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Improving Sleep and PTSD Outcomes in Service Members: A Randomized Controlled Trial Examining the Long-Term Effects of an Integrated Treatment

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Highlights

- Trauma Management Therapy (TMT) integrates PTSD and sleep treatment
- We compared TMT to Compressed Prolonged Exposure in active-duty service members
- TMT group: better sleep efficiency and sleep onset latency at 3 months
- TMT group: better sleep quality, efficiency, and wake after sleep onset at 6 months
- Findings support adding sleep-focused components to PTSD treatment protocols

Abstract

Trauma-induced sleep disturbances often persist after successful post-traumatic stress disorder (PTSD) treatment. While integrated protocols combining sleep and exposure-based treatments may maximize outcomes, prior studies are limited and have largely relied on subjective sleep measures or failed to include long-term follow up assessments. Active-duty service members with PTSD ($n = 82$) were randomly assigned to Compressed Prolonged Exposure (CPE) treatment or Trauma Management Therapy (TMT), which integrates exposure therapy with sleep hygiene training and other skills-based interventions. PTSD symptoms and actigraphy-based sleep were measured at baseline, post-treatment, 3- and 6-month follow-up and data were compared between groups and across time. Post-treatment, both groups showed negligible to small changes in sleep compared to baseline. However, the TMT group evidenced improvements in most sleep parameters by the 3- and 6-month follow-ups, while sleep health generally worsened in the CPE group over time. Between groups, those randomized to TMT exhibited better sleep efficiency ($g = 0.24$) and onset latency ($g = -0.34$) at 3-months follow-up, and better sleep quality ($g = 0.70$), efficiency ($g = 0.51$), and wake after sleep onset ($g = -0.52$) at 6-months follow-up. Within both treatment groups, poorer sleep at the 6-month follow-up was correlated with greater PTSD symptom severity measured at the same time point. Integrated treatment for sleep and PTSD produced superior objective sleep outcomes compared to exposure alone, with the most meaningful improvements in sleep observed 6-months after treatment completion. Several critical directions for future studies are discussed.

Introduction

Post-traumatic stress disorder (PTSD), a prevalent and debilitating condition affecting many active-duty military personnel, involves impairing symptoms of hyperarousal, hypervigilance, negative mood, and avoidance. Sleep disturbances, especially insomnia and nightmares, are also pervasive in treatment seeking samples (Germain, 2013). It is now understood that disturbed sleep is not merely a symptom or consequence of PTSD, but constitutes a predisposing, precipitating, and perpetuating factor for the disorder (Spoormaker & Montgomery, 2008). Understanding the relationship between PTSD and sleep disturbances, especially within the context of treatment, is essential for the development of more effective treatment strategies with outcomes that endure over time.

Association of Sleep and PTSD symptoms during treatment

While sleep disruption itself is an impairing clinical phenomenon, the presence of sleep disturbances in the context of psychiatric disorders can exacerbate symptoms. Both animal and human studies suggest poor sleep quality exacerbates and maintains PTSD symptoms via disruptions to emotional processing, emotional memory consolidation, and fear extinction (Germain, 2013). In clinical samples, residual insomnia after PTSD-focused treatment has been shown to predict poorer global functioning (Brownlow et al., 2016). Available research also suggests that disrupted sleep quality may worsen responses to PTSD treatment. Poor self-reported sleep quality at pre-treatment baseline has been associated with higher PTSD symptoms during treatment (Sullan et al., 2021; Taylor et al., 2020). However, Lommen et al. (2016) only found this relationship in patients with comorbid depression and PTSD, and other research has not found any significant relationships between baseline sleep quality and treatment outcomes (Sexton et al., 2017). Individuals with persistent sleep difficulties (i.e., self-reported insomnia that does not improve through the course of treatment) have also been shown to have more severe PTSD, anxiety, and depression symptoms at treatment conclusion and at 6-month follow-up (Belleville et al., 2011). Finally, residual self-reported insomnia symptoms after PTSD treatment have been associated with higher rates of relapse at 3- and 9-month follow-ups (Kartal et al., 2021; Lommen et al., 2016).

Overall, ongoing research points to sleep disturbance as a transdiagnostic and precipitating risk factor within PTSD treatment, but more research is needed. In particular, prior research has primarily relied on subjective sleep report, which can provide an incomplete or biased characterization of sleep (Lehrer et al., 2022). Subjective ratings are influenced by recall and salience heuristics, mood and symptom severity, and individual differences in sleep perception, and thus may reflect perceived sleep disturbance rather than sleep continuity per se (Arditte Hall et al., 2023; Kobayashi et al., 2012). Objective assessment (e.g., actigraphy) complements self-report by providing multi-night, ecologically valid estimates of sleep timing and continuity (e.g., total sleep time, sleep onset latency, wake after sleep onset) that are less dependent on memory and appraisal and can quantify sleep fragmentation and change over time in ways that questionnaires may not capture (Lehrer et al., 2022; Smith et al., 2018).

For behaviorally oriented intervention research, actigraphy is particularly valuable because it captures sleep in naturalistic settings across multiple nights, allowing investigators to quantify modifiable behavioral targets (e.g., prolonged sleep onset, sleep fragmentation) and

sensitively track trajectories of change that may be missed by retrospective self-report. This distinction is especially relevant in PTSD. Specifically, while subjective report of sleep disruption is considered a hallmark of PTSD and reliably differentiates between PTSD and control groups, studies using objective sleep measures have not always found differences compared to controls (Lewis et al., 2020; Yeh et al., 2021). Exploration utilizing objective sleep measures is therefore necessary to more fully understand the relationship between sleep disturbance and PTSD treatment response.

Impact of PTSD Treatment on Sleep Disturbances

Evidence-based treatments for PTSD such as prolonged exposure (PE) are highly effective in reducing most symptoms of the disorder but do not necessarily result in clinically meaningful improvements in sleep (Brownlow et al., 2016; Gutner et al., 2013; Lancel et al., 2021; Larsen et al., 2019). While some studies have failed to find differences in subjective and objective sleep quality following PTSD treatment (Haynes et al., 2020; Taylor et al., 2020), others suggest that PTSD treatment produces statistically significant but not clinically meaningful improvements to self-reported insomnia, nightmares, and sleep quality (Gutner et al., 2013). Insomnia in particular is the most common residual symptom following PTSD treatment (Larsen et al., 2019), with between 48-67% (Pruiksma et al., 2016; Tanev et al., 2022; Zayfert & DeViva, 2004) of patients continuing to report insomnia at clinically significant levels following otherwise successful treatment. Whether improvements in sleep that do occur are maintained over time is also unclear, as the majority of research studies have not included long-term follow-up, and research that has mixed results. For example, Gutner et al. (2013) found that treatment-related improvements in sleep remained at 9-month follow-up (Gutner et al., 2013), whereas Belleville et al. (2011) reported that initial improvements in sleep following PTSD treatment were followed by significantly worse sleep by 6-month follow-up (Belleville et al., 2011). Overall, many patients continue to experience impairing residual sleep problems even after successful PTSD treatment (Belleville et al., 2011; Pruiksma et al., 2016) which is especially problematic when considering sleep disturbances' associations with higher PTSD and depression relapse rates (Kartal et al., 2021). Accordingly, researchers have emphasized a need for more comprehensive and/or integrated intervention approaches that address both daytime and nighttime PTSD symptoms (Miller et al., 2020).

Treatment Targeting PTSD and Sleep Disturbance

In light of rates of sleep disturbance in PTSD sufferers, surprisingly few studies have examined treatments that target both sleep and other core PTSD symptoms. Among a sample of returning U.S. veterans and post-deployment personnel with PTSD, Walters et al. (2020) compared participants randomized to PE followed by imagery rehearsal therapy (IRT) for nightmares and cognitive-behavioral therapy for insomnia (CBT-I), or PE followed by supportive care. Consistent with prior studies, PE alone did not produce meaningful improvements in sleep, however PE followed by IRT and CBT-I led to meaningful improvements in nightmares and subjective insomnia symptoms, respectively, relative to the supportive care group. However, objective sleep measurement based on actigraphy did not reveal significant improvements at post-treatment in the IRT and CBT-I group. In another study where cognitive processing therapy (CPE) for PTSD was either implemented alone or preceded or followed with a combination of CBT-I and IRT, both combined treatments resulted in significant

improvement in subjectively reported sleep disruption compared to CPE alone (Taylor et al. (2023). Unfortunately, however, objective sleep outcomes were not assessed.

Colvonen and colleagues (2019) conducted a pilot study examining an integrated treatment including PE and CBT-I in a sample of veterans diagnosed with insomnia and PTSD. The 15-week protocol included overlapping treatments until week 6, at which point CBT-I components ended and the remaining protocol focused solely on imaginal and in-vivo exposures. Integrated treatment resulted in clinically meaningful improvements in both PTSD and insomnia, including increases in sleep efficiency and total sleep time based on both sleep diaries and actigraphy. Based on these pilot data, the authors suggest that integrated sleep and PTSD treatments may provide an optimal method for augmenting existing treatments for PTSD, though more research is ultimately needed (Colvonen et al., 2019).

Present Study

To address existing gaps in knowledge, we examined whether two evidence-based PTSD interventions produced sleep-related improvements among military personnel. Trauma management therapy (TMT), a 3-week intensive intervention incorporating exposure therapy, sleep hygiene training, anger management, brief behavioral activation for depression, and social reintegration (Beidel et al., 2019), was compared to compressed prolonged exposure (CPE), which included two weeks of daily PE sessions with the same homework format as typical PE (Foa et al., 2018). Our first aim was to estimate the degree to which TMT and/or CPE impacted sleep problems in those with PTSD based on one week of actigraphy at various timepoints. Specifically, we examined both within and between group changes in sleep quality, duration, efficiency, onset latency, and wake after sleep onset from pre- to post-treatment, 3-month follow-up, and 6-month follow-up. Within the TMT group, which included sleep hygiene training, we expected to observe small to moderate size improvements in sleep, whereas no changes in sleep were anticipated in those treated with CPE.

Our second aim was to evaluate relationships between sleep outcomes and PTSD symptoms at each time point in both treatment groups. We hypothesized that for both the TMT and CPE groups, improvements in sleep would be associated with reductions in other PTSD symptoms. Given the inclusion of a sleep-related component in TMT, we also expected to observe stronger associations between sleep and PTSD symptoms in the TMT compared to the CPE group.

Methods

Participants & Procedures

Data used in this study were part of a multi-center randomized controlled trial comparing TMT and CPE using a parallel group design. Inclusion criteria involved being an active-duty military personnel seeking treatment for PTSD, receiving a PTSD diagnosis using the Clinician Administered PTSD Scale for DSM-5 (CAPS-5), being able to fluently write and read in English and participate in the treatment, and taking a stable dosage of medication (i.e., no changes in psychiatric medication dosage, if applicable, during the 2–3 weeks of active treatment). Exclusion criteria included having a severe comorbid substance use disorder (unless use was

under control for at least 2 weeks, as indicated by Alcohol Use Disorders Identification Test [AUDIT; Saunders et al., 1993] scores showing no problematic drinking), a diagnosis of schizophrenia, other psychotic disorder, or antisocial personality disorder, a diagnosis of moderate to severe traumatic brain injury, or being on benzodiazepines and not wishing to discontinue use. Participants who were pregnant or had acute cardiac difficulties (i.e., angina, myocardial infarction, or severe hypertension) required medical clearance to participate.

A total of 116 eligible participants based on the inclusion and exclusion criteria were recruited at three military installations: Naval Medical Center Portsmouth, Naval Medical Center Camp Lejeune, and Dwight D. Eisenhower Army Medical Center. Participants were referred to the treatment study from mental health clinics at each military installation during 2018-2024. Of those enrolled in the study, 22 participants decided not to start treatment after randomization (i.e., no baseline assessments were completed and no actigraphy data were collected; CPE: $n = 8$; TMT: $n = 14$), resulting in 94 participants with baseline data who initiated treatment (CPE: $n = 54$; TMT: $n = 40$; see Figure 2 for full CONSORT diagram). Reasons for drop out included inability to adhere to the treatment protocol, reassignment to another duty station, or no reason given. Since the primary outcomes in the current study were based on actigraphy and at least 5 nights of actigraphy data are required for reliable sleep assessment (Aili et al., 2017), 12 participants were removed from the sample due to inadequate actigraphy data (<5 nights; CPE: $n = 7$; TMT: $n = 5$), resulting in a final sample size of $n = 82$.

Participants were randomly assigned to either TMT or CPE using a stratified permuted block method. Block size was varied (but with a minimum of 4 participants) to minimize the likelihood that the blind will be broken prior to the start of treatment. A minimum of 4 was needed to ensure a sufficient number of participants for the group component of TMT. Randomization was stratified by clinical site and carried out using a web-based system developed by the project biostatistician.

To address comparability in treatment dose, the protocols were designed to be equivalent in total therapeutic time, including both in-clinic contact and between-session homework. Homework adherence was monitored daily in individual (and, for TMT, group) sessions. Completion of less than 75% of assigned homework was defined a priori as protocol noncompliance and would have triggered removal from the treatment program; no participants were removed for homework noncompliance. Those assigned to the TMT group underwent a three-week intensive outpatient treatment which consisted of daily exposure therapy (augmented with virtual reality) and daily group treatment including sleep hygiene training, anger management, brief behavioral activation for depression, and social reintegration (see Beidel et al., 2019; Beidel et al., 2017 for a detailed description of TMT). The original TMT program did not include a sleep-specific intervention. However, findings from Beidel et al. (2019) indicated that sleep remained largely unchanged and continued to be problematic following both TMT and exposure therapy alone. Given the three-week treatment window and the ability to devote only two sessions to sleep, we consulted with the fifth author (a diplomate in behavioral sleep medicine) to incorporate brief, feasible strategies that could be implemented effectively within this timeframe. Those assigned to the CPE group participated in two weeks of daily PE sessions using the same homework that is used in typical PE sessions (see Foa et al., 2018 for a detailed description of CPE).

Measures

Assessment of Posttraumatic Stress Disorder

PTSD diagnosis and symptom severity were assessed by both the self-administered PTSD Checklist for DSM-5 (PCL-5; Blevins et al., 2015) and by trained clinicians using the Clinician Administered PTSD Scale for DSM-5 (CAPS-5; Weathers et al., 2018). PCL-5 is a 20-item self-reported checklist utilizing a 5-point Likert scale. Two items on the scale designed to assess sleep were excluded from the total score for the current study; this approach has been used previously in PTSD–sleep research and sensitivity analyses (Colvonen et al., 2019). The PCL-5 has good test-retest reliability, convergent validity, divergent reliability, and internal consistency (Cronbach's $\alpha = .94$; Morrison et al., 2021). In our sample, both the full scale ($\alpha = .81$) and the version excluding sleep items ($\alpha = .80$) showed good internal consistency, with the latter used for all analyses to control for confounding relationships. The CAPS-5 is a 30-item semi-structured interview and is considered the gold standard in PTSD assessment. The CAPS-5 assesses the frequency and intensity of each symptom of PTSD and provides a composite rating for each item. Clinicians on site conducted the CAPS-5 interview with participants at each assessment point. A recent study examining the use of the CAPS-5 in military veterans found high interrater reliability for both PTSD diagnosis ($\kappa = .78$ to 1.00) and total severity score (ICC = .91), good test-retest reliability for both PTSD diagnostic status ($\kappa = .83$) and severity score (ICC = .78), and good internal consistency for the severity items ($\alpha = .88$; Weathers et al., 2017).

Assessment of Sleep

Sleep was assessed using Readiband 4™ actigraphs (Fatigue Science), which have been shown to have high sensitivity and medium specificity in detection of sleep when compared to polysomnography (Chinoy et al., 2021). Participants wore the actigraphs for one week prior to the beginning of treatment (pre-treatment), every day during active treatment, immediately after treatment (post-treatment), and for one week prior to the 3- and 6-month follow-up assessments. Readiband actigraphy was used to generate five sleep metrics: total sleep time (TST), sleep efficiency (SE), sleep onset latency (SOL), wake after sleep onset (WASO), and sleep quality (SQ). Actigraphy provides an objective estimate of sleep–wake patterns using the presence/absence of wrist movement and a proprietary scoring algorithm.

TST reflects sleep quantity and was determined using the average total number of hours spent asleep over the last 7 days. SE reflects the consolidation of sleep while in bed and was calculated using total time spent asleep (from sleep onset to final wake time) as the numerator and total time spent in bed (from attempting to sleep until wakefulness) as the denominator (i.e., higher SE indicates more continuous sleep and less time awake in bed). SOL reflects difficulty initiating sleep and was quantified using the average time it takes to transition from wakefulness to sleep at night (longer SOL indicates taking longer to fall asleep). WASO reflects sleep fragmentation/maintenance difficulty and was estimated as the average awake minutes between sleep onset and wake time (higher WASO indicates more disrupted sleep).

SQ ratings (scale from 1 to 10) provide an overall summary of sleep continuity. SQ ratings (1–10) provide an overall summary of sleep continuity. In Readiband scoring, SQ is a vendor-derived index based primarily on actigraphy-estimated awakenings per hour and WASO,

such that fewer awakenings and less time awake yield higher scores. The platform converts these indices to the 1–10 scale using a proprietary, norm-referenced algorithm; vendor documentation provides normative anchors for the inputs (e.g., awakenings/hour: good < 0.37, ok 0.38–0.67, poor > 0.68; WASO: normal < 20 min, ok 21–40 min, poor > 41 min). Because SQ is a vendor-derived composite that incorporates WASO (and awakenings per hour), we expected SQ to correlate with WASO; however, SQ is not redundant with WASO because it also reflects sleep fragmentation (awakenings/hour) and uses a proprietary norm-referenced transformation rather than raw minutes. In the current study, SQ scores of 8 or higher were categorized as good, whereas scores of 5 or lower indicated poor sleep quality.

Interventions

Trauma Management Therapy (TMT)

TMT is a multi-component behavioral treatment combining both individual and small group (typically four patients) sessions with a total of 29 sessions in the span of three weeks (Beidel et al., 2019; Beidel et al., 2017). Individual sessions are comprised of one psychoeducation and 14 imaginal exposure therapy sessions. These sessions incorporated the VR system known as Bravemind (USC Institute for Creative Technologies, 2005), which allows the therapist to design a VR scenario that matches the patient's Criterion A traumatic event, including selecting the relevant sights, sounds and smells that trigger their distress. Group sessions are composed of 14 sessions led by two therapists with a focus on the social and emotional rehabilitation component of the treatment, including social reintegration, anger management, sleep hygiene training, and behavioral activation.

Within the group program, two sessions are dedicated specifically to sleep (Beidel et al., 2017). The first sleep session provides psychoeducation focused on identifying current sleep habits and sleep-related difficulties; circadian rhythm and environmental/behavioral cues that can facilitate or interfere with sleep; calculating sleep efficiency; and behavioral sleep strategies consistent with stimulus control (i.e., “dos and don'ts” for strengthening the bed–sleep association). Participants were instructed to track sleep using daily sleep logs until the subsequent session. In the second sleep session (Week 2), therapists reviewed sleep logs with participants, reinforced adherence and improvements, and problem-solved barriers to implementing sleep strategies. Participants were also introduced to relaxation skills (e.g., deep breathing and progressive muscle relaxation) for difficulty initiating sleep or repeated nighttime awakenings and were provided handouts for home practice. Finally, based on weekly sleep efficiency derived from logs, therapists implemented brief, goal-directed adjustments to time in bed (TIB): TIB was reduced by 15 minutes when sleep efficiency was <80%, and increased by 15 minutes when sleep efficiency was >85% in the context of continued daytime sleepiness. Homework is assigned at the end of individual and group sessions and is an integral part of the treatment.

Compressed Prolonged Exposure (CPE)

CPE consisted of 10 individual imaginal therapy sessions conducted according to the traditional PE protocol (Foa et al., 2018). Sessions were conducted 5 days a week for two weeks. The sessions were recorded, and patients were instructed to listen to re recordings at least once

per day at home. CPE also included the same homework format as typical PE (Foa et al., 2018). Therefore, although the TMT group was three weeks in length and CPE was two weeks, CPE requirement to repeat the exposure therapy in the afternoon resulted in equivalent treatment times for the two groups.

Intent-to-Treat Analyses and Strategy

All intent-to-treat analyses were conducted using R (R Core Team, 2023) and RStudio software (Posit Team, 2023). Sample characteristics by treatment group and time point are included in Table 1. Demographic and clinical variables were compared and there were no differences between groups. Missing data summaries for continuous and categorical variables were then generated using the *finalfit* package (Harrison et al., 2024). Continuous variables were summarized by mean, standard deviation, quartiles, and range, while categorical variables were summarized by missing count, percentage, and level distributions. All available data were included for randomized participants who completed baseline procedures, including those who later discontinued follow-up. Participants who were randomized but never started treatment did not complete baseline assessments (and therefore did not provide baseline actigraphy data) and could not be included in actigraphy-based analyses.

Missing data patterns for all sleep variables by time point and treatment group were visualized using the *naniar* package (Tierney & Cook, 2023), revealing <10% missing data at post-treatment, ~35% at 3-months follow up, and 50% at 6-months follow up. Our analysis was conducted under the Missing at Random (MAR) assumption. Missing data were then handled using multiple imputation with the *mice* package (Buuren & Groothuis-Oudshoorn, 2011), following guidelines by He (2010). The following variables were entered into the model: age, treatment arm (e.g., TMT or CPE), race, sex, trauma type (e.g., MST, combat, or other), all sleep outcomes (at each timepoint), and PCL-5 scores (excluding sleep questions; at each timepoint). We used predictive mean matching for continuous variables, logistic regression for binary variables, and proportional odds logistic regression for ordinal variables, generating 5 imputations with a maximum of 50 iterations each.

Both within-condition (Cohen's d) and between-condition (Hedges' g) effect sizes, along with 95% confidence intervals (CIs), were calculated to quantify the magnitude of changes within each condition across time (pre, post, 3-month, and 6-month follow-ups) and to evaluate differences between TMT and CPE. Consistent with recommendations for interpreting intervention effects (Cumming, 2014; Wasserstein & Lazar, 2016), we focused on effect sizes rather than p -values. Effect sizes meeting or exceeding 0.20 were reported, highlighting clinically meaningful findings, particularly relevant for sleep interventions where small effects can still signal valuable clinical benefits.

Results

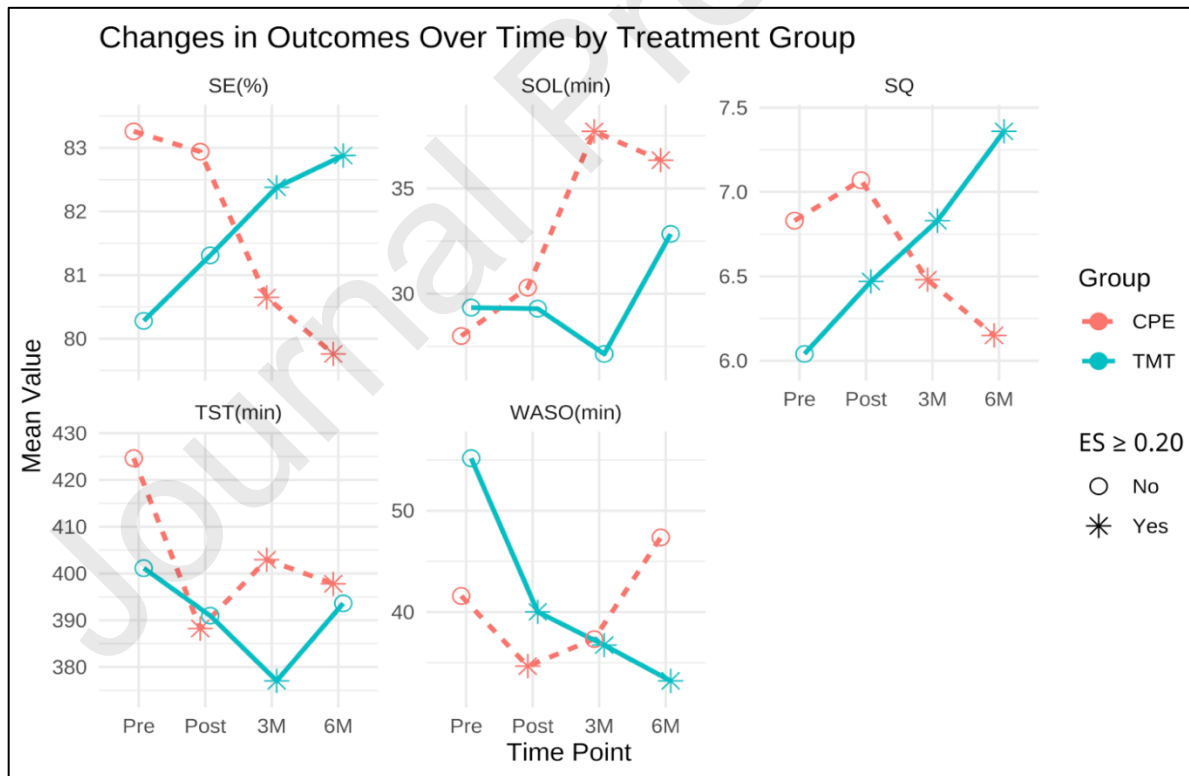
Within Group Changes in Sleep

Means and standard deviations by time point and treatment group are reported in Table 2. Within the TMT group, total sleep time (TST) did not change meaningfully at post-treatment ($d = 0.13$) or 6-months ($d = 0.11$) but did increase slightly at 3-months ($d = 0.30$). Although

changes in sleep efficiency (SE) were negligible from pre- to post-treatment ($d = -0.13$), we observed small increases at 3- ($d = -0.27$) and 6-months ($d = -0.36$). Similarly, examination of sleep onset latency (SOL) showed negligible changes at all time points ($d < 0.20$). In contrast, a small decrease in wake after sleep onset (WASO) was observed from pre- to post-treatment ($d = 0.48$), with a moderate decrease at 3- ($d = 0.65$) and 6-months ($d = 0.76$). Sleep quality (SQ) showed meaningful improvements at all time points ($d = -0.25$ at post-treatment, -0.43 at 3-months, -0.77 at 6-months). Within-group changes across time are reported in Table 3 and illustrated in Figure 1.

Within the CPE group, TST decreased meaningfully from pre- to post-treatment ($d = 0.42$), with small but meaningful reductions at the 3- ($d = 0.23$) and 6-month ($d = 0.30$) follow-ups. Similarly, SE remained relatively unchanged at post-treatment ($d = 0.05$) but showed small to medium reductions after both 3- ($d = 0.39$) and 6-months ($d = 0.54$). SOL did not change meaningfully from pre- to post-treatment ($d = -0.12$), but showed small, meaningful increases at the 3- ($d = -0.31$) and 6-month follow-ups ($d = -0.34$). WASO decreased at post-treatment ($d = 0.22$) but showed negligible changes afterwards (3-months: $d = 0.16$; 6-months: $d = -0.19$). Finally, SQ showed no meaningful change from pre- to post-treatment ($d = -0.14$) but demonstrated small to moderate decreases by the 3-month ($d = 0.20$) and 6-month ($d = 0.40$) follow-ups.

Figure 1. Changes in Outcomes Over Time by Treatment Group



Note. Within-group meaningful difference ($d \geq .20$) from the pre-timepoint score is marked with an asterisk.

Between Group Changes in Sleep

Comparison of changes in sleep between the groups (Table 4) indicated that TST remained similar between groups at posttreatment ($g = 0.04$) and 6-months ($g = -0.06$), though at 3-months, CPE participants slept longer than those in the TMT group ($g = -0.31$). CPE participants initially exhibited slightly higher SE ($g = -0.23$), but by 3-months, TMT participants showed a small improvement ($g = 0.24$), followed by a moderate improvement at 6-months ($g = 0.51$). SOL showed non-meaningful differences between groups at posttreatment ($g = -0.04$) or 6-months ($g = -0.12$), but at 3-months, TMT participants fell asleep more quickly than CPE participants ($g = -0.34$). Differences in WASO indicated that TMT participants initially experienced slightly more WASO than CPE participants ($g = 0.17$), but by 6-months, the TMT group had moderately lower WASO than the CPE group ($g = -0.52$), with a negligible difference at 3-months ($g = -0.03$). SQ was slightly higher in the CPE group than in the TMT group ($g = -0.35$) at post-treatment; however, by the 3-month follow-up, the group difference shifted in favor of TMT ($g = 0.19$), and by 6-months follow-up, the TMT group demonstrated comparative moderate improvement in SQ ($g = 0.70$).

Sleep and PTSD Symptom Correlations

Analytic Strategy

To examine the relationships between sleep changes and PTSD symptoms over time, we conducted correlation analyses (see Table 1 in supplementary material for all comparisons) for sleep and PTSD symptom scores at post-treatment, 3-months, and 6-months. For each sleep variable at each time point, we calculated the mean score and assessed its correlation with PTSD symptoms, as measured by PCL-5 scores, at the same time point. Our focus was on the direction and magnitude of correlations, emphasizing effect sizes of 0.10 or greater to interpret practical associations rather than statistical significance.

Correlations between Sleep Variables and PCL-5 Scores

TMT Group

In the TMT group, TST showed no association with PTSD symptoms at post-treatment ($r = -0.00$) but had a small negative correlation at 3-months ($r = -0.26$), which shifted to a small positive correlation at 6-months ($r = 0.21$), suggesting that longer sleep duration was linked to greater PTSD symptoms at this later time point. SE showed a small negative correlation across all time points, with a small negative correlation at post-treatment ($r = -0.15$), a slightly stronger association at 3-months ($r = -0.28$), and a small negative correlation at 6-months ($r = -0.13$), indicating that greater efficiency was modestly linked to lower PTSD symptoms. SOL exhibited a small positive correlation with PTSD symptoms at post-treatment ($r = 0.27$), indicating that taking longer to fall asleep was associated with more severe PTSD symptoms. However, this association became non-meaningful 3- ($r = -0.03$) and 6-months ($r = -0.09$) later. WASO showed a small positive correlation at post-treatment ($r = 0.10$), became non-meaningful at 3-months ($r = 0.02$), and increased slightly at 6-months ($r = 0.15$), suggesting that greater nighttime wakefulness was modestly associated with higher PTSD symptoms over time. Finally,

SQ was initially unrelated to PTSD symptoms at post-treatment ($r = 0.03$) but showed a small negative correlation at 3-months ($r = -0.08$) and a moderate negative correlation at 6-months ($r = -0.31$), suggesting that as sleep quality improved, PTSD symptoms decreased over time.

CPE Group

In the CPE group, correlations between sleep variables and PTSD symptoms were generally weak across time points (see Table 2 in supplementary material for all comparisons). TST demonstrated a small positive correlation with PTSD symptoms at post-treatment ($r = 0.18$) and 3-months ($r = 0.17$), which slightly decreased at 6-months ($r = 0.12$), suggesting that longer sleep duration was weakly linked to higher PTSD symptoms across time points. SE showed a non-meaningful correlation at post-treatment ($r = 0.06$) and 3-months ($r = 0.07$), and a small negative correlation at 6-months ($r = -0.16$), indicating a weak and somewhat inconsistent relationship over time. SOL remained largely unrelated to PTSD symptoms, with non-meaningful to small correlations at post-treatment ($r = 0.02$), 3-months ($r = -0.02$), and 6-months ($r = 0.12$). WASO showed no association with PTSD symptoms at post-treatment ($r = -0.05$) or 3-months ($r = -0.09$), but by 6-months, a small positive correlation emerged ($r = 0.17$), indicating greater nighttime wakefulness to be modestly associated with increased PTSD symptoms at the final follow-up. Finally, SQ showed no meaningful association with PTSD symptoms at post-treatment ($r = -0.01$) but demonstrated a small positive correlation at 3-months ($r = 0.11$), suggesting that higher sleep quality was slightly linked to increased PTSD symptoms. At 6-months however, this relationship reversed, with a small negative correlation ($r = -0.22$), indicating that poorer sleep quality was weakly associated with greater PTSD symptoms over time.

Discussion

Despite available pilot data and suggestions that integrated treatments targeting PTSD and sleep disturbance may produce optimal outcomes (Colvonen et al., 2019; Miller et al., 2020), treatment studies examining such protocols are generally lacking. The current study sought to begin to fill this gap by directly comparing an evidence-based PTSD intervention that also includes a focus on improving sleep (TMT) to an evidence-based PTSD intervention without a sleep component (CPE). We collected one-week of actigraphy data at four timepoints, including two follow-up assessments, to also examine potential changes in sleep over time.

Within the TMT group, participants showed continual, meaningful improvements from post treatment to 6-months follow up in three out of five objective sleep outcomes including increased SQ and SE and reduced WASO. Changes in SOL and TST within the TMT group were more variable. SOL showed minor decreases initially but worsened at the 6-month follow-up assessment, whereas TST decreased from pre-treatment through the 3-month follow-up before rebounding to near-baseline levels at the 6-month follow-up. In contrast, in the CPE group, all five sleep parameters assessed were worse at the 6-month follow up assessment compared to pre-treatment levels. In fact, small initial improvements in SQ and WASO at post-treatment were followed by a continual worsening of these sleep variables at the 3- and 6-month assessments compared to even baseline levels. CPE participants also showed a progressive decline in SE and prolonged SOL through the follow-up period. Similarly, TST declined and never recovered to baseline levels in the CPE group. Therefore, consistent with conclusions from prior studies,

PTSD-focused treatment alone does not appear to produce meaningful improvements in sleep either acutely or over time (Gutner et al., 2013; Larsen et al., 2019).

Between-condition comparisons further underscore the differential effects of these interventions on sleep. At post-treatment, CPE participants reported slightly better SQ and SE than TMT participants; however, this pattern reversed at both follow-up assessments based on moderate effects sizes. Notably, TST declined in both groups at post-treatment, but only the TMT group demonstrated a rebound to baseline levels after an initial decline. Differences in WASO and SOL similarly favored TMT by the end of the study. Importantly, as reported in Beidel et al. (2025), both treatments produced significant reductions in PTSD symptoms; however, only TMT, an integrated intervention targeting both PTSD and sleep, produced lasting improvements in a majority of objective sleep parameters. These findings converge with growing recognition that sleep disturbances are not merely secondary symptoms of PTSD but integral to the disorder's maintenance and treatment response (Spoormaker & Montgomery, 2008).

Whereas several prior studies among military populations have failed to document improvements in actigraphy-based sleep outcomes following treatment for PTSD (Haynes et al., 2020; Walters et al., 2020), we are aware of only one other study to examine changes in actigraphy-derived sleep variables following an integrated treatment for PTSD and sleep disturbance. In a smaller pilot study, Colvonen et al. (2019) found meaningful improvements in objective sleep patterns among veterans following combined PE and CBT-I treatment, including increases in SE and TST based on large effect sizes. The fact that we observed greater sleep consolidation (i.e., improvements in SE and WASO) but not longer sleep duration in our TMT sample at post-treatment and follow-up may indicate a need to more comprehensively address insomnia in PTSD patients using gold standard treatments (i.e., CBT-I).

Consistent with clinical practice guidelines, sleep hygiene alone is not recommended as a stand-alone treatment for insomnia, and thus large reductions in insomnia severity would not necessarily be expected from brief sleep hygiene education. However, the sleep component in TMT extends beyond sleep hygiene education and includes stimulus-control-consistent strategies, sleep self-monitoring with time-in-bed adjustments based on sleep efficiency, and relaxation skills, delivered within a structured, intensive program with daily therapist review and reinforcement. This treatment model is consistent with our findings, including stronger changes in consolidation indices (SE/WASO) than sleep duration. Indeed, this treatment model may be sufficient to improve sleep consolidation for some patients, but a subset may require a more comprehensive insomnia intervention (e.g., a full CBT-I protocol) or stepped-care augmentation when clinically significant insomnia persists. Notably, even at 6-month follow-up, mean SE remained below commonly used clinical benchmarks (e.g., <85%) in both groups (TMT = 82.88%; CPE = 79.76%), and mean SOL and WASO remained at or above ~30 minutes (SOL: TMT = 32.84 min; CPE = 36.34 min; WASO: TMT = 33.22 min; CPE = 47.36 min; Table 2), supporting the interpretation that additional or more intensive insomnia-focused intervention may be required to move a subset of participants into non-clinical ranges. Although we did not examine reports of insomnia symptoms in our study, subjective reports of insomnia severity decreased in the study by Colvonen and colleagues based on large effect sizes. Overall, these collective results support the use of integrated treatment protocols to comprehensively target the impairing constellation of symptoms present among those with PTSD.

Our findings also suggest that meaningful improvements in sleep are not necessarily linear during or following treatment and may require more time to become established among PTSD patients. For example, TMT participants did not exhibit meaningful improvements in SOL until the 6-month follow-up assessment. TST also decreased from pre-treatment through the 3-month follow-up before rebounding to near-baseline levels at the 6-month follow-up. The fact that challenging exposure sessions overlapped with sleep hygiene training from the outset of TMT may be relevant for understanding the course of these sleep changes. Comparatively, the integrated treatment used by Colvonen et al. (2019) completed six CBT-I sessions prior to beginning exposures (also in-vivo and imaginal), which might have contributed to large improvements in TST. Accordingly, there is need not only for research utilizing integrated treatments for PTSD and sleep disturbance, but for attention to the optimal sequencing and timing of specific intervention components.

Although we observed improvements in several objective sleep indices at follow-up, the practical relevance of these changes is more difficult to determine without patient-reported sleep outcomes (e.g., insomnia severity, overall sleep satisfaction, daytime sleepiness) and functional measures. Future studies should include both actigraphy and daily diaries to evaluate perceived sleep improvement and to benchmark change against established clinical thresholds (e.g., insomnia severity or patient-reported meaningful change), thereby facilitating comparison with CBT-I and other insomnia-focused interventions.

Correlation analyses further underscore relationships between sleep and PTSD symptom severity following treatment and over time. In both groups, PTSD symptoms show similar associations with sleep variables, including negative correlations with SE, SQ, and WASO, and positive correlations with TST and SOL. These findings align with existing research demonstrating disrupted sleep serves as both a consequence and a perpetuating factor of PTSD (Germain, 2013), and that persistent sleep disturbances following PTSD treatment predict poorer functioning and higher relapse rates (Belleville et al., 2011; Kartal et al., 2021).

The present study extends prior research in multiple ways, including examining the impact of two evidence-based PTSD interventions, including an integrated treatment targeting sleep, on actigraphy-based sleep outcomes both immediately after treatment and over time. Despite the strengths of this study, several limitations should be noted. First, while actigraphy provides valuable insights into day-to-day sleep patterns, it is not a direct measure of sleep and has been shown to overestimate TST and underestimate SOL and WASO (Conley et al., 2019) compared to polysomnography (PSG), the gold standard for sleep assessment. Likewise, comparing subjective sleep reports with actigraphy would have been informative for understanding whether objective sleep patterns align with perceptions of sleep. Relatedly, we did not collect subjective sleep outcomes (e.g., daily sleep diaries or standardized insomnia measures) alongside actigraphy data, which limits comparability with insomnia-focused intervention trials where self-report diaries are a core outcome and provide clinically interpretable indices of perceived sleep and insomnia severity. Absence of subjective sleep data also precludes evaluating whether observed actigraphy changes corresponded to participant perceived sleep improvement or daytime functioning. This will be an important focus for future studies.

Second, TMT includes a daily small-group milieu component (e.g., social reintegration, anger management, behavioral activation) that is not matched in CPE. Group-based treatment may confer nonspecific benefits (e.g., social support, accountability, and increased engagement) that could contribute to downstream outcomes, including sleep. However, the clearest between-group differences emerged on sleep parameters that were directly targeted in TMT's sleep modules, whereas CPE does not include sleep-focused skills; thus, we interpret the sleep-specific content as the most parsimonious explanation for the observed sleep effects while acknowledging that nonspecific group effects cannot be fully disentangled in the present design. Future trials could incorporate attention-matched controls or dismantling designs to isolate the unique contribution of sleep-focused components versus group milieu. Additionally, some exclusion criteria (e.g., severe comorbid substance use disorders and moderate-to-severe traumatic brain injury) may limit the generalizability of findings to broader military populations. We did not control for the use of various medications in our study which could have influenced both sleep and PTSD outcomes. Similarly, we did not assess for obstructive sleep apnea; however, no participant reported sleep disordered breathing as a concern. Lastly, common to most PTSD treatment studies, attrition rates at the 6-month follow-up were substantial, potentially impacting the robustness of long-term findings.

In summary, TMT, which includes a sleep hygiene training component, produced superior objective sleep outcomes compared to treatment with PE alone. Findings align with limited prior work demonstrating integrating interventions for PTSD and sleep disturbances result in more meaningful improvements in sleep than PTSD-focused treatment alone (Taylor et al., 2023; Walters et al., 2020). However, changes in sleep patterns are not necessarily linear and detectable improvements in sleep may require time to become established after treatment, necessitating more follow up studies. Other pivotal questions raised by these data relate to adequate 'dosing' of sleep interventions for optimal sleep outcomes as well as the ideal sequencing, timing and focus of sleep intervention components.

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Conflict of Interest Statement

No potential competing interest was reported by the authors.

Table 1*Demographics*

	CPE (<i>n</i> = 47)	TMT (<i>n</i> = 35)	Overall (<i>n</i> = 82)
<i>Age</i>			
Mean (SD)	33.3 (7.44)	35.0 (9.58)	34.0 (8.40)
Median [Min, Max]	36.0 [20.0, 44.0]	38.0 [20.0, 52.0]	36.5 [20.0, 52.0]
<i>Sex</i>			
Female	18 (38.3%)	11 (31.4%)	29 (35.4%)
Male	29 (61.7%)	24 (68.6%)	53 (64.6%)
<i>Race</i>			
White	19 (40.4%)	14 (40.0%)	33 (40.2%)
Black or African American	15 (31.9%)	9 (25.7%)	24 (29.3%)

Table 1*Demographics*

	CPE (<i>n</i> = 47)	TMT (<i>n</i> = 35)	Overall (<i>n</i> = 82)
Hispanic or Latino	5 (10.6%)	8 (22.9%)	13 (15.9%)
Asian	3 (6.4%)	2 (5.7%)	5 (6.1%)
American Indian or Alaska Native	2 (4.3%)	1 (2.9%)	3 (3.7%)
Native Hawaiian or Other Pacific Islander	0 (0%)	0 (0%)	0 (0%)
Other	3 (6.4%)	1 (2.9%)	4 (4.9%)
Trauma Type			
Combat	25 (53.2%)	23 (65.7%)	48 (58.5%)
MST	13 (27.7%)	10 (28.6%)	23 (28.0%)
Other	9 (19.1%)	2 (5.7%)	11 (13.4%)

Note. TMT = Trauma Management Therapy; CPE = Compressed Prolonged Exposure; MST = Military Sexual Trauma

Table 2

Means and Standard Deviations by Time Point and Group

Outcome	Pre				Post				3M				6M			
	TMT		CPE		TMT		CPE		TMT		CPE		TMT		CPE	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
SQ	6.04	1.74	6.83	1.69	6.47	1.70	7.07	1.69	6.83	1.93	6.48	1.79	7.36	1.71	6.15	1.72
SE(%)	80.28	8.30	83.26	6.52	81.31	7.57	82.94	6.53	82.38	7.33	80.65	6.88	82.88	5.73	79.76	6.38
TST(min)	401.14	72.47	424.65	104.60	390.98	80.73	388.24	59.54	377.05	87.63	402.95	77.80	393.64	67.11	397.80	69.10
SOL(min)	29.34	23.49	27.99	19.24	29.29	26.40	30.29	20.12	27.14	16.19	37.71	39.66	32.84	28.43	36.34	29.06

Table 2*Means and Standard Deviations by Time Point and Group*

Outcome	Pre				Post				3M				6M			
	TMT		CPE		TMT		CPE		TMT		CPE		TMT		CPE	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
WASO(min)	55.17	32.56	41.59	31.53	40.02	30.85	34.67	30.74	36.74	22.77	37.33	21.43	33.22	25.05	47.36	28.16

Note: This table displays the means and standard deviations for TMT and CPE groups at each time point; TMT = Trauma Management Therapy; CPE = Compressed Prolonged Exposure; 3M = 3-month follow-up; 6M = 6-month follow-up; SQ = sleep quality; SE = sleep efficiency; TST = total sleep time; SOL = sleep onset latency; WASO = wake after sleep onset

Table 3*Within-Condition Effect Sizes (Cohen's d) and Confidence Intervals*

Outcome	TMT									CPE								
	Pre-Post			Pre-3M			Pre-6M			Pre-Post			Pre-3M			Pre-6M		
	<i>d</i>	LL	UL	<i>d</i>	LL	UL	<i>d</i>	LL	UL	<i>d</i>	LL	UL	<i>d</i>	LL	UL	<i>d</i>	LL	UL
SQ	-0.25	-0.56	0.06	-0.43	-0.78	-0.08	-0.77	-1.24	-0.29	-0.14	-0.48	0.19	0.20	-0.13	0.54	0.40	-0.03	0.83
SE(%)	-0.13	-0.43	0.17	-0.27	-0.67	0.13	-0.36	-0.79	0.06	0.05	-0.28	0.37	0.39	-0.01	0.79	0.54	0.12	0.96
TST(min)	0.13	-0.21	0.48	0.30	-0.08	0.68	0.11	-0.27	0.49	0.42	0.01	0.84	0.23	-0.18	0.65	0.30	-0.13	0.73
SOL(min)	0.00	-0.27	0.27	0.11	-0.37	0.58	-0.13	-0.63	0.36	-0.12	-0.39	0.16	-0.31	-0.74	0.12	-0.34	-0.80	0.12
WASO(min)	0.48	0.01	0.94	0.65	0.21	1.09	0.76	0.23	1.29	0.22	-0.15	0.59	0.16	-0.26	0.58	-0.19	-0.60	0.22

Table 3*Within-Condition Effect Sizes (Cohen's d) and Confidence Intervals*

Outcome	TMT									CPE									
	Pre-Post			Pre-3M			Pre-6M			Pre-Post			Pre-3M			Pre-6M			
	d	LL	UL	d	LL	UL	d	LL	UL	d	LL	UL	d	LL	UL	d	LL	UL	

Note: Effect sizes equal to or greater than .20 are highlighted in bold; TMT = Trauma Management Therapy; CPE = Compressed Prolonged Exposure; 3M = 3-month follow-up; 6M = 6-month follow-up; SQ = sleep quality; SE = sleep efficiency; TST = total sleep time; SOL = sleep onset latency; WASO = wake after sleep onset;

Table 4

Between-Condition Effect Sizes (Hedges' g) and Confidence Intervals for TMT and CPE

Outcome	Post-Treatment			3-Month			6-Month		
	<i>g</i>	LL	UL	<i>g</i>	LL	UL	<i>g</i>	LL	UL
SQ	-0.35	-0.79	0.09	0.19	-0.25	0.63	0.70	0.25	1.15
SE(%)	-0.23	-0.66	0.21	0.24	-0.20	0.68	0.51	0.07	0.95
TST(min)	0.04	-0.40	0.47	-0.31	-0.75	0.13	-0.06	-0.50	0.38
SOL(min)	-0.04	-0.48	0.39	-0.34	-0.78	0.10	-0.12	-0.56	0.32
WASO(min)	0.17	-0.26	0.61	-0.03	-0.46	0.41	-0.52	-0.97	-0.08

Note: Effect sizes equal to or greater than .20 are highlighted in bold; SQ = sleep quality; SE = sleep efficiency; TST = total sleep time; SOL = sleep onset latency; WASO = wake after sleep onset. Negative effect size indicate larger values for the CPE group.

Figure 2

CONSORT Flow Diagram

