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Changes in Sleep Disruption in the Treatment of Co-Occurring Posttraumatic Stress Disorder and Substance Use Disorders

R. Kathryn McHugh[1,2], **Mei-Chen Hu**[3], **Aimee N. C. Campbell**[4,5], **E. Yvette Hilario**[1], **Roger D. Weiss**[1,2], and **Denise A. Hien**[3,6]

[1]Division of Alcohol and Drug Abuse, McLean Hospital, Belmont, Massachusetts, USA

[2]Department of Psychiatry, Harvard Medical School, Boston, Massachusetts, USA

[3]Department of Psychiatry, Columbia University College of Physicians and Surgeons, New York, New York, USA

[4]New York State Psychiatric Institute, Department of Psychiatry, Columbia University College of Physicians and Surgeons, New York, New York, USA

[5]Department of Psychiatry and Behavioral Health, St. Luke's Roosevelt Hospital Center, New York, New York, USA

[6]Department of Psychology, City College of the City University of New York, New York, New York, USA

Abstract

Sleep disruption appears not only to reflect a symptom of posttraumatic stress disorder (PTSD), but also a unique vulnerability for its development and maintenance. Studies examining the impact of psychosocial treatments for PTSD on sleep symptoms are few and no studies to date of which we are aware have examined this question in samples with co-occurring substance use disorders. The current study is a secondary analysis of a large clinical trial comparing 2 psychological treatments for co-occurring PTSD and substance use disorders. Women (N = 353) completed measures of PTSD at baseline, end of treatment, and 3-, 6-, and 12-month follow-ups. Results indicated that the prevalence of insomnia, but not nightmares, decreased during treatment, and that 63.8% of participants reported at least 1 clinical-level sleep symptom at the end of treatment. Improvement in sleep symptoms during treatment was associated with better overall PTSD outcomes over time, $\chi^2(1) = 33.81$, $p < .001$. These results extend the existing literature to suggest that residual sleep disruption following PTSD treatment is common in women with co-occurring PTSD and substance use disorders. Research on the benefits of adding sleep-specific intervention for those with residual sleep disruption in this population may be a promising future direction.

Sleep disruption prior to and soon after trauma exposure is associated with risk for the development of posttraumatic stress disorder (PTSD; Brewin, Andrews, Rose, & Kirk, 1999; Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2010; Harvey & Bryant, 1998; Koren,

Correspondence concerning this article should be addressed to R. Kathryn McHugh, McLean Hospital, Proctor House 3, MS 222, 115 Mill Street, Belmont, MA 02478. kmchugh@mclean.harvard.edu.

Arnon, Lavie, & Klein, 2002; Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002; Wright et al., 2011), suggesting that it may play a role in the development and maintenance of the disorder. Indeed, sleep disruption comprises two core diagnostic symptoms of PTSD: nightmares and insomnia. Sleep disturbance in those with PTSD may also worsen other symptoms and contribute to poor clinical outcomes (Krakow, Hollifield et al., 2001, 2002; Lavie, Katz, Pillar, & Zinger, 1998; Mellman, David, Bustamante, Torres, & Fins, 2001; Nishith, Resick, & Mueser, 2001; Saladin, Brady, Dansky, & Kilpatrick, 1995). This evidence suggesting that sleep disruption is a unique vulnerability factor in PTSD highlights the importance of ensuring that sleep is adequately targeted in the treatment of PTSD. Relatively little, however, is known about changes in sleep disruption in the treatment of PTSD, particularly when PTSD co-occurs with other psychiatric disorders.

Substance use disorders (SUDs) co-occur with PTSD at a high rate (Breslau, Davis, & Schultz, 2003; Sartor et al., 2010) and also are associated with sleep disruption, particularly insomnia. Although sleep disruption can be a direct result of substance use or withdrawal (Mahfoud, Talih, Streem, & Budur, 2009; Vitiello, 1997), sleep difficulties often continue past acute withdrawal from a range of substances of abuse (Brower, Krentzman, & Robinson, 2011; Sharkey et al., 2011). Sleep disruption is associated with greater functional impairment (Arnedt et al., 2007) and greater likelihood of relapse in SUDs (Brower, Aldrich, & Hall, 1998; Conroy et al., 2006) and may serve as a motivator for continued use, given evidence that individuals with insomnia often attempt to self-medicate with alcohol or other drugs (Brower, Aldrich, Robinson, Zucker, & Greden, 2001; Mahfoud et al., 2009; Vitiello, 1997). Results have been mixed, however, with respect to whether the co-occurrence of PTSD and SUDs is associated with elevated sleep disruption relative to each disorder alone (Saladin et al., 1995; Waldrop, Back, Sensenig, & Brady, 2008). The importance of sleep disruption in both of these disorders, their high rates of co-occurrence, and potential interactions among symptoms (e.g., use of substances to cope with poor sleep) underscore the potential importance of addressing sleep symptoms in individuals with co-occurring PTSD and SUDs.

The overarching aim of this analysis was to examine the nature of sleep disruption in the treatment of co-occurring PTSD and SUDs. Although standard frontline treatments for PTSD have been associated with improvement in sleep disruption, many patients continue to experience significant symptoms following treatment, even when other PTSD symptoms improve (Galovski, Monson, Bruce, & Resick, 2009; Zayfert & DeViva, 2004). For example, in a study of cognitive–behavioral therapy for PTSD, despite overall improvements in PTSD symptoms, 70.2% of participants with clinically significant sleep disruption prior to treatment continued to exhibit sleep problems following treatment (Belleville, Guay, & Marchand, 2011). An important and understudied question is whether a failure to achieve improvements in sleep symptoms with treatment is predictive of poorer overall PTSD outcomes over time. Moreover, to our knowledge no published studies to date have examined sleep disruption in the context of treatment of those with co-occurring PTSD and SUDs.

This study is a secondary analysis of a large treatment outcome trial conducted in the National Drug Abuse Treatment Clinical Trials Network comparing the effectiveness of two

active treatments for co-occurring PTSD and SUDs: Seeking Safety, a manualized cognitive-behavioral treatment for PTSD and SUDs (Navajits, 2002) and Women's Health Education, an active comparison treatment primarily consisting of psychoeducation (Hien et al., 2009; Miller, Pagan, & Tross, 1998). Overall study results suggested substantial improvement in PTSD severity in both groups, with no overall significant differences in the intent-to-treat analyses between the treatment groups on outcomes. There were no significant changes in SUD symptoms in either treatment group; however, this may be attributed to high rates of drug abstinence at the time of randomization to this outpatient treatment study (46% abstinent at enrollment). See the Method section for additional information on sample composition and study design.

Our specific aims were to examine changes in nightmares and insomnia during the treatment period and to evaluate whether improvement in these sleep disruption symptoms during treatment was associated with PTSD symptoms at 1-week post-treatment and all follow-up points within 1 year (i.e., whether sleep disruption that did not improve with treatment was a poor prognostic indicator). We hypothesized that nightmares and insomnia would decrease from pre- to posttreatment, and greater improvement in sleep during treatment would be associated with better PTSD outcomes at posttreatment and follow-up. Nightmares and insomnia were evaluated separately given that these are distinct symptoms that are organized in different clusters of the diagnostic criteria for PTSD (reexperiencing and hyperarousal, respectively), a characterization that has been supported empirically (Babson et al., 2011). Given that substance use did not significantly decrease in the main outcomes from this trial, we did not hypothesize that sleep would be associated with substance use outcomes. Nonetheless, we examined this association in an exploratory analysis.

Method

Participants and Procedures

Data were derived from 353 women enrolled in the National Drug Abuse Treatment Clinical Trials Network trial, CTN-0015: Women's Treatment for Trauma and Substance Use Disorders, who were also receiving outpatient substance abuse treatment from seven treatment programs located across the continental United States. Eligible participants (a) met *Diagnostic and Statistical Manual for Mental Disorders, 4th ed.* (*DSM-IV*; American Psychiatric Association [APA], 2000) criteria for full or subthreshold PTSD (subthreshold PTSD required that participants met either symptom cluster C, avoidance or numbing, or D, increased arousal, instead of both), (b) reported substance use within the past 6 months and met criteria for a current diagnosis of drug or alcohol abuse or dependence, (c) were aged 18–65 years, and (d) were proficient in English. Participants were excluded if they showed a significant risk of suicidal/homicidal intent or behavior, had a history of schizophrenia-spectrum diagnosis, or psychosis in the past 2 months. Participants currently taking medication for psychiatric disorders (including sleep disruption) were eligible for the study (47.6% of the sample).

Participants were recruited from within their respective treatment programs and through advertisements over 21 months in 2004 and 2005. Written informed consent was obtained from participants after they received a complete description of the study, and prior to all

assessments. After completing an eligibility screening followed by a baseline assessment, participants were randomly assigned to 6 weeks of Seeking Safety or Women's Health Education (see below). Randomization was stratified by the presence of prescription psychotropic medication and substance use diagnosis (either alcohol use disorder only or drug use disorder with or without alcohol use disorder). Participants completed brief weekly assessments during treatment and at 1 week and 3-, 6-, and 12-months after the end of treatment. Additional description of study participants and design may be found in an earlier publication (Hien et al., 2009).

Sociodemographic variables and baseline (pretreatment) values for PTSD and sleep disruption severity are presented in Table 1. Of the 353 participants, 12.2% (n = 43) reported a prescription for a sleep aid medication at baseline. There were no significant differences between treatment groups on any of these baseline variables.

After randomization, participants met individually with their study counselor to orient them to their assigned group (e.g., group structure and rules). Counselors and supervisors were selected from each treatment program and centrally trained in their respective study interventions. Treatment groups had an open, rolling enrollment format similar to other outpatient treatment modalities (i.e., women could start group at any time). Participants attended two groups per week for approximately 6 weeks and each group session lasted about 75–90 minutes.

Seeking Safety is a structured cognitive–behavioral therapy integrating safety, trauma, and substance use components into each session (Navajits, 2002). Twelve core Seeking Safety sessions were selected from a possible 25 to fit standard outpatient treatment durations. Each session covered a different topic, but included basic education on SUDs and PTSD, skill-building to prevent drug use and manage PTSD symptoms, cognitive restructuring with attention to maladaptive thoughts linked to substance use and trauma symptoms, and a focus on developing effective communication skills to build healthy support networks.

The Women's Health Education control condition was adapted from a treatment grant protocol for female partners of injection drug users (Miller et al., 1998). It is a psychoeducational, manualized treatment focused on health topics of particular relevance to this population (e.g., pregnancy, nutrition, diabetes, hypertension, human immunodeficiency virus, and sexually transmitted diseases). Women's Health Education was designed to provide equivalent therapeutic attention, but without the theory-driven techniques of Seeking Safety, nor any explicit focus on substance abuse or trauma.

Measures

Basic demographic data were collected including age (in years), race/ethnicity, and education (in years). Race/ethnicity was categorized as African American/Black, Caucasian, Latina/Hispanic, or Multiracial/Other (due to sample size restrictions). Prescribed medications were collected and categorized at baseline, including a sleep aid category.

PTSD was measured using two assessments: the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995; Weathers, Keane, & Davidson, 2001) and the Posttraumatic

Stress Disorder Symptom Scale-Self-Report (PSS-SR; Foa, Riggs, Dancu, Constance, & Rothbaum, 1993). The CAPS is a structured interview administered by a trained clinician and measures *DSM-IV* (APA, 2000) PTSD diagnosis through the assessment of symptom frequency and intensity in the past 30 days. The total score combines frequency and intensity of symptoms and has a range of 0–136. The scale was administered at baseline and all follow-up time points. Internal consistency reliability for the CAPS was $\alpha = .90$. The PSS-SR is a 17-item self-report instrument that measures severity (range = 17–85) in the prior 7 days. The PSS-SR was completed at all time points, including weekly during treatment. Internal consistency reliability for the PSS-SR was $\alpha = .87$.

Both the CAPS and the PSS-SR include specific symptom questions of sleep disruption related to distressing dreams/nightmares, and having difficulty/trouble sleeping. The PSS-SR was collected weekly during treatment and thus was used to assess changes in treatment. The CAPS was collected at major assessment points (baseline, end-of-treatment, and follow-up assessments) and thus was used to assess the relationship between sleep disruption changes and other PTSD symptoms over the course of follow-up. On the PSS-SR items were dichotomized to reflect presence or absence of a clinical level of nightmares and insomnia. Scores of 3 (*moderately*) or higher were coded as presence of the symptom. Nightmare and insomnia items from the CAPS were dichotomized with both *moderately or higher* intensity and *at least once* frequency required to be coded as presence of the symptom. Thus, these items were dichotomized to indicate clinical levels of sleep disruption symptoms. In addition, this approach minimized collinearity with total PTSD symptoms. A dummy variable for any improvement in sleep symptoms from baseline to the end of treatment was created by subtracting the summation of sleep symptoms posttreatment score from baseline. Participants who reported no sleep symptoms at baseline and continued to report no symptoms at the end of treatment were coded as exhibiting improvement in sleep symptoms (analyses were run with and without these participants; $n = 19$).

The Addiction Severity Index-Lite (ASI; McLellan, Cacciola, & Zanis, 1997) is an interviewer-administered semistructured interview that assesses substance use in the prior 30 days; it was administered at baseline and all follow-up points. The maximum number of days of use across 10 substance use categories (alcohol, cocaine, marijuana, heroin, other opiates, barbiturates, other sedatives, stimulants, inhalants, and hallucinogens) was categorized into three levels, a common coding convention of the ASI: abstinence (no use), light use (used 1–12 days), and heavy use (used 13 or more days, i.e., more than 3 days per week; McLellan et al., 1985).

Data Analysis

To examine whether sleep symptoms improved with treatment, we conducted separate generalized logistic models for nightmares and insomnia. Self-reported nightmares and insomnia from the PSS-SR were used as the dependent variables in these analyses because these data were collected weekly, thus allowing us to examine change each week. In these models, independent variables included study week and treatment condition, with the presence of a sleep medication at baseline, baseline PTSD severity (PSS-SR total score), baseline sleep disruption severity (either nightmares or insomnia), sociodemographic

variables, and study site in the model. The generalized estimating equation (GEE) approach was used to estimate and test the model.

Next we utilized the CAPS because this clinician-administered measure was available at all major assessment points of interest for this aim. To examine whether improvement in sleep during treatment was associated with PTSD symptoms at 1-week following treatment completion and throughout the follow-up period, including 3-, 6-, and 12-month assessments, we used the GEE approach and with baseline PTSD severity, baseline substance use, presence of a sleep aid at baseline, treatment condition, sociodemographic variables, and study site in the model. Therapy group was not included as an additional fixed effect in the model because for the majority of sites each group was run continuously at each site by a single counselor. Thus, much of this effect is accounted for by the inclusion of site and treatment condition in the model. This same model was then run with substance use in the previous 30 days (defined categorically as abstinence, light use—1–12 days, and heavy use—13 or more days) as the dependent variable. In all models, Treatment × Time interactions were evaluated; these were only included in the final model if they were statistically significant at an α of .05.

The GEE methodology was used for these models because this approach is appropriate for correlated data arising from repeated measurements, does not require a parametric distribution, and allows for the analysis of data with some missing observations. These analyses were conducted using PROC GENMOD in SAS Version 9.3.

Results

We first examined whether sleep disruption symptoms improved during treatment. In the model examining self-reported nightmares, the treatment by week interaction was significant, $\chi^2(1) = 4.84$, $p = .028$, characterized by a higher prevalence of nightmares in the Seeking Safety group relative to the Women's Health Education group in the early weeks of treatment (Week 1: 40.1% Women's Health Education, 44.9% Seeking Safety) with a slightly lower prevalence in the Seeking Safety group later in treatment (Week 7: 23.9% Women's Health Education, 22.2% Seeking Safety). PTSD severity at baseline, $\chi^2(1) = 31.52$, $p < .001$ and presence of nightmares at baseline, $\chi^2(1) = 9.70$, $p = .002$, were significantly positively associated with the presence of nightmares over time. African American participants were less likely than Caucasians to report nightmares, odds ratio (*OR*) = 0.52, 95% confidence interval (CI) = [0.31, 0.87], $p = .011$ (see Table 2).

In the generalized logistic model examining self-reported insomnia over time, there was a significant association between study week and insomnia, $\chi^2(1) = 18.34$, $p < .001$, characterized by decreased rates of insomnia over the study period for both groups, *OR* = 0.88, 95% CI = [0.83, 0.93], $p < .001$. Insomnia at baseline, $\chi^2(1) = 26.26$, $p < .001$, and baseline PTSD severity, $\chi^2(1) = 17.58$, $p < .001$, were positively associated with presence of insomnia over time. There were no main or interaction effects with treatment type and no effect of the presence of a sleep medication at baseline (see Table 3).

We next examined whether improvement in sleep disruption symptoms was associated with PTSD symptoms and substance use at follow-up. The majority of participants reported at least one sleep disruption symptom (nightmares or insomnia) at a clinical level (at least moderate levels of intensity and frequency) on the CAPS at baseline (87.4%), with more than half of participants (54.6%) reporting clinical levels of both insomnia and nightmares. At the end of treatment, 63.8% of participants reported at least one sleep symptom and 28.5% reported both nightmares and insomnia. From baseline to 1 week following treatment completion, 56.5% of participants reported improvement in at least one sleep symptom.

The model examining whether improvement in sleep disruption during treatment was associated with PTSD outcome at posttreatment follow-up indicated a significant effect of improvement in sleep, $\chi^2(1) = 33.81$, $p < .001$, characterized by better PTSD outcomes among those who improved in sleep. PTSD severity at baseline was associated with severity at follow-up, $\chi^2(1) = 31.33$, $p < .001$, and there was a significant main effect of time, $\chi^2(1) = 52.83$, $p < .001$, with PTSD scores reducing over time (see Table 4). Excluding the 19 participants who reported no sleep disruption throughout the trial did not significantly impact these results.

The same model was run examining substance use outcomes. Improvement in sleep disruption during treatment was not associated with substance use outcomes at posttreatment and follow-up, $\chi^2(1) = 0.06$, $p = .813$.

Discussion

Accumulating evidence suggesting that sleep disruption is a common risk and aggravating factor across a wide range of psychiatric disorders has yielded a call to consider sleep disruption as a syndrome that may require specific targeting with intervention, rather than as a symptom that will remit with treatment of the “primary” disorder (e.g., Harvey, Murray, Chandler, & Soehner, 2011; Lavie, 2001; Ross, Ball, Sullivan, & Caroff, 1989). In this secondary data analysis from a large treatment outcome trial for women with co-occurring PTSD and SUDs, we found that insomnia significantly improved during the treatment period. Nightmares exhibited smaller, nonsignificant reductions that were characterized by a Treatment × Time interaction, with a steeper decline in the Seeking Safety condition. Despite this evidence for improvement, only 36.2% of participants reported no clinical level (i.e., lower than moderate frequency and intensity) sleep symptoms at the end of treatment. At the completion of treatment, 11.1% of the sample reported continued nightmares, 24.2% reported continued insomnia, and over 28.5% reported both.

Examination of the impact of improvement in sleep on subsequent PTSD outcomes suggested that those whose sleep improved to a nonclinical level also had better overall PTSD outcomes at the end of treatment and throughout 1-year of follow-up, even when controlling for baseline PTSD symptom severity. Although based on the design of the study we are unable to draw causal conclusions, this finding implies that among those with clinical-level residual sleep disruption, risk for continuation or exacerbation of overall PTSD symptoms may be higher. This is consistent with recent trends in the treatment of PTSD, where studies of the addition of sleep-focused interventions to existing PTSD treatments

have shown early promise (Germain et al., 2012; Krakow, Hollifield et al., 2001). The results of the current study similarly indicate that studies examining the efficacy of sleep interventions (e.g., imagery rehearsal therapy for nightmares, cognitive-behavioral therapy for insomnia) in those with co-occurring PTSD and SUDs could be informative.

Improvement in sleep disruption was not associated with substance use outcomes in this study. These results should be interpreted within the context of the primary outcomes from this study, which indicated no significant reduction in substance use over the course of treatment (this may have been attributable, at least in part, to low levels of use at treatment initiation). It is possible that sleep disruption would also be associated with worse substance use outcomes in a sample with heavier substance use at baseline; thus, further research is needed to determine whether improvements in sleep disruption is associated with substance use outcomes.

Our study focused on self-reported insomnia and nightmares; however, sleep-related disturbances in PTSD include a number of other features, such as breathing and movement symptoms (e.g., Krakow, Melendrez et al., 2001; Ross et al., 1994). The investigation of these symptoms in co-occurring PTSD and SUDs is needed to better understand the range of sleep pathology in this group and the degree to which standard treatments may address these symptoms.

There are several limitations to this study. Most importantly, this trial did not include an independent measure of sleep disruption, and we were thus limited to using sleep items embedded within the two validated PTSD symptom assessments. Consequently, we cannot rule out the possibility that observed effects could be attributed to improvement in PTSD. Nonetheless, when controlling for other PTSD symptoms, these effects remained, enhancing confidence in these results. Moreover, our results are consistent with previous trials suggesting that even when other PTSD symptoms improve, sleep disruption does not necessarily improve (Brower et al., 2011; Sharkey et al., 2011). Increasingly, the use of both self-report and polysomnography to study sleep disruption has become the gold standard in the field of sleep research. Our study was limited by the absence of such physiological measures, and replication of these findings with such methods is needed to enhance confidence in these results. Additionally, because the majority of participants used more than one substance, the ability to control for specific substances of abuse was not possible. Due to the use of rolling admission groups, we were unable to include therapy group in the analysis. Thus, although some of the effect of group is accounted for in models by the inclusion of site and intervention, we cannot fully account for additional variance attributable to group composition. Finally, because this sample was comprised exclusively of women, the generalizability of these findings to men is unknown.

In a large clinical trial, women receiving Seeking Safety for co-occurring SUDs and PTSD evidenced significantly greater reductions over time in nightmares than women who received a comparison health education intervention. Both treatments were associated with significant improvements in insomnia, with no significant differences between groups. A large percentage of the sample continued to experience at least one symptom of sleep disruption at posttreatment. Continued sleep disruption at the end of treatment was

associated with worse PTSD outcomes through 1 year of follow-up. Consideration of the addition of interventions targeted to sleep disruption may be indicated in those with PTSD and SUDs, or considered as an adjunctive intervention if residual sleep disruption is present at the time of treatment completion.

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Table 1

Demographic, Substance Use, and PTSD Characteristics at Baseline

	Seeking Safety (*n* = 176)		Women's Health Education (*n* = 177)	
Variable	***M* or *n***	***SD* or %**	***M* or *n***	***SD* or %**
Age (years)	39.36	9.50	39.04	9.12
Race/ethnicity (%)				
African American/Black	58	33.0	62	35.0
Caucasian	83	47.2	78	44.1
Latina/Hispanic	7	4.0	16	9.0
Multiracial/Other	28	15.9	21	11.9
Education (years)	12.65	2.32	12.42	2.56
Prescribed a medication for sleep (%)	21	11.9	22	14.4
Days of substance use (past 30 days)	8.96	11.14	8.44	11.14
0 (%)	67	38.1	75	42.4
1–12 (%)	56	31.8	47	26.6
≥ 13 (%)	53	30.1	55	31.1
CAPS (baseline)				
PTSD severity (total)	61.56	19.36	64.16	19.40
Insomnia (%)	138	78.4	134	75.7
Nightmare (%)	113	64.2	103	58.2
PSS-SR (baseline)				
PTSD severity (total)	45.53	15.25	45.59	15.34
Insomnia (%)	123	69.9	122	68.9
Nightmares (%)	79	44.9	71	40.1

Note. *N* = 353. No group differences were statistically significant. CAPS = Clinician Administered PTSD Scale; PTSD = posttraumatic stress disorder; PSS-SR = Posttraumatic Stress Scale-Self-Report.

Table 2

Generalized Logistic Model on Nightmares During Treatment

Variable	Estimate	*SE*	*OR*	95% CI
Age	0.01	0.01	1.01	[0.99, 1.03]
Race/ethnicity				
Caucasian (ref)				
African American	−0.65	0.26	0.52**	[0.31, 0.87]
Latina	0.16	0.51	1.17	[0.43, 3.19]
Other	−0.02	0.28	0.98	[0.57, 1.70]
Education	−0.02	0.05	0.98	[0.89, 1.08]
Baseline sleep aid	−0.17	0.27	0.84	[0.50, 1.43]
Baseline nightmare severity	0.78	0.24	2.18**	[1.36, 3.49]
Study week	−0.02	0.05	0.98	[0.89, 1.08]
Treatment condition (Seeking Safety vs. Women's Health Education)	0.51	0.29	1.67	[0.94, 2.94]
Treatment Condition × Week	−0.14	0.06	0.87*	[0.77, 0.98]
PSS-SR total score	0.06	0.01	1.06**	[1.04, 1.08]
Number of days used drugs in the past 30 days at baseline				
0 (ref)				
1–12	−0.06	0.26	0.94	[0.57, 1.57]
13–30	−0.34	0.28	0.71	[0.41, 1.23]

Note. $N = 308$. *OR* = odds ratio; *SE* = standard error; CI = confidence interval; PSS-SR = Posttraumatic Stress Disorder Symptom Scale-Self-Report.

* $p < .05$.

** $p \leq .01$.

Table 3

Generalized Logistic Model on Insomnia During Treatment

Variable	Estimate	*SE*	*OR*	95% CI
Age	0.01	0.01	1.01	[0.99, 1.03]
Race/ethnicity				
Caucasian (ref)	–			
African American	−0.46	0.24	0.63	[0.39, 1.01]
Hispanic/Latina	−0.29	0.42	0.75	[0.34, 1.67]
Other	0.32	0.27	1.38	[0.81, 2.34]
Education	−0.05	0.04	0.95	[0.88, 1.03]
Baseline sleep aid	0.18	0.27	1.20	[0.71, 2.03]
Baseline insomnia severity	1.40	0.24	4.06***	[2.53, 6.49]
Study week	−0.13	0.03	0.88***	[0.83, 0.93]
Treatment condition (Seeking Safety vs. Women's Health Education)	−0.20	0.20	0.82	[0.55, 1.21]
PSS-SR total score	0.04	0.01	1.04***	[1.02, 1.06]
Drug use in the past 30 days (vs. 0 days)				
Used 1–12 days	0.06	0.24	1.06	[0.66, 1.70]
Used 13–30 days	0.02	0.26	1.02	[0.61, 1.70]

Note. $N = 308$. *OR* = odds ratio; *SE* = standard error; CI = confidence interval; PSS-SR = Posttraumatic Stress Disorder Symptom Scale-Self-Report.

*** $p < .001$.

Table 4

Association Between Change in Sleep and CAPS at Posttreatment, 3-, 6-, and 12-Month Follow-up

Variable	Estimate	*SE*	95% CI
Age	0.06	0.14	[−0.21, 0.32]
Race/ethnicity			
Caucasian (ref)			
African American	1.48	2.70	[−3.82, 6.78]
Latina	2.56	5.22	[−7.67, 12.79]
Other	−1.03	3.14	[−7.19, 5.13]
Education	−0.26	0.57	[−1.37, 0.84]
Time	−0.28***	0.03	[−0.34, −0.22]
Treatment condition (Seeking Safety vs. Women's Health Education)	−2.31	2.11	[−6.45, 1.83]
Change in sleep	−15.03***	2.29	[−19.51, −10.55]
Baseline CAPS total score	0.43***	0.06	[0.32, 0.54]
Sleep aid at Baseline	6.60**	2.65	[1.40, 11.79]
Number of days used drugs in the past 30 days at baseline			
0 (ref)			
1–12 days	0.97	2.75	[−4.42, 6.36]
13–30 days	−0.62	3.32	[−7.13, 5.88]

Note. $N = 206$. *SE* = standard error; CI = confidence interval; CAPS = Clinical Administered PTSD Scale; PTSD = posttraumatic stress disorder; PSS-SR = Posttraumatic Stress Scale-Self-Report.

** $p \leq .01$.

*** $p < .001$.