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Examining Insomnia and PTSD Over Time in Veterans in Residential Treatment for Substance Use Disorders and PTSD

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Abstract

Objective/Background: Insomnia occurs in 66–90% of individuals with posttraumatic stress disorder (PTSD) and 36–72% of individuals with substance use disorder (SUD). Individuals with both PTSD and SUD are more likely to have insomnia than individuals with only one disorder. Insomnia is associated with poorer treatment outcomes for both PTSD and SUD, increased daytime symptomology for PTSD, and increased relapse for SUDs. As such, it is important to understand how sleep affects PTSD treatment among patients dually diagnosed with SUD and how sleep changes over time in a residential unit for SUDs.

Participants: Participants were 40 veterans with comorbid PTSD and SUD in a 28-day Substance Abuse Residential Rehabilitation Treatment Program (SARRTP) PTSD track.

Methods: Analyses used mixed models with Time (baseline, posttreatment, 3-month follow-up) to examine PTSD and insomnia severity over time.

Results: Results of the longitudinal mixed model showed that PTSD symptoms improved over time but that insomnia symptoms did not. Although baseline insomnia did not affect follow-up PTSD symptoms, individuals with greater insomnia severity at the start of treatment had more severe baseline PTSD symptomatology. However, there was not an interaction of insomnia and PTSD severity over time such that baseline insomnia did not affect PTSD trajectories.

Conclusions: These findings are consistent with the PTSD outpatient treatment findings and further adds evidence that insomnia is unremitting without direct intervention. Given the relationship insomnia has with PTSD severity, SUD, and relapse, directly targeting insomnia may further help improve both PTSD and SUD treatment outcomes.

Sleep disturbances are the most commonly reported posttraumatic stress disorder (PTSD) symptoms (McLay & Volkert, 2010), with 70–91% of individuals with PTSD having co-occurring insomnia (Maher, Rego, & Asnis, 2006). Insomnia also co-occurs in 25–72% of individuals with substance use disorder (SUD; Substance Abuse and Mental Health Services

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Administration, 2014). Individuals with both PTSD and SUD are more likely to have insomnia than individuals with only one disorder (Saladin, Brady, Dansky, & Kilpatrick, 1995). Additionally, insomnia is associated with poorer treatment outcomes for both PTSD and SUD (Pigeon, Campbell, Possemato, & Ouimette, 2013). Insomnia severity is positively associated with relapse for SUDs (Brower, Aldrich, & Hall, 1998; Smith, Hill, Marshall, Keaney, & Wanigaratne, 2014), as well as worsened daytime symptomology for PTSD (Inman, Silver, & Doghramji, 1990). While integrated treatment programs targeting comorbid PTSD and SUD have been effective (Roberts, Roberts, Jones, & Bisson, 2015), it is unclear how insomnia influences treatment outcomes and whether insomnia improves with PTSD/SUD treatment. Given insomnia's association with worse PTSD symptoms and SUD relapse, it is important to understand how sleep may affect PTSD treatment, predict substance use, and how sleep changes over time through treatment.

Nightmares and difficulties with falling or staying asleep are among the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5) criteria for PTSD (American Psychiatric Association [APA], 2013). However, in patients with PTSD, insomnia has negative health consequences over and above the effects of PTSD. Those who reported insomnia complaints had significantly higher overall scores for PTSD severity at 3-month follow-up than service members without insomnia complaints (McLay & Volkert, 2010). Clum, Nishith, and Resick (2001) found that insomnia accounted for a significant portion of the variance in physical health complaints even after controlling for other PTSD symptoms and depression. Similarly, controlling for PTSD, insomnia accounts for a significant amount of variance in pain disorders, asthma, and hypertension (Green & Kimerling, 2004; Mohr et al., 2003), capacity to carry out daily activities (DeViva, Zayfert, & Mellman, 2004; Neylan et al., 1998), functional impairment, and reduced quality of life (Moul et al., 2002; Reimer & Flemons, 2003; Rosenthal & Meixner, 2003; Roth & Roehrs, 2003). Overall, insomnia is associated with greater severity of PTSD symptoms and poorer quality of life and daily functioning (Belleville, Guay, & Marchand, 2009; Maher et al., 2006).

There is evidence that restorative sleep increases generalization of fear extinction (Germain, Buysse, & Nofzinger, 2008; Pace-Schott et al., 2009), consolidation of emotional memories (Stickgold & Walker, 2007), emotional processing (van der Helm & Walker, 2009), emotional coping (Morin, Rodrigue, & Ivers, 2003), affective learning (van der Helm & Walker, 2009), fear inhibition (van der Helm et al., 2011), and cognitive abilities (Harvey, 2002) that may facilitate successful PTSD treatment. This body of research would suggest that disturbed sleep would be associated with worse PTSD treatment response. However, Lommen and colleagues (2016) used cognitive therapy for PTSD and found sleep disturbances at baseline did not predict the magnitude or speed of PTSD symptom reduction. Additionally, Sexton and colleagues (2017) also found that baseline sleep disturbances did not predict PTSD symptom reduction for prolonged exposure (PE). It should be noted that Sexton and colleagues only had a sample size of 20, which severely limits the ability to identify any interaction effects.

Although nightmares and difficulties with falling or staying asleep are part of diagnostic criteria for PTSD, the diagnostic criteria for SUDs do not include sleep symptoms (APA, 2013). However, there is evidence that insomnia and SUD have a bidirectional relationship

(Pasch, Latimer, Cance, Moe, & Lytle, 2012). Impaired sleep may cause lowered inhibition and poor emotional regulation, which can increase the risk of substance use (Wong, Brower, Nigg, & Zucker, 2010). Substances are often used as a means to cope with problems like pain, anxiety, insomnia related to PTSD, and other sleep difficulties (Conroy & Arnedt, 2014; Bonn-Miller, Babson, Vujanovic, & Feldner, 2010; Vandrey, Babson, Herrmann, & Bonn-Miller, 2014). Substance use is also known to impact sleep; however, the relationship between substance use and insomnia differs depending on the type of substance used, chronic versus acute use, and whether the individual is actively using or in withdrawal (Brower, 2003; Pasch et al., 2012).

Insomnia symptoms may last weeks, months, or even years after the initiation of abstinence of illicit substances and alcohol (Brower, 2003; Currie, Clark, Rimac, & Malhotra, 2003; Substance Abuse and Mental Health Services Administration, 2014; Williams & Rundell, 1981). For example, Drummond, Gillin, Smith, and DeModena (1998) report that in a follow-up study on alcohol-abstinent participants, abnormal patterns of sleep were present after 27 months of complete abstinence. Seventy-six percent of heavy marijuana users who stop abruptly report insomnia symptoms (Budney, Hughes, Moore, & Vandrey, 2004), which occur in the first days (Conroy & Arnedt, 2014) and at two weeks after withdrawal (Bolla et al., 2010); long-term follow-up results were not reported. For cocaine users going through withdrawal, total sleep time is reduced, sleep latency is prolonged, and sleep efficiency is decreased (Schierenbeck, Riemann, Berger, & Hornyak, 2008). The sleep troubles can last months after withdrawal (Teplin, Raz, Daiter, Varenbut, & Tyrrell, 2006). Objective measures of sleep, such as polysomnography, showed consistent deterioration in sleep (Angarita et al., 2014; Johanson, Roehrs, Schuh, & Warbasse, 1999; Kowatch, Schnoll, Knisely, Green, & Elswick, 1992).

In addition to insomnia occurring as a side effect of withdrawal, there is evidence that insomnia is a risk factor for relapse regardless of the type of substance used (Conroy & Arnedt, 2014; Brower, 2003; Brower & Perron, 2010; Currie, Clark, Hodgins, & El-Guebaly, 2004). For example, several 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, Aldrich, Robinson, Zucker, & Greden, 2001) and another study at 14-month follow-up (Drummond et al., 1998). Similar findings were reported for cannabis use where worse sleep quality prior to a quit attempt predicted higher rates of use (Babson, Boden, Harris, Stickle, & Bonn-Miller, 2013). Additionally, poor sleep during abstinence also contributed to relapse (Budney, Moore, Vandrey, & Hughes, 2003; Budney, Vandrey, Hughes, Thostenson, & Bursac, 2008; Vandrey, Smith, McCann, Budney, & Curran, 2011), and similar findings were reported for cocaine (Angarita et al., 2014) and opioid use (Brower & Perron, 2010; Wang et al., 2005).

Only one study has examined sleep, over time, for participants that have both PTSD and SUDs. McHugh et al. (2014) found that insomnia decreased during treatment and that lower insomnia severity at the end of treatment predicted lower PTSD severity at follow-up. This finding is in opposition to many of the findings that suggest insomnia does not decrease during treatment for PTSD (e.g., Keane, Fairbank, Caddell, & Zimering, 1989), decreased but still had clinical insomnia (e.g., Cooper & Clum, 1989; Galovski, Monson, Bruce, &

Resick, 2009; Gutner, Casement, Gilbert, & Resick, 2013), or had a substantial number of participants with clinical insomnia (e.g., Belleville, Guay, & Marchand, 2011; Zayfert & DeViva, 2004). Similar findings are found in the SUD literature (Bolla et al., 2010; Brower et al., 2001; Drummond et al., 1998). McHugh et al.'s result may be due to the noted limitations that they used a single item for insomnia taken from the Clinician- Administered PTSD Scale (CAPS), and did not include an independent measure of sleep. Our study aims to build upon these findings by using independent measures of sleep and PTSD. Additionally, we explore treatment in a residential setting, where PTSD, substance use, and insomnia may be more severe (Doğan, Ertekin, & Doğan, 2005).

As evidence builds that individuals with SUD can tolerate and benefit from evidence-based PTSD treatment (Roberts et al., 2015), integrated and concurrent treatment for PTSD and SUD has become more widely available (MH RRTP Program Locator, 2017). However, the relationship between PTSD/SUD treatment and insomnia is unclear. Given insomnia's association with higher PTSD symptoms and SUD relapse, it is important to understand how sleep affects PTSD treatment as well as how sleep is affected by treatment. Utilizing a sample of veterans engaging in residential PTSD/ SUD treatment, the first aim was to examine how insomnia changed over the course of PTSD and SUD treatment. We hypothesized that insomnia symptoms would not significantly change over the course of treatment. The second aim of this study was to determine whether insomnia severity at the beginning of treatment predicted PTSD severity at baseline as well as PTSD symptoms following treatment. We hypothesized that greater insomnia severity at baseline would predict greater PTSD severity at baseline and follow-up. The third aim of this study was to determine whether insomnia severity at the beginning of treatment predicted changes in PTSD symptoms over the course of treatment. We hypothesized that greater insomnia severity at baseline would predict smaller PTSD symptoms change by the end of treatment.

Method

Participants

Forty veterans in the Substance Abuse Residential Rehabilitation Treatment Program (SARRTP) at the VA San Diego Healthcare System participated in our study. SARRTP is a 28-day treatment program for veterans with SUDs; however 7-day extensions are used if additional time is needed to help meet treatment goals. Veterans entering the program with a history of trauma met with a psychologist for a PTSD assessment. Veterans who met DSM-5 criteria for current PTSD during this assessment were offered concurrent PTSD treatment on the PTSD track in addition to SUD treatment. PTSD treatment included three groups per week (in-vivo exposure, PTSD skills, cognitive-behavioral therapy for trauma). In addition, seven individuals also engaged in individual treatment delivered 2–3 times per week (Cognitive Processing Therapy, $n = 2$; PE, $n = 3$; Trauma-Informed Guilt Reduction, $n = 2$). There were no differences in age, length of stay, or insomnia with PTSD symptoms at all three time points between participants who received individual treatment versus those that received group-only treatment. While on SARRTP, veterans did not receive any sleep-specific treatment apart from medication management. PTSD track participants were offered the opportunity to provide informed consent to complete a battery of self-report measures

and have their treatment and outcomes on the unit followed for research purposes. Data from veterans who consented between February 2015 and April 2016, and who completed baseline PTSD and insomnia measures, were used in the current analyses (N = 40).

Procedures

Study procedures were approved by the local VA Institutional Review Board. The participants that consented to participate in the study completed self-report measures at baseline, posttreatment, and follow-up assessments at three months postdischarge. Some participants could not make it to the VA for an in-person appointment due to distance and others could not be reached by phone to schedule an appointment. Assessments were mailed to these participants along with a stamped, addressed return envelope. Participants were compensated up to $20 at follow-up in coupons redeemable at the VA hospital store or cafeteria.

Measures

Participants completed the following self-report measures at baseline, posttreatment, and the three-month follow-up.

PTSD symptoms

The PTSD Checklist-Specific (PCL; Weathers, Litz, Herman, Huska, & Keane, 1994) is a 17-item self-report measure of PTSD symptoms with good psychometric properties. The PCL maps directly onto DSM-IV diagnostic criteria. All analyses compared the PCL with the 2 sleep items removed to the full validated version. PCL showed strong internal consistency (α = .89).

Insomnia

The Insomnia Severity Index (ISI; Morin & 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. ISI showed strong internal consistency (α = .87). Scores of 0–14 indicate subclinical insomnia and scores of 15 and greater indicate clinical insomnia. A clinical cutoff of 15 on the ISI was used to categorize lower versus higher insomnia groups.

Medications

A chart review for medications at entry and discharge from the SARRTP unit was completed to assess medications that may affect PTSD or sleep. The classes of medications of interest to this study included anxiety, sleep, SSRI, and opioids. We used a dichotomous yes–no for each class of drugs at baseline and posttreatment.

Analytic strategy

Data were analyzed using mixed-model procedures (Raudenbush, Bryk, Cheong, Congdon, & Du Toit, 2011) using IBM SPSS v21. Mixed models allowed for all available data to be

used in the analyses. This approach takes into account all the obtained data and missingness for participants with missing data, reducing the analytic problem presented by missing data. Our Time variable used “baseline” as number of days from SARRTP entry to consent, “posttreatment” as number of days from SARRTP entry to posttreatment assessment, and “follow-up” as number of days from SARRTP entry to follow-up assessment. This approach allowed a flexible and accurate representation of time when a participant filled out questionnaires. Out of concern that the PCL has two sleep variables that may interfere with the ISI analyses, we ran all models using PCL with and without the two sleep variables; there were no significant differences in findings. Given that there were no significant differences, we chose to report results of analyses using the full PCL because this version has been evaluated for psychometric soundness and increases the ability to compare our research to other sleep-related PTSD studies with unaltered PCL totals (e.g., Sexton et al., 2017). Several random factor models using slope and intercept were tested; all models used unstructured covariance terms. Quadratic changes over time were examined, but no curvilinear relationships were found. In examining the effect of baseline sleep on PTSD over time, we used fixed effects of condition (less insomnia severity vs. worse insomnia severity) and Time, with Condition by Time as the interaction factor. All models converged on a solution but only the final models are presented as indicated by significant decreases in Log Likelihood criteria.

Results

Demographics and baseline means and standard deviations of key variables are listed in Table 1. Veterans stayed an average 26.15 (SD = 5.96; Range = 9–36) days on the SARRTP unit. Correlations between baseline ISI and baseline PCL (with sleep items included) showed a significant positive correlation ($r(40) = .54$, $p < .001$) such that worse insomnia severity was associated with more severe PTSD symptoms. Seventy percent of the participants endorsed moderate to severe insomnia, with the other 30% endorsing subclinical levels.

Examining change in PTSD symptoms over time

On average, PTSD symptoms improved 9.35 points on the PCL from baseline to 3-month follow-up. This improvement in PTSD symptoms is both statistically significant ($t[17] = 2.38$, $p = .02$) and clinically meaningful (a 10-point change in the PCL is considered clinically meaningful; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). We found that using a random intercept and slope model fit the data best. Our mixed model showed an intercept estimate of 63.02 (SE = 1.77, $t[40.32] = 35.65$, $p < .001$, 95% CI 59.45, 66.59) and significant Time (estimate = −0.0773, SE = 0.03, $t[19.47] = −2.68$, $p = .015$, 95% CI −0.14, −0.02) such that, on average, individuals decreased by 9.35 points on the PCL by follow-up (121 days after starting SARRTP). We also found a significant random intercept covariance parameter but not a significant slope parameter. This suggests that individuals had significantly different starting PCL scores when they entered the SARRTP program but did not have different slopes over time (see Figure 1 for estimated means).

Examining change in insomnia symptoms over time

We found that using a random intercept model fit the data best. Mixed models estimated the intercept at 18.63 (SE = 0.96, t[48.72] = 19.27, p < .001, 95% CI 16.70, 20.56]. As hypothesized, our mixed model showed that Time was not significant (estimate = −0.023, SE = 0.01, t[36.96] = −1.66, p = .11, 95% CI −0.05, −0.01) such that on average, participants did not significantly decrease their insomnia severity (less than a 3-point decrease on ISI from entry to follow-up). We also found a significant random intercept covariance parameter suggesting that individuals had significantly different starting insomnia scores (see Figure 1 for estimated means).

Examining whether baseline sleep affects PTSD over time

We hypothesized that individuals with higher insomnia severity at baseline would show higher PTSD symptoms at baseline, higher PTSD symptoms at follow-up, and less changes in PTSD symptoms over time. Main fixed effects were Condition (lower insomnia vs. worse insomnia) and Time, with Condition x Time as the interaction factor. We found that using a random intercept and slope model fit the data best. The mixed model showed an intercept of 66.22 (SE = 1.89, t[39.89] = 35.00, p < .001, 95% CI 62.40, 70.05). Time was significant (estimate = −0.112, SE = 0.04, t[19.19] = −3.07, p = .006, 95% CI −0.19, −0.04) such that, on average, individuals' PTSD scores decreased by 13.44 points by follow-up. Veterans with lower insomnia scores, when compared to higher insomnia severity scores, had significantly lower starting PTSD scores (estimate = −10.88, SE = 3.52, t[36.27] = −3.09, p = .004, 95% CI −18.02, −3.75). However, baseline insomnia severity did not predict PTSD severity at follow- up (estimate = −1.06 SE = 6.30, t[23.53] = −0.17, p = .89, 95% CI −14.07, 11.95). Finally, the interaction of Condition (low versus high insomnia severity) by Time was not significant (estimate = 0.09, SE = 0.06, t[15.81] = 1.53, p = .15, 95% CI −0.04, 0.22) such that veterans decreased in PTSD scores at the same rate, regardless of insomnia severity. Parameter estimates were not significant, suggesting that veterans, when grouped by low or high insomnia severity, did not vary significantly by baseline PTSD severity scores and those PTSD scores did not decrease at different rates (see Figure 2 for estimates).

Discussion

The goal of this study was to examine the relationship between PTSD/SUD treatment and insomnia on a residential unit for SUDs. Given the association between insomnia and more severe PTSD symptoms and SUD relapse, it is important to understand how sleep affects PTSD treatment as well as how sleep is affected by PTSD/SUD treatment. Consistent with the PTSD and SUD treatment literature, we found that 70% of participants endorsed moderate to severe insomnia. Additionally, we found that, although PTSD symptoms significantly improved after residential PTSD/SUD treatment, insomnia did not. Our data suggest that a residential PTSD/SUD treatment program, with a controlled environment, sobriety, wake-up, and bedtimes, does not, on its own, reduce insomnia. This finding is especially pertinent given our finding that higher insomnia was associated with higher PTSD severity at treatment entry. However, pretreatment insomnia severity did not predict changes in PTSD symptoms or PTSD symptoms at follow-up. Given insomnia's association with

worse PTSD symptoms and SUD relapse, addressing insomnia directly could be a clinically useful intervention during residential treatment.

There are several reasons why insomnia may not improve in residential treatment. Although insomnia severity is related to PTSD symptom severity and substance use relapse, it may be best conceptualized as a fully independent disorder that is not addressed by PTSD or SUD treatments. Even if insomnia initially occurs as a symptom of PTSD or SUD, it can become an independent disorder when the behavioral and cognitive responses to acute insomnia lead to perpetuating factors and conditioned arousal (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). Perpetuating factors are behaviors that solidify and maintain insomnia such as daytime napping, chronic worry about losing sleep, increased caffeine intake, taking sleeping pills, or drinking to fall asleep. Additionally, repetitive nighttime behaviors associated with PTSD (e.g., nightmares, having to check locks, hyperarousal) may lead to the pairing of the bed with wakefulness and arousal (i.e., conditioned arousal). Thus, the perpetuating factors and conditioned arousal are often responsible for the maintenance of insomnia even after PTSD symptoms or SUDs have been resolved (Bootzin, 1973; Bootzin, Epstein, & Wood, 1991). Additionally, living on a residential treatment program may exacerbate insomnia symptoms. Given the presence of roommates (Doğan et al., 2005), disturbances every two hours by nursing, environmental noise, or ambient noise within the hospital (Bartick, Thai, Schmidt, Altaye, & Solet, 2010; Topf & Thompson, 2001) and the unfamiliar environment (Hinds et al., 2007), sleep may continue to be a problem both at program entry and throughout treatment.

We did not find an interaction between insomnia and PTSD trajectories such that insomnia severity did not interfere with PTSD treatment gains. Our findings are consistent with previous studies, which found that sleep did not interfere with cognitive behavioral treatments for PTSD (Lommen et al., 2016) including PE (Sexton et al., 2017). This growing body of literature suggests that PTSD and SUDs treatments can be effective even in the context of insomnia. Taken together, insomnia may not interfere with the speed of treatment response and, as such, may not be necessary to engage in insomnia-specific treatment prior to trauma or SUD treatment. However, given the growing body of research with animals and healthy humans shows that insomnia affects acquisition, recall, and generalization of new learning that may be necessary for PTSD treatment (Fu et al., 2007; Germain, 2013; Marshall, Acheson, Risbrough, Straus, & Drummond, 2014; Pace-Schott et al., 2009; Ross et al., 1994; Spoormaker et al., 2010, 2), more research is needed to understand the role of insomnia and sleep-specific interventions in PTSD treatment outcomes.

There are reasons to address insomnia even if it does not negatively impact PTSD treatment outcomes. Insomnia by itself is distressing and impairing and is associated with SUD relapse (Brower et al., 1998; Smith et al., 2014) and PTSD severity (Inman et al., 1990). There is research that indicates that residential psychiatric units are ideal settings for treating insomnia both pharmacologically and behaviorally (Crönlein, Langguth, Geisler, Wetter, & Eichhammer, 2014; Morin, Kowatch, & O'Shanick, 1990; Tan et al., 1987). In one study, participants reported an average of 2.5 hr of sleep per night at program entry, which increased to 6 hr of sleep per night after sleep restriction therapy on a residential treatment (Morin et al., 1990). However, poor motivation by residential unit staff to consider

nonpharmacological interventions for sleep difficulties among patients may be a barrier to providing insomnia treatment during residential stay (de Niet, Tiemens, van Achterberg, & Hutschemaekers, 2011). Research examining the effectiveness of and how to best implement insomnia-specific treatment in residential settings is needed.

Cognitive behavioral therapy for insomnia (CBT-I) is the first line treatment of chronic and severe insomnia, as recognized by the National Institutes of Health (NIH) Consensus Statement (NIH, 2005), Academy of Sleep Medicine (Morin et al., 1999), and British Association of Psychopharmacology (Wilson et al., 2010). CBT-I is a behavioral treatment with strong efficacy (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Morin et al., 2006; Murtagh & Greenwood, 1995) that has been studied with patients with PTSD and SUDs (although no studies have examined CBT-I's effectiveness with PTSD and SUD comorbidity). In a review of SUD and insomnia treatment for adolescents, Bootzin and Stevens (2005) found that participants that were exposed to at least four sessions of CBT-I as a part of SUD treatment showed reduced substance abuse problems at a 12- month follow-up. Additionally, CBT-I has been shown to be effective in treating insomnia in individuals with PTSD, increasing sleep efficiency and decreasing daytime PTSD symptoms (Margolies, Rybarczyk, Vrana, Leszczyszyn, & Lynch, 2013; Talbot et al., 2013; Taylor & Pruiksma, 2014). Despite the effectiveness of CBT-I, insomnia treatment is rarely a first-line treatment with individuals who have PTSD or SUDs. Our study suggests that it would be helpful for PTSD/SUD psychologists to treat insomnia while on the residential unit.

There are several limitations to our study. First, our study was limited by the small number of participants. While we were adequately powered to detect main-effect findings, it is possible that we were underpowered for any interactions, limiting our ability to find insomnia's influence on PTSD treatment. Additionally, we were unable to do any illicit drug classification by sleep examinations and had to collapse across all drug users. Future studies could fill this gap by increasing the number of participants studied. Second, we did not have data on substance use outcomes, thus limiting our ability to comment on how PTSD and insomnia may relate to relapse. Third, PTSD and insomnia symptoms were measured by self-report rather than standardized diagnostic interview. This is particularly notable with the insomnia measures, as it is possible that the veterans were overreporting the subjective experience of sleep, when more objective measures would be more accurate (i.e., actigraphy watches and sleep diaries). Fourth, due to insufficient sample size, we were unable to examine whether insomnia affects PTSD trajectories based on group or individual treatment. Future studies should examine the differential impact of insomnia on group versus individual PTSD treatment. Fifth, although multiple efforts were made, 55% of individuals did not complete the follow-up assessments. A bias in the data is possible, in that patients who relapsed may have been less likely to complete the follow-up assessments. Because of these factors and the small sample size, results should be interpreted with caution.

Taken together, our study adds to the sleep and PTSD literature by suggesting that insomnia will not resolve on its own and may require direct interventions in PTSD and SUD treatment programs. Future studies should expand these findings by using objective indices of sleep such as actigraphy watches and sleep diaries. Additionally, examination of sleep on residential units should be expanded to consider the role of obstructive sleep apnea

(Colvonen et al., 2015). Finally, a larger sample size would allow for a detailed examination of drug type on sleep, rather than having to collapse across all drug use.

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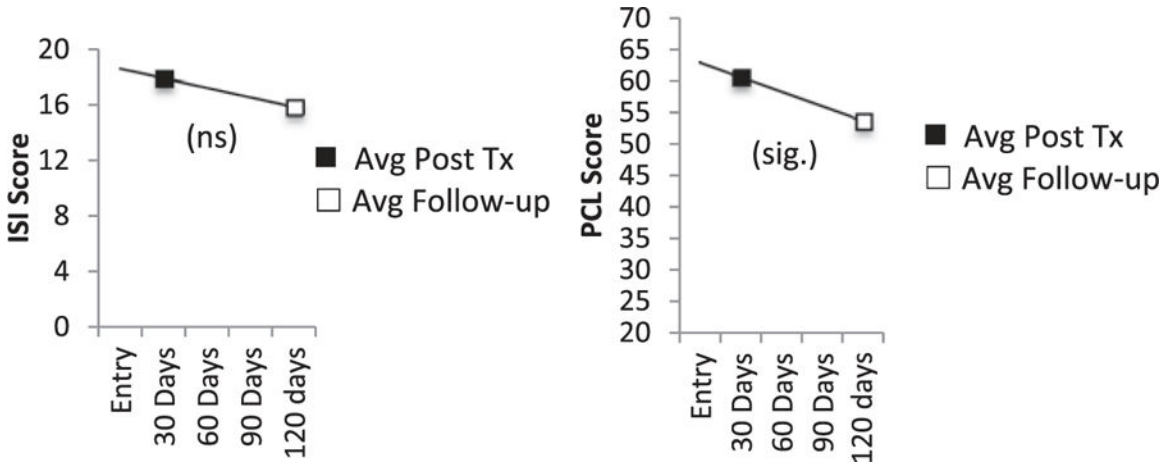

Figure 1.
Estimated insomnia and PTSD scores over time.

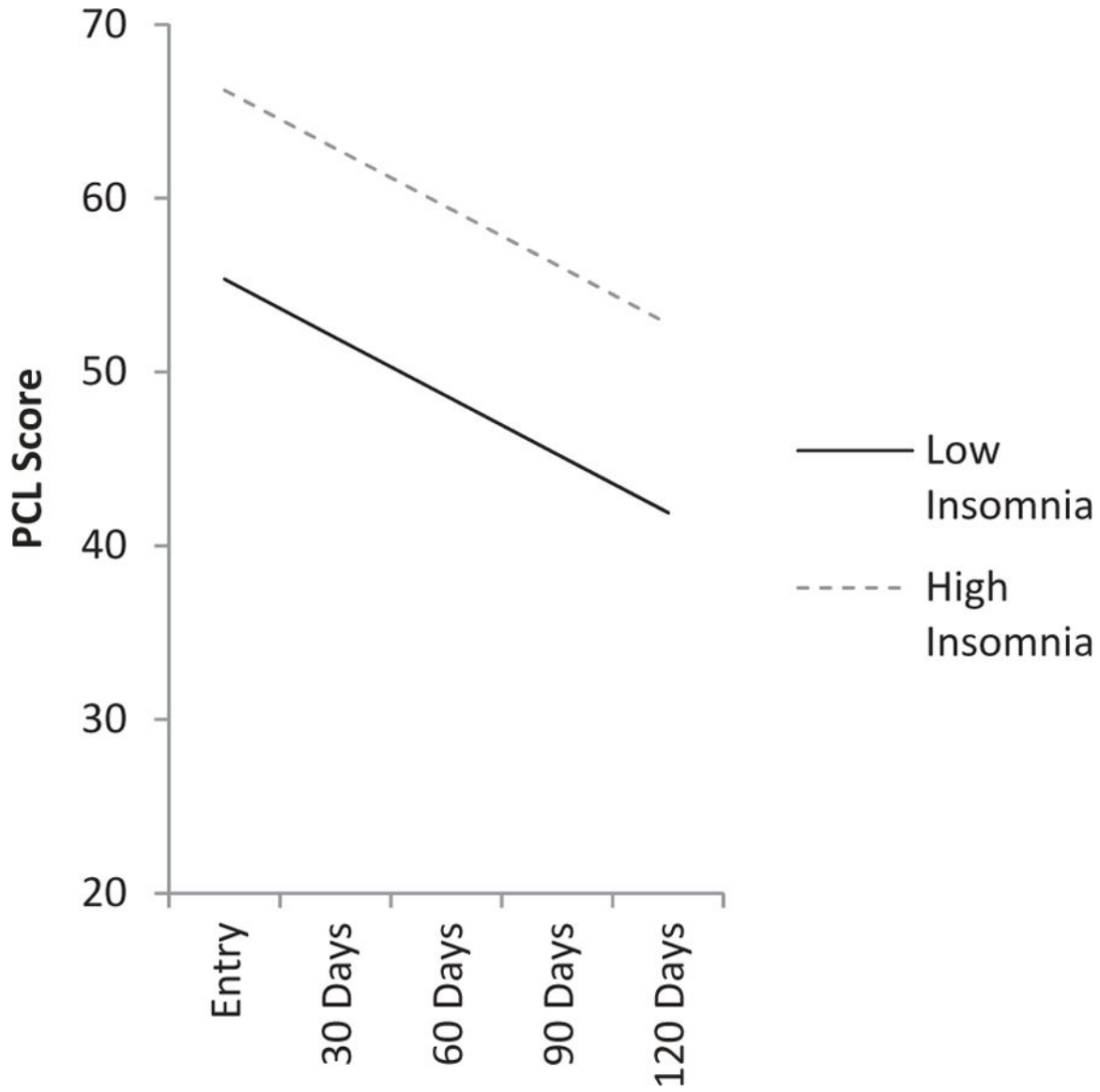

Figure 2.
Estimated PTSD scores over time, by Low versus High Insomnia groups.

Table 1.

Means and standard deviations of key variables ($N = 40$).

	Baseline *M* (*SD*)	Post-Tx *M* (*SD*)	Follow-Up *M* (*SD*)
PTSD Symptoms			
PCL	64.63 (11.15)	58.00 (11.96)	55.50 (13.13)
PCL (no sleep)	56.60 (9.95)	51.09 (10.57)	48.72 (11.34)
Sleep Symptoms			
ISI	18.53 (5.91)	17.51 (6.09)	15.75 (7.57)
Demographics			
Age	41.35 (11.48)		
Gender (% men)	95%		
% Hispanic	20%		
Days on unit	26.15 (5.96)		
Medications			
Sleep	27.5%	37.5%	
Anxiety	17.5%	17.5%	
SSRI	30.0%	30.0%	
Opioids	0%	0%	

Note. PCL-5 = The PTSD Checklist DSM-5; ISI = Insomnia Severity Index.