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Examining Sleep Over Time in a Randomized Control Trial Comparing Two Integrated PTSD and Alcohol Use Disorder Treatments

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Abstract

Study Objectives: Insomnia is highly co-occurring with both posttraumatic stress disorder (PTSD) and alcohol use disorder (AUD). This is concerning since insomnia contributes to worse substance abuse and PTSD, and a host of negative health consequences. No study has tracked how sleep indices and insomnia change related to integrated PTSD and AUD treatment using evidence-based exposure therapy. This study examined how insomnia changes over time in a randomized control trial of two integrated PTSD and AUD treatments.

Methods: Participants were 119 adult veterans (90% male) seeking treatment for AUD and PTSD at a large urban VA. Participants were randomized to either COPE (integrated treatment using prolonged exposure) or Seeking Safety (integrated therapy using cognitive behavioral, interpersonal techniques and case management). Assessments were done at pre- and post-treatment and include: Clinician Administered PTSD Scale, Timeline Follow-back calendar-assisted interview for AU, insomnia severity index (ISI), sleep diary and actigraphy for 7 days.

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Results: ISI showed significant decreases, but a majority remained above the clinical cutoff at post-treatment. Wake after sleep onset decreased, but only by 8 minutes, remaining above clinical thresholds. Decreases in PTSD, but not in heavy drinking, predicted change in ISI. No significant changes were observed in other sleep variables measured.

Conclusions: Findings suggested some statistical improvements in sleep quality, but sleep indices remained above clinical cut-offs. This study provides evidence that insomnia is an independent disorder and not responsive to PTSD or AUD treatments alone. Sleep symptoms should be assessed and treated in patients with comorbid mental health conditions.

Keywords

Insomnia; PTSD; Substance Use; Treatment

1. Introduction

Posttraumatic stress disorder (PTSD) frequently co-occurs with alcohol use disorders (AUDs). Further, insomnia is highly co-occurring with both PTSD and AUD individually (Jenkins et al., 2015) and together (Colvonen et al., 2018a; McHugh et al., 2014). This is concerning given that insomnia contributes to worse PTSD symptoms (Inman et al., 1990), major depression, substance abuse, impaired daytime functioning (Nadorff et al., 2011), higher rates of relapse (Brower et al., 1998; Smith et al., 2014), negative long-term health consequences (Baran et al., 2012), and suicide risk (Nadorff et al., 2011). Examining insomnia in the context of PTSD and AUD treatment can offer insight into AUD and PTSD symptom severity, AUD and PTSD treatment trajectories, and inform recommendations for treatment planning. This study aims to examine how insomnia changes over time within a randomized control trial (RCT) of two integrated PTSD and AUD treatments.

Application of DSM-5 criteria for insomnia suggests a range of 35 – 61% in individuals with PTSD (Jenkins et al., 2015; Krakow et al., 2002; Ohayon and Shapiro, 2000; Plumb et al., 2014). Despite PTSD having two sleep symptoms in the diagnostic criteria, insomnia is best considered an independent disorder (Colvonen et al., 2018b; Germain et al., 2017). Insomnia is related to the development and maintenance of PTSD (Germain et al., 2008) and is associated with worse PTSD symptoms (Edens et al., 2011; Germain et al., 2008). Individuals with PTSD show more trouble falling asleep, staying asleep, excessive daytime sleepiness and early morning awakenings (Belleville et al., 2009; Neylan et al., 1998; Waldrop et al., 2008) and more abnormal sleep architecture (Pressman and Orr, 1997) than individuals without PTSD. Individuals with PTSD also show greater night-to-night variability in their sleep than patients without PTSD (Straus et al., 2015). Further, insomnia symptoms, as an independent disorder, typically do not fully remit following effective PTSD treatment (Colvonen et al., 2018b).

In a review of PTSD treatments on insomnia, Colvonen et al. (2018b) suggest that while insomnia tends to improve over the course of PTSD treatment, it still remains disturbed. For example, six studies examining evidence-based PTSD treatments on insomnia found insomnia did show small decreases over the course of treatment; however, symptoms still remained above the clinical cut-off (Belleville et al., 2011; Galovski et al., 2009; Gutner

et al., 2013; Pruiksma et al., 2016; Schnurr and Lunney, 2019; Walters et al., 2019). Further, some of the sleep improvements seen during treatment tended to deteriorate by the six-month post-treatment assessment (Belleville et al., 2011).

Insomnia also co-occurs in 36–72% of patients with an AUD (Administration, 2014; Brower and Perron, 2010). Self-medication of sleep problems with alcohol is a leading hypothesis regarding why AUD and sleep problems frequently co-occur (Brower et al., 2001). While alcohol reduces sleep onset early in a use cycle, it causes awakenings overnight. Additionally, over a number of days of consistent alcohol use near bedtime, the initial sleep onset effects tend to diminish, but the sleep disruption later in the night persists (Roehrs and Roth, 2001). Further, insomnia severity is not only related to increased drinking (Brower et al., 2001; Roehrs and Roth, 2001), but also AUD severity (Brower and Perron, 2010).

Insomnia symptoms decrease with decreases in alcohol use but may last weeks, months, or even years after the initiation of abstinence from alcohol (Administration, 2014; Brower, 2003; Currie et al., 2003; Williams and Rundell, 1981). For example, Drummond and colleagues (Drummond et al., 1998) report that in a follow-up study of alcohol-abstinent participants, abnormal patterns of sleep were present after 27 months of complete abstinence. In addition to insomnia occurring as a side effect of withdrawal, there is evidence that insomnia is a risk factor for relapse (Brower, 2003; Brower and Perron, 2010; Conroy and Arnedt, 2014). For example, two studies found that increased sleep latency within the first few weeks of inpatient admission increased the odds of relapse to alcohol use at 5-month follow-up (Brower et al., 2001) and at 14-month follow-up (Drummond et al., 1998). As such, residual sleep problems following AUD treatment are a clinically relevant factor for future relapse.

While individuals with both PTSD and AUD are more likely to have insomnia than individuals with only one disorder (Lande, 2012; Saladin et al., 1995), only two studies have examined sleep, over time, for participants that have both PTSD and AUD. Colvonen et al. (2018a) examined 40 veterans with comorbid PTSD and substance use disorder (SUD) in a 28-day Substance Abuse Residential Rehabilitation Treatment Program PTSD track. Importantly, participants were not offered prolonged exposure (PE) therapy or cognitive processing therapy (CPT) during the study. Using independent measures of PTSD and insomnia, the study showed that PTSD symptoms improved over time but that insomnia symptoms did not. These findings are consistent with many of the findings that suggest insomnia does not decrease even when PTSD symptoms do and remains elevated following abstinence. On the other hand, McHugh et al. (2014) found that insomnia significantly decreased during integrated coping skills treatment for PTSD and SUD, though these researchers used a single item for insomnia taken from the Clinician Administered PTSD Scale (CAPS) and did not include an independent measure of sleep. This, unfortunately, anchors the single sleep item to changes in PTSD and limits the validity of their findings.

As evidence builds that individuals with AUD can tolerate and benefit from evidence-based PTSD treatment (Roberts et al., 2015), integrated and concurrent treatment for PTSD and AUD is becoming more widely available. Our study is a secondary analysis of a parent

RCT (Norman et al., 2019) comparing Concurrent Treatment for PTSD and Substance Use Disorder Using Prolonged Exposure (COPE; (Back et al., 2014) versus integrated coping skills therapy, Seeking Safety (SS; (Najavits, 2002). Overall, COPE showed greater reduction in PTSD symptoms than SS and comparable drinking decreases. However, the effect of PTSD/AUD treatment on insomnia remains unclear. Here, we report findings from the first study to examine changes in sleep related to a PTSD and AUD treatment using integrated PE.

Aim 1 examined indices of sleep over time for all participants. We hypothesized that there would be small to moderate improvements in sleep quality and quantity from pre- to post-treatment, but that overall severity would remain within the clinical range. Aim 2 examined how changes in heavy drinking and PTSD symptoms affected changes in sleep. We hypothesized both changes in percent days heavy drinking and PTSD symptoms would predict small changes in sleep variables at post-treatment. Exploratory Aim 3 examined whether insomnia symptoms would change as a function of SS versus COPE treatments.

2. Methods

2.1 Participants

Demographic characteristics are in Table 1. Participants were 119 adult veterans (107 male) seeking treatment at a large urban VA. Patients who potentially had PTSD/AUD based on chart review were referred to the study by mental health providers. Patients also responded to flyers posted around the VA. Eligible participants: (a) had current full or subthreshold PTSD (up to one symptom missing; (Franklin et al., 2018) and current AUD with at least 20 days of heavy alcohol use in the last 90 days not in a restricted environment and (b) wanted to reduce or abstain from alcohol use. Exclusion criteria were: (a) acute suicidality, (b) unmanaged psychosis or mania, and (c) intravenous drug use. Participants were asked not to engage in other PTSD psychotherapy during study treatment. Participation in other mental health treatment (medications and psychotherapy) was tracked.

2.2 Procedures

The study design was approved by the local IRB and registered with ClinicalTrials.gov (NCT01601067). The study was a part of an randomized control trial (RCT) of two active treatments, to compare integrated prolonged exposure therapy, using the Concurrent Treatment for PTSD and Substance Use Disorder Using Prolonged Exposure (COPE; (Back et al., 2014) protocol, with integrated coping skills therapy, Seeking Safety (SS; (Najavits et al., 2006). See Norman et al. (2019) for full methods. Participants gave written informed consent prior to enrollment by the study coordinator. Independent evaluators were blind to treatment assignment for study duration.

Recruitment ran February 2013 to May 2017. Following a phone screen, participants were scheduled to provide informed consent and complete baseline assessments. Participants who met inclusion criteria then met with a study clinician to learn more about both therapies and ask any remaining questions about the treatment process. Balanced block randomization (variable blocks of 8–12) with masked allocation was stratified by gender. Participants were

informed of their treatment condition at their first therapy session. Both therapies were 12 sessions, with the option to complete up to 16 sessions if the participant's therapy goals were not met by 12 sessions. Following, therapy participants completed measures post-treatment, and at 3- and 6-month post-treatment follow-up. Sleep measures were only given at pre- and post-treatment. Compensation was $20 baseline, $30 post-treatment, and $50 per follow-up.

Blinded independent evaluators completed training and achieved at least 90% agreement on clinician administered CAPS-5 item scores prior to conducting assessments. Interrater reliability, conducted on 11% of randomly selected CAPS-5 assessments, was excellent (κ = 0.94 for diagnosis and intraclass correlation coefficient of 0.99; 95% CI 0.98–0.99; (McHugh, 2012; Weathers et al., 2017). All therapy sessions were recorded, and 10% were rated for adherence.

2.3 Measures

2.3.1 PTSD: Clinician Administered PTSD Scale for DSM-5 (CAPS-5; range 0–80), a 30-item structured interview (Weathers et al., 2017) considered the gold standard for PTSD diagnostic assessment, was the primary measure of PTSD severity and diagnosis. Diagnosis was determined using the SEV2 rule, which follows DSM-5 PTSD criteria, counting symptoms rated ≥2 as positive. CAPS-5 diagnosis using SEV2 displayed strong interrater reliability (κ = 0.78), and severity scores showed strong internal consistency (α = 0.88) in the development sample (Weathers et al., 2017). Internal consistency in the current sample was strong (α = 0.83).

2.3.2 Alcohol Use Severity: Timeline Follow-back (TLFB), a calendar-assisted structured clinical interview (Sobell and Timeline follow-back, 1992), was used to assess frequency and quantity of alcohol use. The TLFB shows good psychometric properties (Sobell et al., 1996)., Percentage of heavy drinking days (PHDD) was calculated by dividing the number of days in which 5+ drinks for males or 4+ drinks for females were consumed by the total number of days in the reference period. Toxicology screens were completed during a randomly selected week each month, and breathalyzer tests were administered if there was indication a participant came to an appointment under the influence.

2.3.3 Medications: Modified Interview of Antiretroviral Medication Use (AIAM; (Paterson et al., 2000) was used to assess psychotropic medication use. Categories of medication types were created including: antidepressants, antipsychotics, mood stabilizers, AUD/SUD meds, sedatives, antiadrenergic, psychotropic medication, sleep medications (prazosin, trazodone, zolpidem, hydroxyzine, melatonin), and other drugs.

2.3.4 Insomnia: Insomnia Severity Index (ISI; (Morin and Barlow, 1993) was used to measure insomnia with well-established reliability and validity. The ISI consists of 7 items, three of which assess severity of insomnia (i.e., degree of difficulty falling asleep, staying asleep, and waking too early). The remaining items query satisfaction with sleep pattern, effect of sleep on daytime and social functioning, and concern about current sleep difficulties. Scores range from 0–28; scores of 0–7 are considered "no clinical significance

insomnia", 8–14 are subthreshold, 15–21 are moderate, and 22–28 are considered severe clinical insomnia. Clinical cutoff of 11 or greater was shown to have strong sensitivity and specificity in clinical populations (Morin et al., 2011). ISI showed strong internal consistency (α = .77).

2.3.5 Sleep diaries: Patients completed a daily sleep diary for 7 days at baseline and post-treatment. The sleep diary included items, such as total time spent in bed, subjective sleep latency (sSL; i.e., how long it took to fall asleep the first time), and number and duration of awakenings, as well as three calculated variables: subjective total sleep time (sTST), subjective wake after sleep onset (sWASO; i.e., amount of time spent awake during the night after falling asleep initially), and sleep efficiency (sSE; i.e., total sleep time divided by total time in bed).

2.3.6 Actigraphy: Respironics Actiwatch 2 and Actiware software (Respironics) were used to collect and analyze actigraphy data. Watches were worn for 7 days at baseline and post-treatment. A single scorer, not blind to participants' condition status, manually set rest intervals corresponding to participants' time in bed each night. Participants' diary-reported bed times and wake times were used as guidelines, though rest intervals could be extended up to 60 min on either side to account for obvious sleep outside self-reported time in bed. This procedure was similar to methods used in other sleep studies (e.g., (Straus et al., 2015)). Sleep detection settings were set to medium. Automatic algorithms were then used to generate values for objective time in bed (oTIB), objective total sleep time (oTST), objective wake after sleep onset (oWASO), and objective sleep efficiency (oSE).

2.3.7 Night-to-night variability.—The root mean squared successive difference (RMSSD) served as the index of night-to-night variability due to its ability to detect changes from one night to the next. RMSSD has shown to differentiate between PTSD groups and insomnia only groups (Straus et al., 2015). RMSSD values were obtained for sTST, sSL, sWASO, and sSE, as well as oTST, oWASO, and oSE.

2.4 Treatments

COPE and SS were delivered in 90-minute individual sessions. Therapy was 12-sessions, with the option of completing up to 16-sessions if the participant and therapist agreed treatment goals were not yet met. Participants were encouraged to attend therapy 1–2 times per week on consecutive weeks, but allowed up to 6 months to finish treatment.

2.4.1 COPE: COPE (Back et al., 2014) is an integrated PTSD/AUD treatment that augments PE with cognitive behavioral relapse prevention skills for AUD in each session. COPE includes in-vivo exposures to trauma reminders (starting in session 3) and repeated imaginal exposures to the trauma memory (starting in session 4). The COPE manual includes 12 sessions. For participants who completed 13–16 sessions, up to four AUD skills were repeated.

2.4.2 SS: SS (Najavits, 2002) is a present-focused, PTSD/AUD integrated therapy that teaches cognitive behavioral and interpersonal techniques and case management. SS consists

of 24 modules. Each module includes safe coping skills. Trauma is discussed in the context of how it is affecting the patient's life in the present. For this study, session topics were predetermined for sessions 1 through 12 based on previous research. Participants completing 13–16 sessions selected from the remaining topics.

2.5 Analyses

Data were analyzed using descriptive statistics, regressions, and paired sample t-tests. Effect sizes were calculated using G*Power (Faul et al., 2007) for the parent paper. Aim 1 used paired t-test to examine how sleep variables changed from pre- to post-treatment. Aim 2 analyses created change scores for PTSD and heavy drinking from baseline to post-treatment. We ran separate regressions on change in heavy drinking and change in PTSD on sleep variables at post-treatment (e.g., ISI), controlling for baseline sleep variable scores, gender, number of days abstinent at baseline, AUD medications, and the sleep medication prazosin. AUD medications and prazosin were added because they correlated with change in PTSD and AUD outcome variables and may influence changes in sleep variables.

RMSSD was used to examine night-to-night variability in sleep indices. The RMSSD uses the following formula, where subscript I refers to the temporal order of each night:

$$RMSSD = \sqrt{\frac{\sum_{i=1}^{n-1} (x_{i+1} - x_i)^2}{n-1}}$$

3. Results

3.1 Attrition

Attrition rates from pre- to post-treatment were within the expected range for a PTSD and AUD study (33%). Individuals who did not fill out post-treatment PTSD and AUD questionnaires had more heavy drinking days (M = 62.20%, SD = 23.86) at baseline and less TST (M = 363 minutes, SD = 93.60) as indicated by the diary compared to individuals who filled out baseline and post assessments (M = 45.09%, SD = 25.29; M = 403 minutes, SD = 70.72). None of the other sleep variables or PTSD symptoms at baseline were different between groups.

The rates for the attrition on the actigraphy watch and sleep diaries were higher at 51% and 46% respectively. Participants who did not fill out post-treatment diaries had more heavy drinking days (M = 57.90%, SD = 25.95), lower actigraphy TST (M = 353 minutes, SD = 79.36), and lower actigraphy SE (M = 76.16%, SD = 8.44) at baseline compared to individuals with both baseline and post-treatment diaries (M = 41.89%, SD = 22.53; M = 413 minutes, SD = 53.54; M = 80.65%; SD = 7.70). Participants who did not use the post-treatment actigraphy had more heavy drinking days (M = 56.59%, SD = 26.57) and lower diary SE (M = 79.01%, SD = 12.93) at baseline compared to individuals with both baseline and post-treatment actigraphy (M = 45.62%, SD = 24.88; M = 84.03%, SD = 8.70).

3.2 Changes in Sleep Indices Over Time

We examined subjective and objective measures of sleep before and after treatment (See Table 2 for means, statistics, and effect sizes). 83.9% of participants met criteria for ISI cut-offs at pre-treatment and 64.6% met criteria at post-treatment. Insomnia severity showed statistically significant decreases over time, but a majority of a participants still remained above the clinical cutoff at post-treatment (ISI >= 11). Actigraphy-derived oWASO significantly decreased by 6 minutes with the change sWASO being non-significant (8 minute decrease), both with small effect sizes (r = .30, and .36 respectively). Both WASO times remained above the clinical indicated scores of 30 minutes at about 42 minutes following treatment. Sleep diary nap length increased 10 minutes from baseline to post-treatment. The night-to-night variability of oTST significantly decreased, suggesting more consistent oTST following treatment. The sleep diary also showed significantly less night-to-night variability in sWASO post treatment. Means and night-to-night variability of all the other sleep indices did not significantly change from pre to post-treatment.

3.3 Changes in Alcohol Use and PTSD Symptoms Impacting Changes in Sleep

We examined how changes in heavy drinking and PTSD symptoms affected changes in sleep from baseline to post-treatment. Gender, use of alcohol reduction medications, use of prazosin, and number of days abstinent at baseline were added as covariates into the models. Change in CAPS scores predicted post-treatment ISI scores, (β = −.39, $t(77)$ = −5.04, p < .001). For approximately every 6-point decrease in PTSD symptoms over the course of treatment, insomnia severity decreased by 1 point on the ISI. Change in percent days heavy drinking did not predict post-treatment ISI (β = .04, $t(77)$ = 0.45, p =.65).

Neither change in PTSD symptoms nor change in percent days heavy drinking predicted changes in any other actigraphy- or sleep diary-derived sleep indices.

3.4 Changes in Sleep Indices by Treatment

In exploratory analyses, we examined whether insomnia and quality/quantity of sleep changed as a function of SS versus COPE treatments (see Table 3). There were no differences between treatment condition on any diary or actigraphy sleep variables. Effect sizes showed moderate positive effects for Diary SL, WASO, TST and actigraphy SE in the COPE conditions when compared to SS.

4. Discussion

This study examined the effects of integrated PTSD and AUD treatment on both subjective and objective sleep parameters in veterans. This is the first study to examine changes in sleep in integrated trauma exposure and AUD treatment. Findings suggested some statistically significant improvements in sleep quality and insomnia symptoms, but sleep indices remained above clinical cut-offs. Specifically, self-reported insomnia symptoms showed statistical decreases, but decreased less than 4 points and 64.6% of the participants still met insomnia disorder criteria at post-treatment. Actigraphy-derived WASO significantly decreased by 6 minutes (from 48 minutes to 42 minutes) but remained above the clinically indicated cut-off score of 30 minutes. Sleep diary nap length increased from

16 minutes to 27 minutes; however, the clinical utility of this finding is limited. Finally, objective night-to-night variability in TST was improved with treatment suggesting more consistent TST following treatment.

We examined how change in PTSD symptoms and heavy drinking affected change in sleep parameters. We found that larger reductions in PTSD symptoms, but not percent days heavy drinking, were associated with larger decreases in insomnia symptoms. It should be noted that the ISI only had small changes over time and a majority of participants were still above the clinical cut-off; interestingly, no other sleep indices changed as a function of PTSD symptom change. These results are consistent with studies examining the effect of PTSD treatment on sleep (Colvonen et al., 2018b). Several studies suggest treating PTSD improves sleep, but does not bring insomnia symptoms below clinically significant thresholds (Belleville et al., 2011; Galovski et al., 2009; Gutner et al., 2013; Pruiksma et al., 2016; Schnurr and Lunney, 2019). One notable difference between the current study and previous research is the focus on patients with comorbid AUD and PTSD. Effect sizes observed in the current study are generally much smaller than previous research in non-comorbid samples, even though insomnia symptoms were less severe at baseline. For example, Walters et al., 2019 found similar effects on the ISI (.52), but showed larger effects for increases in SE, SL, and WASO. This suggests that the presence of AUD may make sleep symptoms *less* responsive to PTSD and AUD treatments.

There are several possible explanations for these findings. First, it is possible that there may be discrepant patterns of self-reported insomnia symptoms (e.g., ISI) and physically measured sleep (e.g., actigraph). It has been suggested that patients with PTSD may over-estimate the magnitude of sleep disturbance, which has been described as ‘paradoxical insomnia’ or ‘sleep state misperception’ (Ghadami et al., 2015; Hurwitz et al., 1998). That is, PTSD may be associated with a negative cognitive bias towards the self-perception of sleep, rather than a true difference. Thus, as PTSD improved, the perception of insomnia as indicated by the ISI decreased, while the objective measures of sleep stayed static. However, the evidence against this view are the high levels of SL (39 minutes) and WASO (41.61 minutes) seen at post-treatment, suggesting clinically low levels of sleep quality even at post-treatment.

Our study also examined changes in sleep indices as a function of COPE compared to SS. While changes in sleep were not significantly different by treatment condition, the effect sizes were larger for COPE than SS. This is consistent with our findings that changes in PTSD may account for more variance than changes in AU, as COPE showed significantly lower PTSD symptoms post-treatment. However, these findings should be interpreted with caution due to non-significant findings, and possible differences in number of medications taken by treatment condition. Additionally, sleep was not a primary outcome in the larger trial, so the current analysis may have been underpowered to find these effects.

Extant literature shows that insomnia symptoms decrease with decreases in AU but may still last weeks, months, or even years after the initiation of abstinence of alcohol (Administration, 2014; Brower, 2003; Currie et al., 2003; Williams and Rundell, 1981). In the current study, change in percent days heavy drinking did not predict change in

insomnia. This may be due to the evidence, similar to PTSD, that insomnia becomes a fully independent disorder as a result of AUD's effects on sleep, and thus, is non-responsive to AUD treatments. This effect is probably particularly strong in co-occurring PTSD and AUD due to the additive effects of two sleep-disrupting disorders. A final possibility is that most of the effects of decreasing percent days heavy drinking on insomnia happened before randomization. In our analyses, we had to control for number of days abstinent at baseline since it was positively associated with better baseline sleep indices.

The current study adds to the literature suggesting that insomnia is an independent disorder and should be treated as such (Colvonen et al., 2018b; Germain et al., 2017). Our study showed combined PTSD/AUD treatment did not much improve sleep, which is especially concerning given the high rates of sleep disturbance in PTSD (Maher et al., 2006; Neylan et al., 1998; Seelig et al., 2010), AUD (Administration, 2014; Brower and Perron, 2010), and in comorbid groups (Lande, 2012; Saladin et al., 1995). Though our treatments reduced both PTSD symptoms and alcohol use, the high rate of residual insomnia is especially problematic because sleep disturbance is thought to contribute to daytime PTSD symptoms (Edens et al., 2011; Germain et al., 2008), and is a risk factor for alcohol use relapse (Brower, 2003; Brower and Perron, 2010; Conroy and Arnedt, 2014; Drummond et al., 1998). That is, the behavioral and cognitive responses to acute insomnia from alcohol use lead to perpetuating factors (e.g., napping, sleeping pills) and conditioned arousal that keep insomnia static (Colvonen et al., 2018b). As such, clinicians should carefully evaluate sleep symptoms in patients with comorbid PTSD and AUD and consider adjunctive sleep treatment, such as Cognitive Behavioral Therapy for Insomnia (CBT-I), as indicated. While adding CBT-I has been shown effective as an adjunctive treatment with PTSD (Colvonen et al., 2019) it is unclear the ordering of treatments when dealing clinically with PTSD and AUD together. There is evidence that CBT-I, alone, is not enough to decrease relapse rates (Arnedt et al., 2011), and may need to be combined with AUD treatment.

The current study has some limitations worth noting. First, we only recruited veterans for this study, so findings may not generalize to civilian groups. Second, though the use of both subjective and objective sleep measures is a strength of the current study, the parent paper did not power for examining sleep in their study, reducing statistical power to detect effects. Third, the study was generally permissive in its inclusion criteria, which increased generalizability but limited our ability to control for other factors that might have influenced sleep-related outcomes. Fourth, this study did not screen for untreated obstructive sleep apnea (OSA), which may account for minimal changes in sleep variables. OSA is highly comorbid in PTSD and AUD, and may interfere with PE (Colvonen et al., 2018b; Mesa et al., 2017; Reist et al., 2017). Recent literature suggests the classic predictors of OSA (e.g., body mass index, age, smoking status) are not reliable predictors of OSA in younger veterans with PTSD (Colvonen et al., 2015). As such, future studies for PTSD and AUD should screen for OSA using overnight objective screening, even in younger, healthier veterans with PTSD.

Another limitation was high attrition rates in post-treatment PTSD and SUD (33%), actigraphy (51%) and sleep diary (46%) data that may affect interpretation of the findings. Generally, individuals that only filled out baseline sleep data had more heavy drinking

days, less TST, and lower SE at baseline compared to participants with two timepoints. As such, it is possible the individuals with worse TST and SE at baseline improved the most over treatment. However, more likely, the more severe presentation of drinking and sleep at baseline would lead to higher likelihood of drop-outs. Future research should examine baseline predictors of PTSD/AUD on treatment response.

In sum, the current treatment study suggests combined PTSD/AUD interventions are effective in reducing PTSD symptoms and alcohol use, but only modestly improve insomnia symptoms and sleep quality and quantity. The current study provides additional evidence that insomnia is an independent disorder and sleep symptoms should be carefully assessed and treated in patients with comorbid mental health conditions. Future studies would do well to answer the best ordering of AUD, PTSD, and insomnia treatments.

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Table 1.

Demographic Characteristics (N = 119)

Characteristics	Total (n=119)	COPE (n=63)	SS (n=56)
Age, mean (SD)	41.6 (12.6)	43.2 (13.5)	39.7 (11.3)
Sex *n* (%)			
Men	107 (90%)	56 (89%)	51 (91%)
Women	12 (10%)	7 (11%)	5 (9%)
Marital status *n* (%)			
Not married	87 (73%)	45 (71%)	42 (75%)
Married	32 (27%)	18 (29%)	14 (25%)
Education *n* (%)			
High school/GED	11 (10%)	6 (10%)	5 (9%)
Some college	65 (58%)	33 (55%)	32 (62%)
College graduate	36 (32%)	21 (35%)	15 (29%)
Ethnicity *n* (%)			
Hispanic	35 (30%)	18 (29%)	17 (30%)
Non-Hispanic	83 (70%)	44 (71%)	39 (70%)
Race *n* (%)			
White	78 (66%)	41 (65%)	37 (67%)
Black	16 (13%)	8 (13%)	8 (15%)
Asian	6 (5%)	3 (5%)	3 (5%)
Other	18 (16%)	11 (17%)	7 (13%)
No. of lifetime trauma exposure events, mean (SD)	8.3 (2.7)	8.5 (2.6)	7.9 (2.8)
Taking psychotropic medication *n* (%)	78 (66%)	49 (78%)	29 (52%)
Taking sleep medication *n* (%)	53 (44.5%)	33 (52.4%)	20 (35.7%)
Taking AUD medication *n* (%)	45 (37.8%)	27 (42.9%)	18 (32.1%)
Interviewer-rated PTSD severity (CAPS-5)	42.7 (9.5)	43.2 (8.8)	42.0 (10.3)
Baseline substance use (TLFB), mean (SD)			
% days drinking alcohol in past 90 days (SD)	67.2% (22.9)	65.7% (24.5)	68.8% (21.1)
% days heavy drinking (5+ men, 4+ women) in past 90 days (SD)	51.5% (26.1)	52.5% (25.6)	50.4% (26.9)
% days drug use in past 90 days (SD)	16.6% (30.9)	16.4% (31.2)	16.8% (30.9)
Total No. of sessions attended (SD)	9.8 (4.9)	8.4 (4.6)	11.4 (4.8)
Mean number of weeks in treatment (SD)	13.61 (6.46)	12.21 (6.46)	15.19 (6.40)

Note: AUD – Alcohol use disorder; PTSD – Posttraumatic stress disorder; CAPS-5 – Clinician Administered PTSD Scale for DSM-5; TLFB – Timeline Follow-Back.

Table 2.

Pre and Post Treatment Variables (N = 119)

Characteristics	Baseline (N = 119)	Post-Tx (n = 80)	Paired T Tests	Effect Size (Cohen d)
CAPS Total	42.68 (9.48)	28.78 (14.66)	$t(80) = 8.38$, $p < .001$*	*r = 0.93*
% Days Heavy Drinking	51.53 (26.14)	16.72 (23.80)	$t(80) = 9.21$, $p < .001$*	*r = 1.03*
Insomnia Severity (ISI)	17.46 (6.45)	13.61 (6.92)	$t(78) = 4.59$, $p < .001$*	*r = 0.52*
% Insomnia Severity >= 11	83.9%	64.6%		
Sleep Diary Variables	**(n = 71)**	**(n = 38)**		
Sleep Latency (Mins)	37.21 (29.08)	39.26 (32.98)	$t(27) = -0.27$, *p = 0.78*	*r = 0.05*
WASO (Mins)	49.67 (35.00)	41.61 (33.49)	$t(30) = 2.00$, *p = 0.06*	*r = 0.36*
TST (Mins)	391.32 (80.04)	396.15 (80.39)	$t(26) = 0.54$, *p = 0.60*	*r = 0.12*
SE (%)	81.45 (10.98)	82.02 (9.38)	$t(26) = 0.05$, *p = 0.96*	*r = 0.01*
# of nightmares	0.88 (0.81)	0.81 (0.62)	$t(23) = 0.15$, *p = 0.88*	*r = 0.03*
Nap Length (Mins)	16.63 (29.66)	27.01 (40.04)	$t(30) = -2.08$, *p = 0.04**	*r = 0.37*
Actigraphy Variables	**(n = 108)**	**(n = 53)**		
WASO (Mins)	48.03 (23.93)	42.44 (19.68)	$t(52) = 2.22$, *p = 0.03**	*r = 0.30*
TST (Mins)	367.05 (81.12)	374.97 (65.24)	$t(52) = 0.41$, *p = 0.68*	*r = 0.06*
SE (%)	76.33 (10.43)	79.14 (7.44)	$t(50) = -0.88$, *p = 0.39*	*r = 0.03*
Actigraphy: Night-to-Night Variability	**(n = 108)**	**(n = 47)**		
Total Time in Bed	113.05 (74.08)	110.70 (56.77)	$t(46) = 1.43$, *p = 0.16*	*r = 0.21*
Sleep Efficiency	15.19 (11.98)	10.98 (6.23)	$t(44) = 1.33$, *p = 0.19*	*r = 0.23*
TST	117.57 (80.33)	96.49 (46.46)	$t(44) = 3.81$, *p < .001**	*r = 0.57*
WASO	31.27 (23.75)	25.21 (17.52)	$t(44) = 0.93$, *p = 0.36*	*r = 0.13*
Diary: Night-to-Night Variability	**(n = 70)**	**(n = 37)**		
Total Time in Bed	112.77 (90.51)	113.62 (84.74)	$t(31) = 0.51$, *p = 0.62*	*r = 0.11*
Sleep Efficiency	11.32 (9.19)	11.50 (11.89)	$t(25) = 0.03$, *p = 0.98*	*r = 0.01*
TST	112.71 (88.28)	123.30 (93.93)	$t(25) = 0.13$, *p = 0.90*	*r = 0.03*
WASO	41.76 (38.74)	29.15 (30.94)	$t(29) = 2.15$, *p = 0.04**	*r = 0.33*
Sleep Latency	27.67 (29.70)	32.62 (32.18)	$t(26) = -0.19$, *p = 0.85*	*r = 0.04*

Note:

*$P < .05$; CAPS – Clinician Administered PTSD Scale; WASO – Wake after sleep onset; TST – Total sleep time; SE – Sleep efficiency

Table 3.

Changes in Sleep Indices by Treatment Condition from Pre- to Post-Treatment.

Change	COPE (n = 44)	SS (n = 36)	Paired T Tests	Effect Size (Cohen d)
CAPS Total	−17.86 (15.24)	−8.82 (11.00)	$t(78) = -3.08$, $p < .003$*	*r = 0.68*
% Days Heavy Drinking	−18.61 (45.27)	−18.00 (38.09)	$t(78) = -0.07$, $p = 0.95$	*r = 0.02*
Insomnia Severity (ISI)	−3.69 (5.64)	−2.19 (5.46)	$t(76) = -1.20$, $p = 0.24$	*r = 0.27*
Sleep Diary Variables	**(n = 18)**	**(n = 13)**		
Sleep Latency(Mins)	−7.17 (15.20)	6.52 (24.34)	$t(26) = 1.66$, $p = 0.11$	*r = 0.67*
WASO (Mins)	−18.42 (28.60)	4.98 (29.83)	$t(29) = 1.26$, $p = 0.22$	*r = 0.46*
TST (Mins)	12.71 (142.08)	−27.26 (73.10)	$t(25) = -0.96$, $p = 0.35$	*r = 0.35*
SE (%)	1.91% (8.72)	−1.46% (9.15)	$t(25) = -0.96$, $p = 0.35$	*r = 0.38*
Actigraphy Variables	**(n = 32)**	**(n = 21)**		
WASO (Mins)	2.55 (11.73)	5.51 (15.75)	$t(76) = 0.74$, $p = 0.47$	*r = 0.21*
TST (Mins)	8.90 (72.84)	−12.87 (76.55)	$t(51) = -1.03$, $p = 0.30$	*r = 0.29*
SE (%)	3.9% (9.43)	−0.5% (10.65)	$t(49) = -1.50$, $p = 0.14$	*r = 0.44*

Note:

*$P < .05$; CAPS – Clinician Administered PTSD Scale; WASO – Wake after sleep onset; TST – Total sleep time; SE – Sleep efficiency