

Residual Insomnia and Nightmares Postintervention Symptom Reduction Among Veterans Receiving Treatment for Comorbid PTSD and Depressive Symptoms

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While evidence-based interventions can help the substantial number of veterans diagnosed with comorbid PTSD and depression, an emerging literature has identified sleep disturbances as predictors of treatment nonresponse. More specifically, predicting effects of residual insomnia and nightmares on postintervention PTSD and depressive symptoms among veterans with comorbid PTSD and depression has remained unclear. The present study used data from a clinical trial of Behavioral Activation and Therapeutic Exposure (BA-TE), a combined approach to address comorbid PTSD and depression, administered to veterans ($N = 232$) to evaluate whether residual insomnia and nightmare symptoms remained after treatment completion and, if so, whether these residual insomnia and nightmare symptoms were associated with higher levels of comorbid PTSD and depression at the end of treatment.

Participants (ages 21 to 77 years old; 47.0% Black; 61.6% married) completed demographic questions, symptom assessments, and engagement-related surveys. Hierarchical multiple linear regression models demonstrated that residual insomnia was a significant predictor of PTSD and depression symptom reduction above and beyond the influence of demographic and engagement factors (e.g., therapy satisfaction). Consistent with previous research, greater residual insomnia symptoms were predictive of smaller treatment gains. Findings illustrate the potential significance of insomnia during the course of transdiagnostic treatment (e.g., PTSD and depression), leading to several important clinical assessment and treatment implications.

Keywords: PTSD; depression; insomnia; treatment engagement; veterans

THE LIFETIME PREVALENCE of posttraumatic stress disorder (PTSD) in the general population is about 1 in 10 (8.3%; Kilpatrick et al., 2013), but rates are even higher among veterans. A meta-analysis of 33 studies estimated that nearly 1 out of every 4 (23%) veterans has PTSD (Fulton et al., 2015). Onset of PTSD increases the risk for later development of depression (Ginzburg et al., 2010; Stander et al., 2014), and therefore, unsurprisingly, approximately half of the veterans with a PTSD diagnosis also meet criteria for major depressive disorder (MDD; Ikin, Creamer, Sim, & McKenzie, 2010; Rytwinski, Scur, Feeny, & Youngstrom, 2013). Compared to veterans who have depression without PTSD, veterans with both diagnoses are more likely to report suicidal ideation, experience more emotional distress, endorse lower levels of social support, attend more outpatient visits, and incur higher overall costs for outpatient mental health care (Campbell et al., 2007; Chan, Cheadle, Reiber, Unützer, & Chaney, 2009).

Fortunately, interventions with decades of research support are available to address both PTSD and depression. For the treatment of PTSD, imaginal exposure (i.e., repeated retelling of the trauma narrative) and in vivo exposure (i.e., exposure to situations that serve as trauma reminders) are among the therapeutic techniques with the most research support to date (Lancaster, Teeters, Gros, & Back, 2016; Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010; S. Taylor et al., 2003). Each of these techniques involves acting in opposition to one's emotional tendencies—actively approaching situations and thoughts that would normally be avoided due to fear. Behavioral activation (BA) therapy for depression shares a similar rationale; BA involves encouraging depressed patients, who would typically

isolate themselves and withdraw from activities, to instead actively schedule and participate in enjoyable and meaningful activities (Lejuez, Hopko, & Hopko, 2001). More recently, research has demonstrated that these treatment techniques can be delivered seamlessly in veterans with PTSD and depression as well as comorbid PTSD and depression (Gros, 2014).

Among veterans, exposure therapy for PTSD (Rauch et al., 2009; Tuerk et al., 2011; Yuen et al., 2015) and BA for depression (Hershenberg, Smith, Goodson, & Thase, 2017; Jakupcak et al., 2010), as well as a combined approach to address comorbid PTSD and depression called Behavioral Activation and Therapeutic Exposure (BA-TE; Acierno et al., 2016; Gros et al., 2011; Gros et al., 2012a), all produce clinically significant improvements in symptoms. However, a more careful examination of the data, looking beyond group averages, suggests that there is still much room for improvement. A proportion of veterans prematurely discontinues treatment (e.g., 13%–39% in exposure therapy for PTSD, 16% in BA for depression, and 20.6% in BA-TE; Acierno et al., 2016; Steenkamp, Litz, Hoge, & Marmar, 2015; Hershenberg et al., 2017), and another subgroup of veterans does not fully respond to treatment (Rauch et al., 2009). For example, researchers found that 20% of veterans receiving prolonged exposure for PTSD, one specific type of exposure therapy for PTSD, did not experience clinically significant improvement (Rauch et al., 2009). Among veterans receiving BA, Hershenberg and colleagues (2017) found that 42% did not demonstrate reliable improvement, and 65% did not experience clinically significant improvement in depression symptoms. Although on average veterans respond well to gold-standard treatments for PTSD and depression, the percentage of premature discontinuation and nonrespondents is substantial. These findings have spurred a number of studies aimed to identify predictors of treatment outcome.

Nonresponsiveness to PTSD treatment has been associated with factors such as pretreatment severity, comorbid personality disorders, high expressed emotion in the family, heightened neural response to fear-related stimuli, deficits in inhibition and in processing of contextual cues, and lower physiological response to trauma-related cues at baseline (Bryant et al., 2008; Norrholm et al., 2016; Schottenbauer et al., 2008; Van Rooij, Geuze, Kennis, Rademaker, & Vink, 2015). Similarly, nonresponsiveness to treatment for depression has been associated with factors such as psychiatric comorbidities, higher number of past hospitalizations, functional and structural biomarkers in the frontal-striatal limbic network, delays in initiating

treatment, and lower treatment outcome expectancies (Beard et al., 2016; Berlim & Turecki, 2007; Fu, Steiner, & Costafreda, 2013). Many of these factors are not promising candidates for further psychotherapy. However, emerging research in both PTSD and depression is pointing to insomnia as a likely predictor of treatment outcome (Dolsen, Asarnow, & Harvey, 2014; Wright et al., 2011), and it has been well-established that cognitive behavioral therapy for insomnia is an effective insomnia treatment in a number of comorbid populations (Smith, Huang, & Manber, 2005; Taylor & Pruiksma, 2014; Wu, Appleman, Salazar, & Ong, 2015) including PTSD (Baddeley & Gros, 2013; DeViva, Zayfert, Pigeon, & Mellman, 2005; Ho, Chan, & Tang, 2016; Ulmer, Edinger, & Calhoun, 2011) and depression (Ashworth et al., 2015; Balleisio et al., 2018; Manber et al., 2008).

Nightmares are another sleep-related problem that may be tied to treatment outcome, though this relationship is less clear. While most participants in studies have reported experiencing reduction in nightmares following PTSD treatment, some do not see significant improvement (Gutner, Casement, Gilbert, & Resick, 2013; Pruiksma et al., 2016; Zayfert & DeViva, 2004). Similar to insomnia, there are evidence-based interventions, such as Image Rehearsal Therapy (IRT) and Exposure, Relaxation, and Rescripting Therapy (ERRT), that serve as effective psychotherapy treatments for trauma-related nightmares (Davis & Wright, 2006; Krakow & Zadra, 2006).

Epidemiological studies suggest that the sleep disturbance is common in PTSD, as estimates of insomnia vary from 40%–50% in patients with PTSD (Neylan et al., 1998; Ohayon & Shapiro, 2000); with up to 90% reported in veteran samples with PTSD (Lewis, Creamer, & Failla, 2009). Prevalence of nightmares in PTSD patients ranges between 50%–70% (Leskin et al., 2002; Neylan et al., 1998) and sleep-disordered breathing around 50% (Krakow et al., 2004). Insomnia and nightmares may contribute to the severity of PTSD (Krakow et al., 2004; Pigeon, Campbell, Possemato & Ouimette, 2013; Spoormaker, & Montgomery, 2008) and have been identified as risk factors for the subsequent development of PTSD (Germain, 2013; Harvey & Bryant, 1998; Koren, Amon, Lavie, & Klein, 2002; Wright et al., 2011). In a longitudinal study of Iraq combat veterans, Wright and colleagues (2011) demonstrated that insomnia indeed increased the risk for subsequent development of PTSD, while PTSD did not increase associated risks for subsequent insomnia. Similarly, Gehrman and colleagues (2013) found that insomnia symptoms and short sleep duration (i.e., < 6 hours)

reported predeployment increased odds of developing PTSD following deployment. Even among patients who respond well to trauma-based exposure therapy for PTSD, 48% report residual insomnia symptoms (Zayfert & DeViva, 2004) in a study of civilian patients and 57% in a study of military service members (Pruiksma et al., 2016). For the majority of individuals in both studies, insomnia persisted in the absence of continuing nightmares. Other studies have demonstrated how residual insomnia symptoms were associated with poorer response to PTSD treatment (López, Lancaster, Gros, & Acierno, 2017).

Among patients with depression, the statistics are similar; 83% report nightmares and/or insomnia (Stewart et al., 2006), and 30%–56% meet diagnostic criteria for insomnia (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). Accumulating evidence suggests that insomnia plays a causal role in onset of depression (Dolsen et al., 2014; Staner, 2010; Taylor, 2008; Wright et al., 2011); individuals who endorse insomnia symptoms are at least twice as likely to develop depression (Baglioni et al., 2011; Neckelmann, Mykletun, & Dahl, 2007; Taylor, Lichstein, & Durrence, 2003). Furthermore, data gathered from clinical trials of both psychotherapies and pharmacotherapy for depression suggests that the presence of insomnia symptoms reduces the likelihood of response to treatment (Troxel et al., 2012) and even in those who reach remission during depression treatment, 50% report continued insomnia (Carney, Segal, Edinger, & Krystal, 2007). However, the psychotherapies represented in this meta-analysis of clinical trials did not include BA. Much less is known about the relationship between nightmares and depression, as nightmares have primarily been associated with PTSD, though one study found nightmares were a significant predictor of both depression and suicidal ideation (Cukrowicz et al., 2006). Furthermore, the prevalence of residual insomnia and nightmares, and their role in predicting response to therapy among veterans with comorbid PTSD and depression, is unclear.

Although about half of veterans with PTSD also have major depressive disorder (Rytwinski et al., 2013), the role of insomnia and nightmares in predicting posttreatment symptoms for comorbid PTSD and depression is still unknown. This study will therefore examine the association of residual insomnia and nightmare symptoms with posttreatment symptoms among veterans receiving exposure therapy and BA for comorbid PTSD and depression (BA-TE). BA-TE is an evidence-based intervention demonstrating engagement of patients (comparable attrition of 29.9% dropout; Gros et al., 2012a) and significant reductions in PTSD and depressive symptoms ($ts > 3.1$; $ps < .01$; $ds > .50$; Strachan

et al., 2012). Results of the current study will provide further insight into whether insomnia and/or nightmare symptoms are viable candidates for early intervention, to assist in boosting response to treatment for comorbid PTSD and depressive symptoms. Based on previous findings (DeViva et al., 2005; Pruiksma et al., 2016; Zayfert & DeViva, 2004), we hypothesized that changes in insomnia and nightmare symptoms would be significantly predictive of PTSD and depressive symptom changes during the BA-TE intervention, in that smaller improvements in insomnia symptoms would be associated with smaller improvements in overall PTSD and depressive symptoms. Given that engagement in therapy is predictive of treatment efficacy, engagement variables were also examined to control for effects of therapy satisfaction and number of therapy sessions attended.

Methods

PARTICIPANTS

Participants were recruited primarily through referrals from physicians and other medical staff at a large southeastern VA medical center (VAMC) and its affiliated community-based outpatient clinics. Eligibility was determined by an in-person intake assessment delivered by master's-level clinicians. Veterans and military personnel meeting DSM-IV-TR criteria for PTSD or subthreshold PTSD per the Clinical Administered PTSD Scale (CAPS; Blake et al., 1995; Acierno et al., 2016). Subthreshold PTSD was operationalized as meeting PTSD Criteria A (traumatic event) and B (reexperiencing), and either C (avoidance) or D (hyperarousal), as well as both duration and impairment criteria (Blanchard, Hickling, Taylor, Loos, & Gerardi, 1994). To enhance generalizability of findings, individuals on psychotropic medications were not excluded from participation, pending a period of stabilization (e.g., 4 weeks after new medication). Veterans from all theaters that met eligibility criteria were included. Exclusion criteria were active alcohol or substance abuse disorder within the past 6 months, an active psychotic disorder, and severe suicidal ideation with plan and intent. Alcohol use disorder was assessed by chart review as well as by using the Alcohol Use Disorders Identification Test, and substance use disorder was assessed using the Drug Abuse Screening Test. Suicidal ideation was assessed using question #9 (suicidal thoughts or wishes) on the Beck Depression Inventory-II and by directly asking the veteran about intent to harm him/herself. Participants receiving psychiatric medication were also not excluded, but were required to be stable on their medication for at least 3 weeks before starting treatment. For the purposes of the present study,

participation in both therapy and research-based assessments were required for participation.

Participants ($N = 232$) were predominantly male (92%), ranged in age from 21 to 77 years ($M = 45.5$ years; $SD = 14.9$), the majority were married (61.6% married, 13.6% never married, 17.2% separated/divorced), and about half of the sample identified as White (49.1% White; 47.0% Black/African American). More than half of the sample (62.4%) reported no suicidal ideation at baseline, and many reported that they were employed (42.7%). Some of the participants (36.9%) were classified as disabled, which was defined as anyone having a VA-rated service connection (e.g., to physical and/or mental health disabilities). Vietnam War veterans accounted for 25.4% of the sample ($n = 59$), Persian Gulf War veterans comprised 23.7% ($n = 55$), and OEF/OIF/OND veterans comprised 50.9% ($N = 118$). Additionally, 92.5% of the respondents to the combat exposure scale reported exposure to combat in a war zone.

PROCEDURES

A full description of the study procedures is reported elsewhere (Gros et al., 2011). All protocols were approved by the local VAMC Research and Development committee as well as the Institutional Review Board at the affiliated university. Participants meeting eligibility requirements were randomized to BA-TE delivered in person (BATE-IP) or via home-based telehealth (BATE-HBT). In addition to completion of the structured clinical interview (CAPS) used for eligibility criteria, baseline measures completed upon randomization and before treatment sessions began consisted of a battery of self-report scales measuring psychological symptoms, satisfaction, and overall perceptions (i.e., the PTSD Checklist-Military Version (PCL-M), the Beck Depression Inventory-II (BDI-II), and the Charleston Psychiatric Outpatient Satisfaction Scale (CPOSS). All participants consented to receive eight 90-minute sessions of BA-TE administered by three master's-level therapists, all with experience in conducting exposure-based therapy for PTSD in prior clinical trials. The specific number of sessions for each participant was determined on a case-by-case basis, taking the participant's progress into account. The mean number of sessions completed was 5.64 ($SD = 3.24$), with 64.9% ($n = 172$) of participants receiving the maximum of 8 sessions. After completing treatment, participants were administered a 1-week posttreatment assessment that consisted of another structured clinical interview (CAPS) and a battery of self-report scales measuring psychological symptoms, satisfaction, and overall perceptions (i.e., the PTSD Checklist-Military

Version [PCL-M], the Beck Depression Inventory-II [BDI-II], and the Charleston Psychiatric Out-patient Satisfaction Scale [CPOSS], respectively, see below). Clinical assessors were blind to participant condition.

MEASURES

PTSD

PTSD symptoms were measured via the CAPS and PTSD Checklist – Military version (PCL-M; Weathers, Huska, & Keane, 1991). The CAPS is a semistructured clinical interview designed specifically for assessing PTSD diagnosis and evidences excellent psychometric properties, including strong convergent and discriminant validity, as well as adequate test-retest and interrater reliability (Blake et al., 1995; Weathers, Keane, & Davidson, 2001).

The PCL-M is a 17-item self-report measure that assesses severity of distress associated with each of the 17 symptoms of DSM-IV PTSD on a 5-point scale (1 = *not at all*, 5 = *extremely*). The PCL-M has good convergent and discriminant validity and demonstrates adequate test-retest reliability and internal consistency among veteran samples (Wilkins, Lang, & Norman, 2011).

Depression

The BDI-II is a 21-item measure designed to assess the cognitive, affective, behavioral, motivational, and somatic symptoms of depression in adults and adolescents (Beck et al., 1996). Each item is rated on a 0 to 3 scale with different responses based on the targeted symptom content. The BDI-II has demonstrated excellent test-retest reliability over a 1-week interval ($r = .93$), excellent internal consistency ($\alpha s < .92$), and convergent and discriminant validity in multiple samples (Beck et al.).

Insomnia

Consistent with previous research (López et al., 2017), two structured clinical interview items from the CAPS (Blake et al., 1995) were used to assess baseline and posttreatment insomnia severity. The first collected information on insomnia frequency by asking, “Have you had any problems falling asleep or staying asleep?” with Likert-type response options of: *Never* (0), *Once or twice* (1), *Once or twice a week* (2), *Several times a week* (3), or *Daily or almost every day* (4). The second collected insomnia intensity by asking, “How much of a problem did you have with your sleep?” with Likert-type response options of *None* (0), *Mild* (1), *Moderate* (2), *Severe* (3), or *Extreme* (4). For both questions participants were asked to consider difficulties with sleep onset, sleep maintenance, and waking too early, as well as number of hours slept per night compared to number of hours of

desired sleep. These two items were summed to create an “insomnia symptom” variable.

Nightmares

Similar to the “insomnia symptom” variable, two structured clinical interview items from the CAPS (Blake et al., 1995) were used to assess baseline and posttreatment nightmares. Information on nightmare frequency was collected by asking, “Have you ever had an unpleasant dream about [reported traumatic event]?” with Likert-type response options of: *Never* (0), *Once or twice* (1), *Once or twice a week* (2), *Several times a week* (3), or *Daily or almost every day* (4). The second structured interview item collected information about intensity of nightmare-related distress by asking the question, “How much distress or discomfort did these dreams cause you?” with Likert-type response options of *None* (0), *Mild* (1), *Moderate* (2), *Severe* (3), or *Extreme* (4). For the intensity of nightmare difficulties, participants were asked to consider whether the dreams woke them up, how long it took to get back to sleep, if dreams ever affected anyone else (and how so), etc. Responses for the nightmare frequency item and the nightmare intensity item were summed to create a “nightmare symptom composite” variable.

Engagement Variables

Therapy satisfaction was measured via the CPOSS. The CPOSS is a 16-item measure assessing four domains of treatment satisfaction (Frueh et al., 2002; Gros, Stauffacher Gros, Acierno, Frueh, & Morland, 2013). Example items assessing satisfaction include: “helpfulness of the services you have received,” “respect shown for your opinions about treatment,” “overall quality of care provided,” and “would you recommend this clinic to a friend or family member.” The CPOSS has evidenced good psychometric properties and is a predictor of postintervention treatment outcomes (Gros et al., 2013). Number of therapy sessions attended was also included as an engagement variable and was operationalized as the sum total of BA-TE sessions attended either in person or via telehealth by the participant. Treatment condition was included to control for any engagement effects of participants who received BA-TE via telehealth versus in person.

STATISTICAL ANALYSES

Hierarchical multiple linear regression models were used to predict posttreatment PTSD and depression symptom severity on the PCL-M and BDI-II, respectively. The statistical significance level was set a priori at $p < .05$. Analyses included responses from participants who completed treatment as well as participants who were considered treatment

dropouts determined by the number of sessions completed (i.e., operationally defined as participants completing less than half of the sessions, thus, 4 sessions or less; [Acierno et al., 2016](#)). Separate hierarchical regression models were used to test the effect of insomnia and nightmares on postintervention PTSD (i.e., sum of PCL-M postintervention score excluding the score of the insomnia item and the score of the nightmare item) and depression (i.e., sum of BDI-II postintervention score excluding the score of the insomnia item) symptoms while controlling for demographics, engagement variables (e.g., number of therapy sessions attended, patient satisfaction and perceptions of quality of service received using the CPOSS, treatment condition), and baseline levels of symptoms. Variables included in each block for each model were: (a) participant demographic characteristics including age, marital status, employment status, and race; (b) baseline PTSD or depressive symptoms and the baseline CAPS insomnia or nightmare scores (sum of frequency and intensity); (c) engagement variables including number of intervention sessions completed, overall quality of care provided (CPOSS), and telehealth versus in-person therapy format (treatment condition); and (d) residual change in CAPS sleep composite scores (predicting postintervention CAPS sleep composite scores from baseline CAPS sleep composite scores). To reduce shared variance in the regression models, total sum scores for baseline and postintervention PTSD and depression scores do not include item scores reported on individual insomnia- and nightmare-related items (i.e., items 13 and 2, respectively, on the PCL-M, and item 16 on the BDI-II).

Results

MISSING DATA

ANOVAs were conducted to test for significant differences in baseline measures of individuals who completed posttreatment self-report surveys versus those that did not. No significant differences between groups on baseline measures of depressive symptoms, PTSD, sleep, and demographic data (age, race, marital status, employment status) were observed. Data were examined for patterns of missing values. Missing data ranged from 1.4% to 12% for demographic variables, 2.9% to 10.8% for baseline measures, and 40.1% to 55.6% for postintervention measures collected 8 weeks after study participation began. Missing value analysis in SPSS was conducted with longitudinal variables. EM (Expectation-maximization) analyses were used to estimate means, correlations, and covariances and determine any systematic patterns of missing data. [Little's \(1988\)](#) MCAR chi-square test was performed

to examine whether data were missing completely at random and found that missing data were likely MCAR, $\chi^2(134) = 133.17, p = .504$. Given that most of the data is intact except for postintervention scores and that data is likely to be missing completely at random, listwise deletion was used to handle missing data.

PRELIMINARY ANALYSES

Baseline depressive symptoms averaged a mean score of 23.62 ($SD = 11.09$; ranged from 1–52) with a postintervention depressive symptoms mean score 18.13 ($SD = 12.09$; ranged from 0–49). For PTSD symptoms, baseline PCL scores were consistent with meeting full PTSD diagnostic criteria and averaged a mean score of 54.75 ($SD = 13.17$; ranged from 23–80). Postintervention PCL scores yielded an average of 45.15 ($SD = 16.90$; ranged from 16–80). For more description of the sample, please refer to the principal study by [Acierno and colleagues \(2016\)](#). Of the sample, 86% of the participants ($N = 229$) positively endorsed sleep problems related to insomnia (Yes or No) for Criterion D of the PTSD DSM I diagnosis. More specifically, reported frequencies of insomnia-related problems on the CAPS assessment ranged as follows: 11% of participants endorsed “Never,” 2.6% “Once or twice,” 9.9% “Once or twice a week,” 18.7% “Several times a week,” and 57.5% of the sample endorsed “Daily or almost every day” problems with insomnia. Participants also reported the intensity of insomnia-related problems as none (11.8%), mild (3.7%), moderate (24.0%), severe (38.4%), and extreme (22.1%). CAPS insomnia composite scores averaged a mean score of 5.63 ($SD = 2.40$; ranged from 0–8) at baseline and an average mean score of 4.56 ($SD = 2.82$; ranged from 0–8) at postintervention. Similarly, 77.7% ($n = 213$) of the sample endorsed problems with nightmares (Yes or No). Of the participants endorsing problems with nightmares, reported frequencies revealed that 20.4% of participants described nightmare-related problems as “Once or twice,” 32.1% reported “Once or twice a week,” 16.1% “Several times a week,” and 10.9% of the sample endorsed “Daily or almost every day” problems with nightmares. The CAPS nightmare composite scores averaged a mean score of 4.05 ($SD = 2.38$; ranged from 0–8) at baseline and an average mean score of 3.10 ($SD = 2.40$; ranged from 0–8) at postintervention. CPOSS scores yielded an average score of 65.49 ($SD = 11.11$; ranged from 34–80). All intercorrelations between independent variables included in the regression models were < 0.64 .

Univariate and multivariate outliers, as well as normality and linearity, were examined and found

within normal ranges. Regarding univariate normality, skewness (-.26 to .42) and kurtosis (-0.90 to -.34) were not problematic for baseline or postintervention variables since values for asymmetry and kurtosis between -2 and +2 are considered acceptable in order to prove normal univariate distribution (George & Mallery, 2010). Plots of residuals of the final steps of each regression model (Normal P-P Plot using SPSS v.21) demonstrated that errors of the regression line adhered to assumptions of normality.

Hierarchical Regressions

Two hierarchical regression models (with four steps per regression model) were used to examine the influence of insomnia and nightmare symptoms on posttreatment PTSD and depressive symptoms after controlling for demographic variables, engagement variables, and baseline levels at pre-intervention of depression and PTSD scores. For postintervention depressive symptoms (post-BDI Score; Table 1), variables entered in stage one of the regression were age, marital status, employment, and race, and these variables did not contribute significantly to the regression model, $\Delta R^2 = .04$, $F_{\text{inc}}(4, 94) = 0.92$, $p = .46$. Adding the baseline CAPS insomnia composite item, baseline CAPS nightmare composite item, and baseline depressive symptoms in stage two of the regression explained an additional 36.8% of the variation in postintervention depressive symptoms and contributed significantly to the model, $F_{\text{inc}}(3, 91) = 18.78$, $p < .001$. Addition of the engagement variables in stage three of the regression model did not contribute significantly, $\Delta R^2 = .03$, $F_{\text{inc}}(3, 88) = 1.45$, $p = .24$. However, the addition of the postintervention CAPS sleep composite scores (insomnia and nightmares) explained an additional 10% of variation in postintervention BDI scores above and beyond the effects of baseline sleep and depressive symptoms, and the increase in variance explained was statistically significant, $F_{\text{inc}}(2, 86) = 9.21$, $p < .001$. When all independent variables were included in step four of the regression model predicting postintervention depressive symptoms, baseline BDI scores ($\beta = .61$, $p < .001$), and the postintervention CAPS frequency and intensity of insomnia composite scores ($\beta = .28$, $p = .003$) were statistically significant predictors. Postintervention CAPS nightmare composite was not significant ($\beta = .15$, $p = .14$).

A similar pattern of results was observed for the hierarchical regression analysis examining the effects of insomnia and nightmares on severity of PTSD symptoms at the end of treatment (see Table 2). Demographic variables entered in stage one of the regression model did not predict significant variance in postintervention PCL scores, $\Delta R^2 = .02$,

$F_{\text{inc}}(4, 97) = 0.61$, $p = .66$. As with the first model, addition of baseline levels of PTSD symptoms, the CAPS baseline insomnia composite score, and the CAPS baseline nightmares composite score in stage two explained an additional 32.7% of the variance in postintervention PTSD symptoms and contributed significantly to the model, $F_{\text{inc}}(3, 94) = 15.82$, $p < .001$. Unlike the previous model examining depressive symptoms, inclusion of engagement variables (attendance, patient satisfaction, treatment format) in the third stage added significantly to the model predicting postintervention PTSD symptoms, $\Delta R^2 = .06$, $F_{\text{inc}}(3, 91) = 2.80$, $p = .04$. Consistent with previous models, addition of the postintervention CAPS insomnia composite scores in stage four also significantly predicted postintervention PCL scores above and beyond the effects of other variables, $\Delta R^2 = .12$, $F_{\text{inc}}(2, 89) = 11.64$, $p < .001$, and explained 12.3% of the variance of PTSD symptoms after the intervention. When all independent variables were entered in the model in stage four, statistically significant predictors included postintervention residual insomnia symptoms ($\beta = .32$, $p = .001$) and baseline PCL scores ($\beta = .58$, $p < .001$).

Discussion

The present study investigated the relationship between postintervention insomnia and nightmare symptoms and symptoms of PTSD and depression during treatment for comorbid PTSD and depressive symptoms via BA-TE. Consistent with our hypotheses, the overall findings demonstrated that pre- to posttreatment changes in insomnia symptoms were predictive of posttreatment PTSD and depressive symptoms above and beyond demographic and process variables across the two models. More specifically, residual symptoms of insomnia (while controlling for baseline levels) were predictive of posttreatment severity in PTSD and depressive symptoms above and beyond the other factors. Baseline insomnia and PTSD/depressive symptoms remained significant as well in the final models. These findings replicate and extend previous research in PTSD-specific psychotherapies to a treatment developed to address comorbid PTSD and depressive symptoms (López et al., 2017), adding to the growing literature on the significance of insomnia in treatment outcome (DeViva et al., 2005; McHugh et al., 2014; Pruiksma et al., 2016; Troxel et al., 2012; Zayfert & DeViva, 2004). Nightmares, however, were not related to posttreatment PTSD and depressive symptoms, indicating perhaps successful reduction in nightmares throughout treatment left minimal residual nightmare symptoms to impact other treatment outcomes. This is consistent with previous studies that

Table 1
Hierarchical Regression Analysis: Sleep Predicting Postintervention Depressive Symptoms

Variable	B	SE B	β	p	R ²	ΔR^2
Step 1					.04	
Age	-.03	.09	-.03	.76		
Marital Status	.05	2.19	.002	.98		
Employment status	4.17	2.63	.17	.12		
Race	-1.84	2.12	-.09	.39		
Step 2					.41 ***	.37 ***
Age	.04	.08	.05	.60		
Marital Status	.47	1.76	.02	.79		
Employment status	1.51	2.13	.06	.48		
Race	-4.03	1.73	-.20 *	.02		
CAPS Baseline Insomnia Composite	.87	.44	.17 *	.05		
CAPS Baseline Nightmare Composite	-.02	.47	-.003	.97		
Baseline BDI-S Score	.70	.10	.59 ***	<.001		
Step 3					.43	.03
Age	.03	.08	.03	.70		
Marital Status	.33	1.79	.02	.86		
Employment status	.91	2.15	.04	.67		
Race	-4.38	1.73	-.22 *	.013		
CAPS Baseline Insomnia Composite	.97	.45	.19 *	.03		
CAPS Baseline Nightmare Composite	-.06	.48	-.01	.89		
Baseline BDI-S Score	.71	.10	.60 ***	<.001		
Number of Sessions Completed	-1.35	2.17	-.05	.54		
Treatment condition	-.74	1.10	-.06	.51		
CPOSS Total Score	-.17	.09	-.15	.08		
Step 4					.53 ***	.10 ***
Age	.05	.07	.05	.53		
Marital Status	.56	1.65	.03	.73		
Employment status	.24	2.01	.01	.91		
Race	-2.92	1.63	-.15	.08		
CAPS Baseline Insomnia Composite	.31	.45	.06	.50		
CAPS Baseline Nightmare Composite	-.60	.50	-.11	.23		
Baseline BDI-S Score	.73	.09	.61 ***	<.001		
Number of Sessions Completed	-1.49	2.00	-.06	.46		
Treatment condition	-.83	1.01	-.06	.42		
CPOSS Total Score	-.08	.09	-.07	.38		
CAPS Post Insomnia Composite	1.30	.43	.28 **	.003		
CAPS Post Nightmare Composite	.78	.52	.15	.14		

Note. BDI-S = Beck's Depression Inventory minus Sleep item; CPOSS = Charleston Psychiatric Outpatient Satisfaction Scale; valid N = 149 (listwise).

* $p < .05$

** $p < .01$

*** $p < .001$

found more improvement in nightmares than insomnia following PTSD treatment (Gutner et al., 2013; Pruiksma et al., 2016; Zayfert & DeViva, 2004).

The present study represents the second study on residual insomnia symptoms in participants with PTSD and comorbid symptoms. In the first study, McHugh and colleagues (2014) found that participants with comorbid PTSD and substance use disorders evidenced residual insomnia symptoms following completion of psychotherapy for PTSD and substance use, and that the change in residual symptoms was associated with PTSD and substance

use improvements during treatment. Relatedly, the present study demonstrated a similar pattern of findings in participants with comorbid PTSD and depressive symptoms following completion of psychotherapy for PTSD and comorbid depressive symptoms. Together, these findings are in line with literature on the transdiagnostic nature of insomnia, as it appears to be present across psychiatric diagnoses (Harvey, Murray, Chandler, & Soehner, 2011), with particularly high rates of comorbidity with PTSD and depression (Gros, Magruder, Ruggiero, et al., 2012; Gros, Price, Magruder, and Frueh, 2012;

Table 2
Hierarchical Regression Analysis: Sleep Predicting Postintervention PTSD Symptoms

Variable	B	SE B	β	p	R^2	ΔR^2
Step 1					.02	
Age	.04	.13	.04	.75		
Marital Status	.33	2.81	.01	.91		
Employment status	4.92	3.58	.14	.17		
Race	1.17	2.90	.04	.69		
Step 2					.35***	.33***
Age	.10	.11	.09	.37		
Marital Status	-.14	2.35	-.01	.95		
Employment status	3.77	2.97	.11	.21		
Race	-3.60	2.51	-.13	.15		
CAPS Baseline Insomnia Composite	1.45	.61	.22*	.019		
CAPS Baseline Nightmare Composite	.34	.68	.08	.62		
Baseline PCL-S Score	.72	.12	.54***	<.001		
Step 3					.41*	.06*
Age	.07	.11	.06	.49		
Marital Status	.20	2.34	.01	.93		
Employment status	2.89	2.94	.08	.33		
Race	-4.20	2.46	-.15	.09		
CAPS Baseline Insomnia Composite	1.65	.60	.25**	.007		
CAPS Baseline Nightmare Composite	.19	.67	.03	.78		
Baseline PCL-S Score	.77	.12	.58***	<.001		
Number of Sessions Completed	-2.39	3.07	-.07	.44		
Treatment condition	.79	1.52	.04	.61		
CPOSS Total Score	-.34	.14	-.21*	.015		
Step 4					.53***	.12***
Age	.13	.10	.11	.20		
Marital Status	.04	2.11	.02	.99		
Employment status	2.46	2.66	.07	.36		
Race	-1.53	2.29	-.06	.51		
CAPS Baseline Insomnia Composite	.58	.59	.09	.33		
CAPS Baseline Nightmare Composite	-.61	.67	-.08	.37		
Baseline PCL-S Score	.77	.11	.58***	<.001		
Number of Sessions Completed	-2.43	2.76	-.07	.38		
Treatment condition	.65	1.37	.04	.64		
CPOSS Total Score	-.21	.13	-.13	.10		
CAPS Post Insomnia Composite	1.96	.57	.32**	.001		
CAPS Post Nightmare Composite	1.23	.67	.17	.07		

Note. PCL-S = PTSD Symptom Checklist minus Sleep item; CPOSS = Charleston Psychiatric Outpatient Satisfaction Scale; valid N = 141 (listwise).

* $p < .05$

** $p < .01$

*** $p < .001$

Gros, Simms, & Acierno, 2010). This hypothesis is supported by research on the reciprocal relation between emotion regulation and sleep as well as the shared neurobiological mechanisms between sleep and the affective disorders (Dolsen et al., 2014; Harvey et al., 2011; Taylor, 2008).

This study failed to find a significant relationship between nightmares and response to trauma treatment, though this may be related to methodological limitations. Specifically, Davis, Pruiksmá, Rhudy, and Byrd (2011) found nightmares that began posttrauma were associated with higher depression and PTSD

than pretrauma nightmares. This information was not collected as part of the current study; thus, timing of nightmares may have been a confounding variable that was not included.

Related to the transdiagnostic perspective, Harvey and colleagues (2011) advocated for sleep-specific treatment interventions to be incorporated within evidence-based psychotherapies designed for other psychiatric diagnoses and/or comorbidities, such as BA-TE. For example, although BA-TE includes components designed to treat PTSD symptoms (situational and imaginal exposures) as well as

depressive symptoms (behavioral activation), no insomnia-specific treatment components are present. Similar to discussion of how insomnia treatment may be a helpful addition to PTSD treatment (Spoormaker & Montgomery, 2008), Harvey and colleagues listed several potential candidate sleep treatments that could be easily incorporated into other treatments, including sleep education, motivational interviewing, and regularizing the sleep cycle. Notably, newer psychotherapy protocols for the affective disorders, such as transdiagnostic behavior therapy, were developed with these suggestions in mind and contains an optional module for sleep disturbance (Gros, 2014). Future research should replicate the procedures of the present study and related studies (López et al., 2017; McHugh et al., 2014) to determine if incorporating additional insomnia and nightmare treatment components affect the residual insomnia symptoms evidenced in prolonged exposure, BA-TE, and other studied treatments.

There are several limitations of the present study. The sample was limited to veterans diagnosed with PTSD, and PTSD related to combat exposure, and a diagnosis of major depressive disorder was not required for participation. The procedures were limited to a single treatment, BA-TE, and without a specific measure for sleep impairment/disorder(s). Although consistent with previous research (López et al., 2017), the measure of insomnia and nightmare symptoms was generated from another measure for PTSD, rather than a stand-alone sleep measure. Although the findings were consistent across symptoms of PTSD and depression self-report measures and sleep items, measures targeting insomnia and nightmares, and assessment of sleep disorders should be included in future studies. While data from a sample of veterans suggests that average scores on insomnia measures are generally similar among veterans with comorbid PTSD compared to veterans with primary insomnia (Straus et al., 2015), use of insomnia- and nightmare-specific psychometric instruments such as the Insomnia Severity Index (Morin, 1993), the Sleep Condition Indicator (Espie et al., 2014), the Trauma Related Nightmare Survey (Davis, Wright, & Borntrager, 2001), or the Structured Clinical Interview for Sleep Disorders for the DSM-V (which assesses for both insomnia and nightmares; Taylor et al., 2018) would allow for assessment of clinically meaningful change in insomnia as well as how much insomnia affects clinically meaningful changes in PTSD and depressive symptoms. Measures of this type should be incorporated into comorbid PTSD and depressive symptom assessment materials to better inform treatment practices and better understand specific aspects of sleep (e.g., difficulty falling asleep, frequent waking, insomnia disorder, nightmare

disorder, poor sleep hygiene, etc.) that contribute to more persistent transdiagnostic symptomatology.

Subsequent studies should also examine specificity of relationships between the transdiagnostic symptom of insomnia and nightmares and different treatment-specific modalities. For example, not only have insomnia symptoms improved after trauma-focused cognitive behavioral therapy (Galovski et al., 2009), but there also seem to exist bidirectional associations with PTSD and depressive symptoms being reduced in the context of sleep-specific cognitive behavioral therapy (Ho et al., 2016; Talbot et al., 2014; Taylor & Pruiksma, 2014). While the role of REM sleep in the promotion of consolidating and dampening intensity of emotional memories helps explain how insomnia might interfere with fear extinction in PTSD treatment (Pace-Schott, Germain, Milad, 2015a; Pace-Schott et al., 2015b), more research is needed to understand the transdiagnostic relationship of insomnia and nightmares with trauma correlates like depression. Subsequent studies may consider symptom profile analyses to help identify subclusters of symptoms across comorbid populations that are driving the associations between insomnia, nightmares, and other anxiety and affective disorders. Such findings may inform how to improve patient care in veteran populations, where insomnia seems to be a prevalent problem for veterans generally, not just among those with PTSD and depressive symptoms (Lewis, Creamer, & Failla, 2009).

The present study investigated the relations between residual insomnia and nightmare symptoms and postintervention PTSD and depressive symptoms during the course of BA-TE for comorbid PTSD and depressive symptoms. Residual insomnia symptoms were predictive of severity in postintervention symptoms of PTSD and depressive symptoms after receiving BA-TE, though nightmares were not. These findings further emphasize the transdiagnostic nature of insomnia, potentially highlighting the need for the incorporation of sleep-specific treatment components into the existing psychotherapies for the affective disorders.

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