.58)	8.57 (5.58)	.59	.42	1.45	.67

(table continues)

Table 8 (continued)

Mean Scores across Time for the Primary Outcome Variables by Group Status

Outcome variable	T1	T2	T3	T4	Within-subjects effect size (*d*)			
Group status	*M* (SD)	*M* (SD)	*M* (SD)	*M* (SD)	d_1	d_2	d_3	d_4
Nightmare Distress Scale								
CPAP-compliant	29.73 (9.17)	28.15 (9.13)	28.32 (8.00)	27.00 (9.58)	.26	.35	-.03	.18
CPAP non-compliant	33.32 (10.56)	30.92 (9.01)	31.11 (11.68)	29.78 (9.23)	.33	.60	-.03	.23
Epworth Sleepiness Scale								
CPAP-compliant	9.25 (6.16)	12.91 (6.20)	9.59 (6.22)	9.33 (5.71)	-.07	-.02	1.05	2.13
CPAP non-compliant	12.05 (5.67)	11.77 (5.77)	11.50 (5.68)	11.28 (4.74)	.24	.16	.10	.15

Note. The within-subjects Effect size was calculated using Morris and DeShon's (2002) equation 8, which corrects for the dependence between repeated measure means. Baseline (T1) sample sizes: CPAP-compliant (n = 25) and CPAP non-compliant (n = 22). Pretreatment 1 (T2) sample sizes: CPAP-compliant (n = 15) and CPAP non-compliant (n = 13). Post-treatment 1 (T3) sample sizes: CPAP-compliant (n = 24) and CPAP non-compliant (n = 21). Post-treatment 2 (T4) sample sizes: CPAP-compliant (n = 25) and CPAP non-compliant (n = 19). d_1 = T1 & T3 within-subjects effect size; d_2 = T1 & T4 within-subjects effect size; d_3 = T2 & T3 within-subjects effect size; d_4 = T2 & T4 within-subjects effect size.

Table 9

Follow-up Comparison of the Estimated Marginal Means from the Mixed-model Repeated Measures Analyses Separated by Group Status

Outcome variable	T1	T2	T3	T4	
Group status	*EMM* (SE)	*EMM* (SE)	*EMM* (SE)	*EMM* (SE)	Significant comparisons
PCL-S total score					
CPAP-compliant	56.14 (2.97)	55.65 (3.15)	46.73 (2.97)	47.03 (2.96)	T1 > T3***, T1 > T4*** T2 > T3***, T2 > T4***
CPAP Non-compliant	57.59 (3.22)	52.07 (3.41)	55.03 (3.23)	53.46 (3.58)	T1 > T2**
Overall Sample	56.86 (2.19)	53.86 (2.30)	50.88 (2.36)	50.25 (2.20)	(Not interpreted)
PSQI Global Severity Index					
CPAP-compliant	13.76 (.77)	13.81 (.85)	9.60 (.77)	10.56 (.77)	T1 > T3***, T1 > T4*** T2 > T3***, T2 > T4***
CPAP Non-compliant	15.07 (.83)	13.86 (.90)	13.15 (.84)	12.13 (.88)	T1 > T4**

*$p < .05$. **$p < .01$. ***$p < .001$.

(table continues)

Table 9 (continued)

Follow-up Comparison of the Estimated Marginal Means from the Mixed-model Repeated Measures Analyses Separated by Group Status

Outcome variable	T1	T2	T3	T4	
Group status	*EMM* (SE)	*EMM* (SE)	*EMM* (SE)	*EMM* (SE)	Significant comparisons
Overall Sample	14.41 (.56)	13.84 (.62)	11.37 (.57)	11.35 (.58)	(Not interpreted)
PSQI-PTSD Addendum					
CPAP-compliant	8.73 (1.13)	9.11 (1.22)	6.54 (1.13)	7.04 (1.13)	(Not interpreted)
CPAP Non-compliant	9.28 (1.33)	8.06 (1.44)	7.32 (1.33)	8.44 (1.35)	(Not interpreted)
Overall Sample	9.00 (.86)	8.58 (.93)	6.93 (.86)	7.74 (.87)	T1 > T3**, T2 > T3**
Nightmare Distress Scale					
CPAP-compliant	29.73 (1.33)	31.41 (1.42)	32.54 (1.34)	32.01 (1.33)	(Not interpreted)
CPAP Non-compliant	26.33 (1.49)	28.12 (1.59)	28.23 (1.48)	29.07 (1.49)	(Not interpreted)
Overall Sample	28.03 (.98)	29.81 (1.03)	30.38 (.98)	30.54 (.98)	T1 > T3**, T1 > T4** T1 > T2**

*$p < .05$. **$p < .01$. ***$p < .001$. (table continues)

Table 9 (continued)

Follow-up Comparison of the Estimated Marginal Means from the Mixed-model Repeated Measures Analyses Separated by Group Status

Outcome variable	T1	T2	T3	T4	
Group status	*EMM* (SE)	*EMM* (SE)	*EMM* (SE)	*EMM* (SE)	Significant comparisons
Epworth Sleepiness Scale total					
CPAP-compliant	8.85 (1.16)	11.53 (1.24)	9.83 (1.17)	9.29 (1.17)	T2 > T1**
CPAP Non-compliant	12.26 (1.26)	11.20 (1.31)	11.49 (1.24)	10.45 (1.27)	
Overall Sample	10.55 (.86)	11.36 (.90)	10.66 (.85)	9.87 (.87)	(Not interpreted)
Conscientiousness Scale					
CPAP-compliant	34.36 (1.47)	35.34 (1.56)	36.04 (1.48)	35.99 (1.47)	(Not interpreted)
CPAP Non-compliant	30.63 (1.58)	31.49 (1.67)	32.14 (1.58)	31.65 (1.60)	(Not interpreted)
Overall Sample	32.49 (1.08)	33.41 (1.14)	34.09 (1.08)	33.81 (1.09)	(Not interpreted)

Note: Significant pairwise comparisons by group status were only interpreted if indicated by a significant compliance status x time interaction for the respective model. Significant pairwise comparisons for the overall sample were only interpreted if indicated by a significant main effect for time for the respective model. T1 = Baseline assessment; T2 = Pretreatment assessment; T3 = Post-treatment 1 assessment; T4 = Post-treatment 2 assessment.

*$p < .05$. **$p < .01$. ***$p < .001$.

Discussion

The purpose of this investigation was to expand on previous research that has examined the relationship between PTSD and sleep-disordered breathing. Specifically, this study prospectively examined posttraumatic stress symptoms following CPAP treatment for obstructive sleep apnea in a veteran population. We hypothesized that veterans compliant with their CPAP treatment would realize improvement in PTSD symptoms compared to pretreatment levels. Consistent with this hypothesis, and in line with previous retrospective work (Krakow et al. 2000; Youakim et al. 1998), our findings indicated that successful treatment of OSA with CPAP was associated with a subsequent reduction in PTSD symptoms. For veterans who were not compliant with CPAP treatment, no significant change in PTSD symptoms was observed.

Given this pattern of results, we also conducted follow-up analyses to investigate the possibility that the influence of successful CPAP treatment on PTSD symptoms may be more pronounced on one or more of the specific PTSD symptom domains (reexperiencing, avoidance/numbing, and hyperarousal). Interestingly, our mixed model repeated measures results did not show statistically significant effects for any one specific PTSD symptom domain. Although the interpretability of these follow-up analyses was limited by low statistical power, these results suggest that improvement in OSA is associated with a generalized pattern of reduction in PTSD-related distress, not specific to any cluster of symptoms. This finding is somewhat less intuitive given that PTSD-related sleep disturbance is specifically measured by the reexperiencing and hyperarousal symptom domains. One possible explanation for our findings is that OSA-related sleep disturbance may be functionally different than PTSD-related sleep disturbance. For instance, it is well-established in the literature that OSA is related to poor sleep quality,

sleep maintenance, and daytime fatigue (Phillipson, 1993; Wright et al., 1997) whereas the most commonly reported PTSD-related sleep dysfunction is difficulty with sleep initiation, sleep maintenance, and nightmares. Thus, the improvement in sleep processes associated with the successful treatment of obstructive sleep apnea may not overlap entirely with PTSD specific sleep disturbance. Another possibility is that the improvement in sleep quality associated with successful treatment of OSA may be associated with subsequent improvements in psychological functioning more generally and may not be entirely specific to PTSD. Although this latter hypothesis cannot be ruled out, it is less likely given the significantly elevated rates of OSA observed in samples of PTSD compared with general population samples (Krakow et al., 2000; Krakow et al., 2001; Orr et al., 2010; Youakim et al., 1998).

Nonetheless, the present findings provide additional support to previous retrospective work suggesting that the successful treatment of OSA with CPAP may also reduce posttraumatic stress symptoms. These results are encouraging and may have important clinical implications. For example, treatment providers working with Veterans with PTSD may wish to incorporate appropriate OSA screening measures in addition to assessing for more behaviorally-based (PTSD-related) sleep disturbance. For veterans with untreated concurrent obstructive sleep apnea, PTSD symptoms may be exacerbated and sleep disturbance may not fully resolve with PTSD treatment alone. In addition, sleep processes have been proposed as an important factor in emotional memory processing and emotion regulation (van der Helm & Walker, 2009). Research suggests that the ability to encode new information as well as consolidate new information into long-term memories is heavily sleep-dependent processes (Walker, 2009; Walker & Stickgold,

2004). Given that several current PTSD treatments hypothesize that symptom resolution occurs as a result of successful traumatic memory (re)processing (see Prolonged Exposure - Foa, Hembree, & Rothbaum, 2007; Cognitive Processing Therapy - Resick, Monson, & Chard, 2008), untreated concurrent conditions such as OSA that impact sleep processes may result in the reduced efficacy of these trauma processing oriented PTSD treatments. Treatment providers working with individuals with PTSD may enhance treatment outcomes by ensuring adequate identification and treatment compliance for physical sleep disorders like OSA.

Unfortunately the research highlighted across these areas is ongoing and the mechanisms underlying the relationship between PTSD and OSA are unknown. At present, theories regarding the possible impact that OSA has on PTSD treatment are speculative. Substantially more research is needed to better define the possible mechanisms linking PTSD and OSA. It is possible that pre-existing or sub-threshold OSA may place individuals at greater risk to develop PTSD following a traumatic event or conversely, that individuals experiencing PTSD-related sleep disturbance may confer increased risk to develop OSA.

Other Aspects of Psychological Functioning

In addition to PTSD symptoms, the current study prospectively evaluated several other aspects of psychological functioning among veterans receiving CPAP treatment for OSA. The first factor examined was self-reported sleep quality. We hypothesized that veterans who complied with the CPAP treatment would realize an improvement in perceived sleep quality relative to their respective pre-treatment ratings. Mixed-model analyses revealed a statistically significant effect of time, such that the overall sample reported an improvement in sleep quality from pre-to-post treatment. This finding was

somewhat surprising considering participants who were not CPAP-compliant would not be expected to see a reduction in sleep disturbance related to OSA. Although the improvement in sleep quality for CPAP non-compliant participants was somewhat less pronounced compared to the reduction for CPAP-compliant participants, it was statistically significant. A similar finding emerged for the second factor, PTSD-related sleep dysfunction, whereby the overall sample reported an improvement in PTSD-related sleep dysfunction from pre-to-post treatment. No significant differences were found for individuals that were CPAP-compliant compared to those who were non-compliant.

One possible explanation for these findings is that participants may have altered their sleep habits in ways other than implementing the CPAP treatment. For example, all participants met with a member of the Neurology Clinic staff following their overnight sleep study. In addition to reviewing the results of the study and diagnostic considerations, these veterans were given information on improving sleep hygiene. Participants were also encouraged to make lifestyle changes, including losing weight and reducing alcohol consumption, as ways to decrease OSA severity. It is possible that participants, who chose not to utilize their CPAP treatment at the optimal therapeutic level, implemented other changes to their sleep routine or lifestyle which may have led to an improvement in perceived sleep quality. Although the current study did not evaluate behavioral modifications to the sleep routine, it is possible that CPAP utilization, improvements in sleep hygiene, or a combination of both resulted in the overall sample reporting increased sleep quality and decreased PTSD-related sleep dysfunction. Another possibility is that these findings are explained, simply by the phenomenon of regression to the mean.

Nonetheless, it is encouraging that in a sample of veterans diagnosed with OSA in addition to reported PTSD-related distress, that improvements in sleep can be realized. It is important to note that the current study was underpowered to detect differences between the groups. Future studies examining sleep indices among individuals with OSA and PTSD may wish to simultaneously monitor multiple aspects of sleep behavior in addition to CPAP compliance. In addition, a longer-term follow-up period would be helpful to determine whether or not the observed change in perceived sleep quality found in this study represent sustainable improvement across time.

In addition to measures of sleep quality, this study examined a third factor, nightmare-related distress. We hypothesized that participants who were CPAP-compliant would report decreases in nightmare-related distress compared to pre-treatment report. Contrary to our hypothesis, results did not show a significant post-treatment improvement in nightmare-distress compared to pretreatment report. Of note, a significant increase in nightmare distress was reported from the baseline time point to pretreatment time point for the full sample; however, no significant differences were found between the pretreatment time point and either post-treatment time point. This finding was unexpected, given that previous cross-sectional research suggested that successful treatment of OSA with CPAP was associated with subsequent reductions in nightmare frequency and intensity (Krakow et al., 2000 & Youakim et al., 1998). One hypothesis proposed to explain these previous results is that the dysfunction in REM stage sleep attributed to OSA may be associated with an increase in nightmare experience for individuals with PTSD, as dreaming/nightmares is more frequently reported following REM stage sleep awakening compared with awakening from non-REM sleep (Hobson

1988). The current results do not support previous cross-sectional findings as no significant change in nightmare distress was observed for CPAP-compliant individuals who would be expected to demonstrate decreased awakenings during REM stage sleep following successful OSA treatment.

Our findings are more consistent with a literature review conducted by Whittman and colleagues (2007) that concluded the occurrence of posttraumatic nightmares throughout the sleep cycle could not be explained by exclusively by altered REM sleep, thus the improvement in REM stage sleep density associated with CPAP treatment would not be expected to decrease nightmare distress related to the occurrence in nightmares across other sleep stages. It is important to note, however, that the Nightmare Distress Scale used in the current study measures nightmare-related distress and not the frequency in which nightmares occurred. Thus it is unclear if CPAP-compliant individuals in the sample may have experienced fewer nightmares producing an equivalent level of distress or if no change in the experience (frequency or intensity) of nightmares occurred overall. Furthermore, comparisons regarding change in nightmare distress from pre-to-post CPAP treatment may have limited by a low rate of nightmare endorsement at the baseline (M = 31.39; SD = 9.88) and pretreatment (M = 29.54; SD = 9.00) time points. The rate of nightmare endorsement in this study may reflect the fact that the sample was not recruited based on a diagnosis of PTSD, but rather comprised of individuals that endorsed a range of posttraumatic stress symptoms.

A final factor examined by this study was daytime somnolence. Subjectively reported daytime somnolence is a well-recognized symptom of untreated OSA. Research has long demonstrated that successful resolution of OSA with CPAP results in an almost

immediate reduction of daytime somnolence (Engelman et al., 1999; Jenkinson et al., 1999; Kribbs et al., 1993). Our analyses were inconsistent with this line of research as results did find a change in daytime somnolence among CPAP-compliant participants. Although unanticipated, there are several possible explanations for the current results. First, as previously acknowledged, the current study was statistically underpowered and it is possible that if a daytime somnolence effect was present, it was too small for the current sample size to detect. Another possibility is that veterans in the CPAP-compliant group, although averaged ≤ 4 hours of use, may not have used their CPAP at the optimal therapeutic level every night. To this point, a study conducted by Kribbs and colleagues (1993) found that sleeping without CPAP for a single night resulted in almost complete reversal of daytime alertness gains derived from sleeping with CPAP. Follow-up inspection of the current sample revealed only 32% (n = 8) of the CPAP-compliant group reported utilizing the CPAP machine at the optimal therapeutic level for all 28 days of the follow-up period. A final possibility is that in a proportion of individuals with OSA and concurrent psychopathology, OSA may not be the principal cause of daytime somnolence (Antic et al. 2010). For example, in a general population sample, depression was found to be the most significant risk factor for daytime somnolence followed by BMI, age, sleep duration, diabetes, smoking, and lastly OSA (Bixler et al., 2005). Posttraumatic distress has not been directly examined in this regard; however, given the findings in the Bixler (2005) study, it is possible that daytime somnolence may be influenced by psychological conditions like PTSD and successful treatment of OSA may not alleviate more psychopathological-related daytime somnolence.

Cross-sectional Comparisons by CPAP Compliance Status

The current study examined several factors thought to be associated with CPAP-compliance and non-compliance. First we examined coping strategies and found that participants who were not CPAP-compliant endorsed using both the avoidant strategy of "venting" as well as "instrumental support" coping at significantly higher rates than CPAP-compliant participants. In regard to venting, research has consistently linked avoidance style coping, including venting, to worse functional health outcomes including more severe PTSD symptoms (Aldwin & Revenson, 1987; Ashton et al., 2005; Penley, Tomaka & Wiebe, 2002; Tiet et al., 2006). Given this relationship, it is not surprising that we observed a correlation between higher levels of venting, CPAP-noncompliance, and sustained posttraumatic stress symptoms. Conversely, CPAP non-compliant participants also reported using a significantly higher level of instrumental support compared to CPAP-compliant participants. Instrumental support is considered to be a problem-focused (or approach-focused) coping strategy, which has been found to be associated with more positive health-related outcomes (Penley et al., 2002). Instrumental support largely consists of providing or seeking out tangible goods and services to assist with problem solving. Research examining spousal support in relation to CPAP compliance has found that an individual's perception of spousal support only predicted compliance in individuals with severe OSA (Baron et al., 2011). Furthermore, this study found that perceived spousal pressure to use CPAP was associated with lower CPAP adherence rates. Although perceived support or pressure for CPAP use was not measured in the current study, the higher rates of instrumental support used among CPAP non-compliant

individuals warrant further investigation given the low rates of CPAP compliance observed in individuals with OSA.

We also compared the endorsement of side effects associated with CPAP treatment among participants who were CPAP-compliant and non-compliant. Participants who were not CPAP-compliant reported significantly more problems across several areas including more problems with mask irritation and more CPAP-related sleep disturbance. These findings are consistent with previous research suggesting that greater endorsement of perceived CPAP side effects is positively associated with treatment noncompliance (Engleman, Martin, & Douglas, 1994).

Methodological Considerations

This study had several notable limitations that should be considered when interpreting the current findings. As previously noted the sample size was small and likely limited the strength of our analytic approach to further examine change in PTSD symptoms and other aspects of psychological functioning. Previous research has demonstrated the difficulties of conducting longitudinal research with clinical populations (Coen & Patrick, 1996; Patel, Doku, & Tennakoon, 2003), which include difficulty recruiting and retaining participants. Although the high retention (95.7% at post-treatment 1 time point & 93.6% at post-treatment 2 time point) was a strength of the current study, we also sought to limit the impact of a small sample size and attrition by utilizing a mixed-modeling repeated measures statistical approach. Mixed-effect models provide a flexible framework for repeated measures analyses by utilizing all available data for each participant (Gueorguieva & Krystal, 2004). In addition, we took precautions to decrease the likelihood of detecting Type-I error by subjecting all post-hoc model comparisons to a Bonferonni-Holm adjustment (Holm, 1979).

Another limitation of the current study is the lack of random assignment to study groups. This research study was designed to be quasi-experimental in nature in order to examine change in PTSD symptoms following CPAP treatment initiation for OSA as prescribed by the VA Neurology Sleep Clinic. Study groups (CPAP-compliant or CPAP non-compliant) were created following the conclusion of all data collection. Although random assignment to study groups is preferable, temporal and logistical restraints, in addition to the potential adverse health consequences of randomly assigning individuals to use or not use their CPAP treatment limited this study's internal validity. To attend to this issue and potentially reduce threats to internal validity, the current study implemented a non-equivalent dependent variable as an additional measure of control on the impact of CPAP treatment on PTSD symptoms. Results found that participants did not report a change in conscientiousness level (non-equivalent dependent variable) from pre-to-post treatment. The lack of observed change in reported conscientiousness provided additional strength to the finding that successful treatment of OSA is associated with reductions in PTSD symptoms.

Some additional limitations worth noting concern the generalizability and stability of current findings. Of the 489 participants invited to participate in this project, 71 (14.5%) completed the baseline assessment. It is possible that this self-selected sample is not characteristic of the veteran population more generally. The sample was predominantly male (89.2%), thus it is unclear if these results would hold consistent among female veterans. Additionally, this study measured posttraumatic stress symptoms and not posttraumatic stress disorder. Although we used a well-known and validated measure of PTSD symptoms (PCL-S) we did not have the ability to conduct structured

clinical interviews to make formal PTSD diagnoses. Future studies should consider using a diagnostic measure of PTSD like the Clinician Administered PTSD Scale (Blake, Weathers, Nagy, & Kaloupek, 1995) as it would allow for diagnostic comparisons as well as a more nuanced examination of change in PTSD symptoms. In regard to CPAP compliance, participants in this study self-reported usage. In two cases, follow-up with objective measurement at three months indicated exaggerated CPAP use. Given the self-reported follow-up period (1-month) represented approximately one-third of the objective SmartCard review (3-month), it is possible other participants may have exaggerated their use and went undetected. To avoid this limitation and increase sensitivity to the level of CPAP treatment adherence, future studies incorporate objective measurement of CPAP utilization across follow-up periods. Finally, the current study monitored participants across a four week follow-up period. The results suggested that CPAP compliance was associated with a decrease in posttraumatic stress symptoms; however, it is unclear if this reduction in symptoms would be maintained through continued CPAP utilization. Future studies should include longer follow-up periods in order to evaluate the stability of the current findings.

Conclusions

Although few published studies have specifically examined the relationship between PTSD and obstructive sleep apnea, the available data suggests a strong association between these two diagnoses. The current study adds to this literature by demonstrating that successful treatment of OSA is associated with a reduction in PTSD symptoms over time. Future research should attempt to expand these findings by 1) attempting to replicate the current results using an experimental methodology; and 2)

attempt to elucidate the possible mechanisms underlying the relationship between PTSD and OSA.

The current findings also have important clinical considerations. It appears that individuals with PTSD are at elevated risk for concurrent OSA and may benefit from identification and treatment of this physical sleep disorder. It is important for treatment providers working with individuals with PTSD or OSA to expand their assessment of sleep dysfunction to include both the evaluation of primary physical sleep disorders as well as secondary symptoms of a psychiatric diagnosis. Without a more complete diagnostic understanding of an individual's sleep dysfunction (i.e., identifying and treating only physical sleep disorders or only psychiatric symptoms), it is possible that an individual will continue to experience sleep difficulty with the potential to undermine treatment for one or both conditions.

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